Cognitive decline before and after mid-to-late-life smoking cessation: a longitudinal analysis of prospective cohort studies from 12 countries

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Summary

Background Whether short-term improvements in cognitive performance observed following smoking cessation are transient or if longer-term cognitive trajectories are also improved is unclear, particularly when adults are middle-aged or older at smoking cessation. We examined whether long-term cognitive trajectories improved following mid-to-late-life smoking cessation.

Methods In this longitudinal study, we used data from three nationally representative cohort studies from 12 countries including 18 years of cognitive data (2002–20). Participants who quit smoking during follow-up were matched with an equal number of continuing smokers according to key demographic, socioeconomic, and cognitive criteria. We used piecewise linear mixed models to examine memory and fluency decline before and after smoking cessation and during a comparable time period in continuing smokers.

Findings We included data from 9436 participants who smoked (4718 [50·0%] smokers who quit matched with 4718 [50·0%] continuing smokers, aged 40–89 years, with 4886 [51·8%] women and 4550 [48·2%] men). In the six years before smoking cessation, matched smokers who quit and continuing smokers had similar rates of memory and fluency decline (difference in memory decline [smokers who quit–continuing smokers] –0·03 SDs [95% CI –0·06 to 0·01], p=0·16; difference in fluency decline –0·01 [–0·04 to 0·03], p=0·76). In the six years following smoking cessation, smokers who quit had memory and fluency scores that declined more slowly than continuing smokers (difference in memory decline 0·05 SDs [0·00–0·10], p=0·036; difference in fluency decline 0·05 SDs [0·01–0·10], p=0·030). Coefficients for interaction with age at smoking cessation suggested results did not differ by age at smoking cessation (p>0·05 for all).

Interpretation In middle-aged and older smokers with initially similar cognitive trajectories, smokers who quit subsequently had more favourable trajectories than continuing smokers regardless of age at cessation. As older adults are less likely than younger people to attempt smoking cessation, improvements in long-term cognitive trajectories might provide an additional motivation to quit.

Funding National Institute on Aging, National Institute for Health and Care Research.

Lancet Healthy Longev 2025; 6: 100753

Published Online October 13, 2025
https://doi.org/10.1016/j.lanhl.2025.100753

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Introduction

Alzheimer's disease and related dementias are the eighth leading cause of death,[1] with an estimated 56·9 million people living with dementia globally.[2] Diagnosis of Alzheimer's disease—the most common cause of dementia—occurs following a decades-long process of neuropathological changes and cognitive decline.[3] As such, targeting modifiable risk factors for cognitive decline from midlife onwards has become a primary focus of research aimed at prevention.[4] Among these risk factors, cigarette smoking has emerged as a potential cause of accelerated cognitive decline.[4] Smoking is thought to contribute to neurodegeneration via oxidative stress and inflammation, while also increasing risk of cardiovascular disease, leading to cognitive decline and increased dementia risk.[5]

Although the association between smoking and cognitive health is well established, the long-term cognitive effects of smoking cessation—particularly smoking cessation during mid-to-late life when smoking might have already begun to affect cognitive ageing—is less clear. Two previous small-scale smoking cessation trials of middle-aged and older adults (ages 68–88 years[6] and 35–70 years[7]) have shown short-term improvement in cognitive performance in the 6–24 months following smoking cessation. Whether these improvements are transient or will translate to slower cognitive decline in the long term, mitigating the effect of smoking on cognitive ageing, is not known.

As older adults suffer the most severe health consequences of smoking[8] and are less likely to attempt smoking cessation than younger people,[9] identifying compelling reasons for older adults to quit—such as reversibility of cognitive harms—remains an important focus for public health initiatives. In this study, we used 18 years of cognitive data from 9436 participants (aged 40–89 years) in

Research in context

Evidence before this study

We searched PubMed for publications until Nov 19, 2024, using the search terms "smoking", "cognitive decline", "smoking cessation", "cognitive function", and "cognitive ag*ing". Smoking is associated with accelerated cognitive decline and increased risk of dementia, and short-term improvement in cognitive performance has been observed following smoking cessation in small-scale trials. Whether this improvement in cognitive performance is transient or if longer-term cognitive trajectories are also improved—ie, cognitive decline slows following smoking cessation—is not known, particularly for middle-aged or older smokers, in whom smoking might have already begun to affect cognitive health and ageing.

Added value of this study

With less than 10% of serious attempts to quit smoking succeeding after 1 year, identifying novel and compelling reasons to attempt to quit remains an important focus for public health initiatives. This focus is particularly relevant for older adults who are both less likely than younger people to try to quit smoking and also experience the largest burden of health consequences of smoking. Our large-scale longitudinal study using cognitive outcomes collected over 18 years showed that in middle-aged and older smokers with initially similar cognitive trajectories, those who quit smoking had slower cognitive decline following smoking cessation than matched individuals who did not quit smoking regardless of age at quitting. These findings suggest the potential reversibility of smoking-related cognitive harms and could motivate older adults to try to quit smoking, offering new evidence to support the public health message that it is never too late to quit.

Implications of all the available evidence

Cumulative evidence indicates that the cognitive harms of smoking are not necessarily permanent and might be mitigated by smoking cessation. Our findings suggest that smoking cessation even later in life can have meaningful benefits for long-term cognitive function.

three large-scale, nationally representative, ageing studies including 11 European countries and the USA. We examined cognitive trajectories before and after mid-to-late-life smoking cessation compared with a matched control group of participants who did not quit smoking over a comparable time period to assess whether trajectories improved following smoking cessation.

Methods

Data sources

In this secondary analysis of an observational cohort study, data were drawn from the English Longitudinal Study of Ageing (ELSA), the Survey of Health, Ageing and Retirement in Europe (SHARE), and the Health and Retirement Study (HRS). ELSA (2002–03 to 2021–23), SHARE (2004–05 to 2019–20), and HRS (1992–93 to 2022) are nationally-representative cohort studies of individuals aged at least 50 years and their partners (of any age) residing in England, 28 countries of Europe and Israel, or the USA. ELSA, SHARE, and HRS are approximately biennial surveys, with similar design to facilitate harmonisation.[10–12] All cohorts were granted relevant local ethics approval, with written informed consent given at each interview. Of the HRS family of studies, these three studies were chosen for their long follow-up periods and comparable cognitive tests.

Waves 1–9 of ELSA (2002–03 to 2018–19), 1–2 and 4–8 of SHARE (2004–05 to 2019–20; wave 3 of SHARE is a life history module not related to the main interview), and 8–14 of HRS (2006–07 to 2018–19) including refreshment cohorts were used in the present analysis to harmonise study years between surveys on the basis of the availability of comparable cognitive and smoking data (appendix p 29). Wave 1 of ELSA and SHARE and Wave 8 of HRS were therefore considered to be the baseline waves in the present study. To make follow-up consistent between cohorts, SHARE countries participating in the first wave of data collection in 2004–05 with at least four consecutive waves were included (Austria, Germany, Sweden, the Netherlands, Spain, Italy, France, Denmark, Switzerland, and Belgium).

See Online for appendix

To be able to examine smoking cessation during midlife in addition to late life, in this analysis we included participants and partners aged at least 40 years who reported smoking at the baseline waves of the present study and had at least two waves of data. We included participants aged 40 years and older to capture smoking cessation across the full midlife period (roughly ages 40–64 years) as well as later life; restricting age to 50 years and older would have missed smoking cessation that occurs in early midlife. Data from all cohorts were pooled.

Smoking status

At each interview, participants were asked if they "smoke cigarettes at all nowadays" (ELSA), "smoke at the present time" (SHARE), or "smoke cigarettes now" (HRS). Participants were split into "smokers who quit" (who answered "no" to the smoking question for at least one wave) and "continuing smokers" (reported smoking at all waves). If a smoker who quit subsequently reported smoking again during the follow-up period, the wave at which they reported smoking again and subsequent waves were excluded from analyses.

Cognitive function

The cognitive domains examined were episodic memory and verbal fluency, which are key for day-to-day function and show decline with dementia,[13] and are relevant to health and wellbeing. All cohorts used the same tests of

memory and fluency, enabling harmonised analyses. At each interview, memory was assessed using immediate and delayed recall tasks[14] and fluency using the animal naming task (appendix p 5).[15] Raw mean cognitive scores by wave in each country are presented in the appendix (pp 9–10); these values reflect a mix of cohort entry patterns, intermittent missingness, and differences in age distribution, and should not be interpreted as within-person change. Given absolute differences in cognitive scores between countries, cognitive scores were standardised by country (ie, with mean 0 and SD 1 in each country).

Other covariates

Covariates included birth year, country, sex (male or female), education level (lower than upper secondary, upper secondary, or above upper secondary), household wealth (standardised to country and year), alcohol consumption (whether consumes alcohol or not), psychiatric conditions (yes or no, based on self-report of clinically diagnosed psychiatric condition, score on the eight-item Centre of Epidemiologic Studies Depression Scale $\geq$3, or 12-item EURO-D score $\geq$4[16,17]), and self-report of clinical diagnosis of each of the following conditions (yes or no): high blood pressure, diabetes, lung disease, cardiovascular disease, stroke, and cancer. Covariates were drawn from the baseline wave.

Matching

In similar methods to previous studies,[18,19] we selected the control group (continuing smokers) by coarsened exact matching,[20] using age at baseline, sex, education level, birth year, country, and mean standardised cognitive score at baseline (ie, the mean of standardised memory and fluency scores). Participants were assigned to a matching stratum on the basis of these characteristics (appendix p 6). Because we matched according to cognitive score at baseline, we did not exclude individuals with cognitive impairment or dementia to include the full spectrum of cognitive health in both groups.

Statistical analysis

We used piecewise linear mixed models to examine differences in cognitive trajectories between smoking groups, with separate models for each cognitive domain. Linear mixed models use all available data regardless of length of follow-up, and handle non-monotone missingness patterns and attrition assuming data missing-at-random.[21] All models included one random intercept and two slopes (for before and after smoking cessation) at the individual level with an unstructured covariance matrix to account for differences in cognitive trajectories between individuals.

In piecewise regression modelling, slope is allowed to differ before and after a predefined timepoint ($t = 0$), and each participant's follow-up period is centred at $t = 0$. This piecewise model structure allows us to estimate whether trajectories diverge following smoking cessation while accounting for and comparing pre-cessation trends within a single framework. Modelling both periods simultaneously is necessary to assess the difference-in-difference in decline and to support causal inference by showing similar pre-cessation trends between groups. Time was assigned the value of 0 at the age when participants first reported smoking cessation ($age_{t=0}$; appendix p 11) or, for continuing smokers, the median age at smoking cessation in the participant's matching stratum.

Two time terms were included in each model to correspond to the periods before and from $t = 0$: *pre time* for $t<0$ and *post time* for $t\geq0$. All models included smoking group (smoker who quit or continuing smoker), *pre time*, *post time*, and interactions of smoking group with *pre time* and *post time*. Models were also adjusted for $age_{t=0}$ centred at age 65 years (the median age at smoking cessation), interactions of $age_{t=0}$with *pre time* and *post time* (to allow cognitive trajectories to differ depending on age), birth year, country, sex, education, wealth, alcohol consumption, psychiatric conditions, and chronic conditions (appendix p 8).

We focused on reporting results for the 6 years before and after $t = 0$, corresponding to 12 years in total (the maximum follow-up period for HRS, the cohort with the shortest follow-up). We reported two differences between smoking groups to assess whether cognitive trajectories improved following smoking cessation: (1) the difference in 6-year cognitive decline between smokers who quit and continuing smokers during $t<0$ and $t\geq0$, where a positive value would indicate smokers who quit had slower cognitive decline than continuing smokers for the given time period; and (2) the difference-in-difference, calculated by subtracting the difference in 6-year cognitive decline between smoking groups for $t<0$ from the difference in 6-year cognitive decline between smoking groups for $t\geq0$. A positive value for difference-in-difference indicates the cognitive trajectory improved for smokers who quit relative to continuing smokers.

Finally, to visualise differences in cognitive decline, we plotted 6-year cognitive trajectories before and from $t=0$ for smokers who quit and continuing smokers with covariates at their reference values (born 1945–49, residing in England, male, upper secondary education, mean wealth, consumes alcohol, no psychiatric conditions, no chronic conditions, and aged 65 years at $t = 0$). We also plotted the average marginal effect of smoking group for the same period. Analyses were performed in StataMP (version 18.0) with a two-sided p value below 0·05 considered significant.

We did several additional analyses. First, we included interactions between smoking group, $age_{t=0}$, and pre time or post time to assess whether results differed depending on age of smoking cessation. Second, although heaviness of smoking might affect findings, variable missingness prevented us from including this covariate in the main analysis; we did a sensitivity analysis where we imputed daily number of cigarettes (appendix p 4). Third, there might be

	Smokers who quit (n=4718)	Continuing smokers (n=4718)	p value
Standardised cognitive score (mean, SD)	0·06 (0·75)	0·05 (0·75)	0·61
Age, years (mean, SD)	58·3 (7·6)	58·4 (7·6)	0·89
Birth year (median, IQR)	1949 (1942–1954)	1950 (1943–1955)	0·0071
Country			
Austria	211 (4·5%)	234 (5·0%)	>0·99
Germany	222 (4·7%)	199 (4·2%)	..
Sweden	197 (4·2%)	197 (4·2%)	..
Netherlands	257 (5·4%)	257 (5·4%)	..
Spain	247 (5·2%)	247 (5·2%)	..
Italy	315 (6·7%)	315 (6·7%)	..
France	252 (5·3%)	252 (5·3%)	..
Denmark	288 (6·1%)	288 (6·1%)	..
Switzerland	169 (3·6%)	169 (3·6%)	..
Belgium	277 (5·9%)	277 (5·9%)	..
England	871 (18·5%)	871 (18·5%)	..
USA	1412 (29·9%)	1412 (29·9%)	..
Sex			
Male	2273 (48·2%)	2277 (48·3%)	0·95
Female	2445 (51·8%)	2441 (51·7%)	..
Race*			
White	1756 (37·2%)	1775 (37·6%)	0·83
Non-White	525 (11·1%)	508 (10·8%)	..
Missing	2437 (51·7%)	2435 (51·6%)	..
Education†			
Low	1416 (30·0%)	1470 (31·2%)	0·45
Intermediate	2587 (54·8%)	2533 (53·7%)	..
High	715 (15·2%)	715 (15·2%)	..
Standardised wealth (mean, SD)	−0·13 (0·77)	−0·16 (0·73)	0·040
Consumes alcohol	3182 (67·4%)	3164 (67·1%)	0·71
Psychiatric conditions	1502 (31·8%)	1720 (36·5%)	<0·0001
Reports diagnosis of:			
High blood pressure	1490 (31·6%)	1498 (31·8%)	0·88
Diabetes	450 (9·5%)	469 (9·9%)	0·53
Cancer	237 (5·0%)	261 (5·5%)	0·29
Lung disease	377 (8·0%)	437 (9·3%)	0·031
Cardiovascular disease	444 (9·4%)	499 (10·6%)	0·064
Stroke	166 (3·5%)	210 (4·5%)	0·024
Years in study smoking (mean, SD)	5·9 (3·8)	5·7 (2·6)	0·0005

Data are n (%), unless otherwise indicated. *Race and ethnicity data are not available in the Survey of Health, Ageing and Retirement in Europe. †Low education is lower than upper secondary, intermediate is upper secondary, and high is above upper secondary.

Table 1: **Baseline characteristics**

an acute improvement in cognitive performance following smoking cessation,[7] the cognitive score measured at $t=0$ could show a discrete improvement that deviates from t]he previous cognitive trajectory observed for $t\leq 0$, particularly as there might be a gap between smoking cessation and reporting smoking cessation. We reran analyses including additional terms in the model (a discontinuity indicator and random slope for discontinuity) to allow for this discrete change. We also reran analyses including the following in models: self-reported physical activity (moderate to vigorous physical activity weekly or not), time-varying covariates until $t=0$, higher-order time terms (quadratic and cubic) to examine non-linearity of cognitive trajectories, and interactions between time and time-invariant covariates.

We ran exploratory models to assess heterogeneity in the results by country or cohort (appendix pp 12, 30). Finally, we ran unadjusted models in the complete unmatched sample with $age_{t=0}$ for continuing smokers assigned as the median age at quitting smoking overall. These models (appendix pp 13, 31) do not account for baseline imbalances or confounding and should not be used for inference.

Role of the funding source

The funders of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report.

Results

Of 16 883 ELSA, SHARE, and HRS participants aged at least 40 years who smoked at baseline, 1114 (6·6%) were missing all cognitive data, 359 (2·1%) were missing covariate data, and 1224 (7·7%) had fewer than two waves of data and were excluded from analyses. Of the remaining 14 186 (84·0%) participants, 4718 (33·3%) smokers who quit were matched with 4718 (33·3%) continuing smokers, leading to an analytic sample of 9436 participants (1742 [18·5%] ELSA, 4870 [51·6%] SHARE, and 2824 [29·3%] HRS; appendix pp 32–34). Excluded participants were generally similar to included participants, but were less healthy and had worse memory scores (appendix p 14).

At baseline, smokers who quit and continuing smokers were similar with respect to age (mean age 58·3 years [SD 7·6] for smokers who quit and 58·4 years [SD 7·6] for continuing smokers), birth year (in real terms—only a difference of 1 year), country, sex (2245 [51·8%] women and 2273 [48·2%] men for smokers who quit and 2441 [51·7%] women and 2277 [48·3%] men for continuing smokers), education level, alcohol consumption, prevalence of high blood pressure, diabetes, cancer, and cardiovascular disease, and baseline cognitive performance (table 1). Smokers who quit were slightly less wealthy than continuing smokers (p=0·040), and less likely to have psychiatric conditions (p<0·0001), lung disease (p=0·031), or stroke (p=0·024). For those who reported number of daily cigarettes, the mean for smokers who quit was 12·1 (SD 8·9) compared with 13·5 (9·7) for continuing smokers. The mean age at time $t=0$ was 64·2 years (8·1) for smokers who quit and 64·0 years (7·3) for continuing smokers. The median follow-up period was 8 years (IQR 5–12) for smokers who quit and 6 years (2–9) for continuing smokers.

A year increase in age corresponded to an average decline in memory performance of 0·03 SDs (95% CI 0·02 to 0·03) or fluency performance of 0·02 SDs (0·01 to 0·02). The average marginal memory decline in the 6 years before $t=0$ in the sample overall was 0·04 SDs (0·02 to 0·06): 0·05 SDs (0·03 to 0·08) for smokers who quit and 0·03 SDs (0·01 to 0·06) for continuing smokers (figures 1, 2). Smokers who

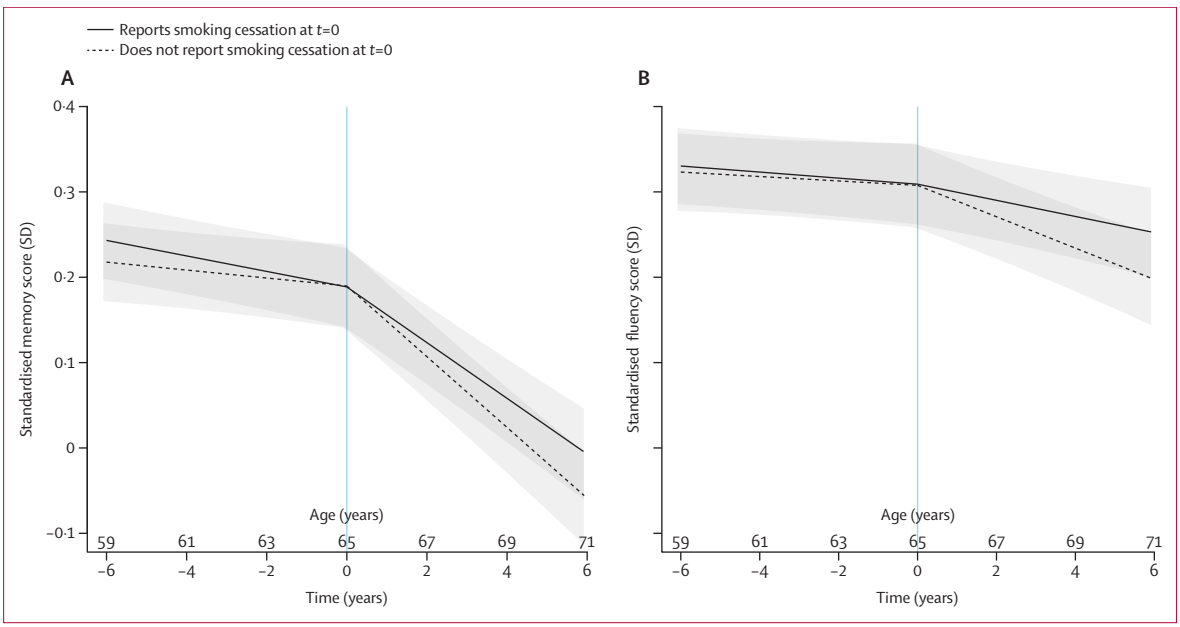

Figure 1: **12-year memory and fluency trajectories before and after smoking cessation (n=9436)**
Time (*t*)=0 is age of reporting smoking cessation or equivalent age for continuing smokers; plotted results are for when participants are aged 65 years at *t*=0. Models include smoking group, *pre time*, *post time*, interactions of smoking group with *pre time* and *post time*, and are adjusted for $age_{t=0}$, interactions of $age_{t=0}$ with *pre time* and *post time*, birth year, sex, education, standardised wealth, alcohol consumption, psychiatric conditions, and self-reported chronic conditions, and plotted results are for reference values of covariates (born 1945–49, resident in England, male, upper secondary education, mean wealth, consumes alcohol, no psychiatric conditions, and no chronic conditions). Shading indicates 95% CIs.

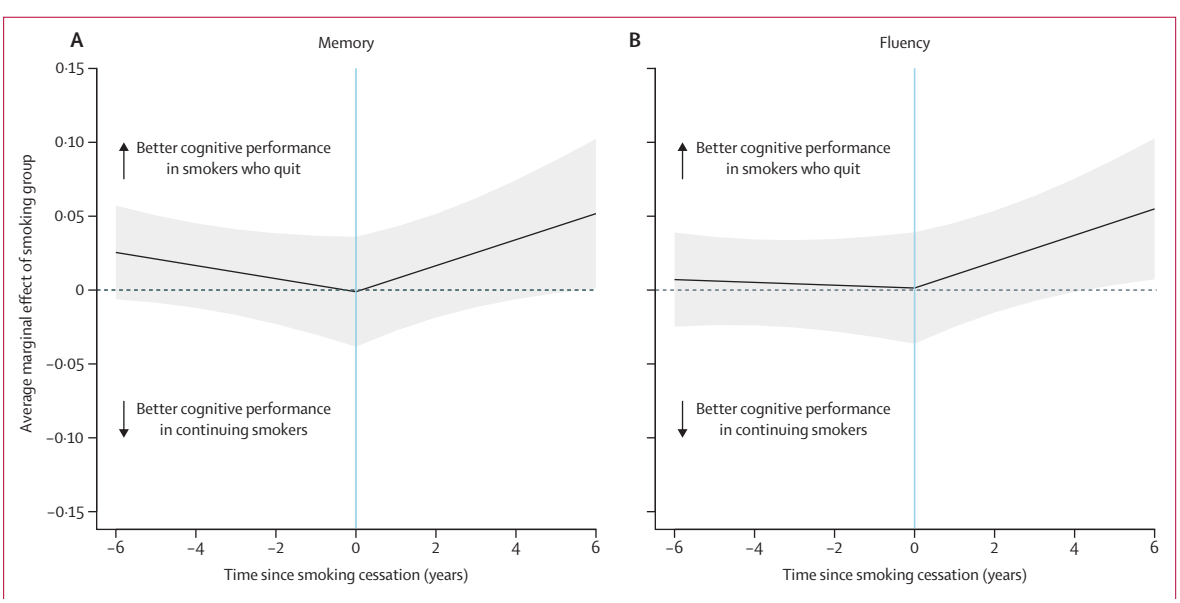

Figure 2: **Average marginal effect for smoking group**
Average marginal effect for smoking group (smoker who quit or continuing smoker). Y-axis indicates magnitude of cognitive difference between smoking groups. A value above 0 indicates better cognitive performance in smokers who quit and a value below 0 indicates better cognitive performance in continuing smokers. Shading indicates 95% CIs.

quit and continuing smokers had a similar rate of memory decline for $t<0$. The difference in memory decline between smokers who quit and continuing smokers over the 6 years preceding $t=0$ was –0·03 SDs (–0·06 to 0·01; p=0·16; table 2).

The average marginal memory decline in the 6 years after $t=0$ was 0·21 SDs (95% CI 0·19–0·24) in the sample overall: 0·19 SDs (0·16–0·22) for smokers who quit and 0·24 SDs (0·20–0·30) for continuing smokers (figures 1, 2). In the period $t\geq0$, smokers who quit declined more slowly than continuing smokers, with smokers who quit declining 0·05 SDs (0·00–0·10) less than continuing smokers during the 6-year period following $t=0$ (p=0·036). The memory

	Difference in 6-year cognitive decline, smokers who quit–continuing smokers		Difference-in-difference
	$t<0$	$t\geq 0$	
Memory			
Estimate (95% CI)	–0·03 SDs (–0·06 to 0·01)	0·05 SDs (0·00 to 0·10)	0·08 SDs (0·01 to 0·15)
p value	0·16	0·036	0·028
Fluency			
Estimate (95% CI)	–0·01 (–0·04 to 0·03)	0·05 (0·01 to 0·10)	0·06 (–0·01 to 0·13)
p value	0·76	0·030	0·098

Time (t)=0 is age of reporting smoking cessation or equivalent age for continuing smokers. Difference in 6-year cognitive decline (quitters–continuing smokers) corresponds to difference in cognitive decline over 6 years in SDs between quitters and continuing smokers for given time t; a positive value indicates quitters decline more slowly than continuing smokers for given time t. A positive value for difference-in-difference indicates improvement in cognitive trajectories for quitters relative to continuing smokers. All models include smoking group, *pre time*, *post time*, interactions of smoking group with *pre time* and *post time*, and are adjusted for $age_{t=0}$, interactions of $age_{t=0}$ with *pre time* and *post time*, birth year, sex, education, standardised wealth, alcohol consumption, psychiatric conditions, and self-reported chronic conditions.

***Table 2*: Difference in 6-year cognitive decline between quitters and continuing smokers (n=9436)**

trajectory of smokers who quit improved after smoking cessation, with a value for difference-in-difference of 0·08 SDs (0·01–0·15; p=0·028; table 2).

The average marginal fluency decline in the 6 years before $t=0$ in the sample overall was 0·02 SDs (95% CI 0·00 to 0·03): 0·02 SDs (–0·01 to 0·04) for smokers who quit and 0·01 SDs (–0·02 to 0·04) for continuing smokers (figures 1, 2). During $t<0$, there was a similar rate of fluency decline between smokers who quit and continuing smokers. The difference in fluency decline between smoking groups during this period was –0·01 SDs (–0·04 to 0·03; p=0·76; table 2).

The average marginal fluency decline in the 6 years after $t=0$ in the sample overall was 0·08 SDs (95% CI 0·05 to 0·10): 0·05 SDs (0·02 to 0·09) for smokers who quit and 0·11 SDs (0·07 to 0·14) for continuing smokers (figures 1, 2). In the period $t\geq 0$, the rate of fluency decline was slower for smokers who quit than for continuing smokers. Smokers who quit had fluency scores that declined 0·05 SDs (0·01 to 0·10) less than those of continuing smokers over the 6-year period from $t=0$ (p=0·030). The difference-in-difference for fluency was 0·06 SDs (–0·01 to 0·13), indicating an improvement in fluency trajectory following smoking cessation; however, this improvement did not reach statistical significance (p=0·098).

Results were similar for all ages at smoking cessation (appendix p 15), when adjusted for daily number of cigarettes (appendix pp 16–17), and when allowing discrete changes in cognitive score at $t=0$ (appendix pp 18–19), with no evidence of a discrete improvement in cognitive score at $t=0$ for either smoking group (appendix p 20). Conclusions were unchanged in other sensitivity analyses (appendix pp 21–28).

Discussion

The key result arising from this longitudinal study using 18 years of data from 9436 middle-aged and older adults is that in smokers who initially had similar cognitive trajectories, regardless of age at smoking cessation, individuals who quit smoking had more favourable trajectories following smoking cessation than continuing smokers: the rate of cognitive decline was slower for smokers who quit than for continuing smokers in the period after smoking cessation. The results suggest the importance of smoking cessation, even in later life, for long-term cognitive health.

The main strengths of this study are its long follow-up period, age range, use of data from large-scale ageing studies, and use of matching. The long follow-up period meant there was sufficient cognitive data to break follow-up into two periods (before and after smoking cessation), allowing us to examine cognitive decline over a longer period of time than in previous studies, which also did not include a pre-cessation period to establish cognitive trends.[6,7] The age range allowed us to examine smoking cessation in an age group that is particularly relevant to public health efforts. Using data from nationally representative studies enhances generalisability and improves on previous results from small-scale trials. Finally, matching improves on standard observational methods, strengthening the inferences that can be drawn by ensuring smoking groups are comparable with respect to key characteristics.

There are limitations to the present study. Unmeasured confounding could still threaten causality, and the analysis rests on the assumption that the smoking groups are similar. We were not able to use other quasi-experimental methods for causal inference (eg, difference-in-difference) because of violations to the assumptions of these models[22]—eg, the decision to quit smoking might depend on cognitive function. However, using piecewise models allowed us to make similar comparisons with fewer assumptions. Differences between cohorts could affect the accuracy and precision of the results—eg, between-cohort differences in wording of the smoking question could affect the ability to capture occasional smokers. However, we accounted for between-country and between-cohort differences by adjusting for country and standardising cognitive scores. Differential attrition between smoking groups could affect the results as the ability of mixed models to handle missing data assumes data are missing-at-random; however, follow-up durations were similar between smoking groups, with the difference in median follow-up corresponding to one wave of data collection, suggesting differential attrition is unlikely to have affected the findings. We depended on self-report for chronic conditions, which might result in some misclassification, although evidence suggests that self-reporting for the included conditions generally has good sensitivity and specificity when compared with medical records.[23] The analysed sample was somewhat healthier than those participants excluded from the analysis, which might affect generalisability. Furthermore, because the study comprises older smokers surviving long enough to be included, the analysis is likely to have included individuals whose health is less affected by smoking compared with the average

smoker because of a combination of individual characteristics (eg, smoking history or genetics) and environmental factors. However, this detail does not pose a limitation, because surviving older smokers are the target population for intervention. We could not include a more detailed measure of alcohol consumption because these questions were not administered consistently across cohorts. Despite the relationship between smoking and mortality, we could not examine whether terminal decline affected the results because of a lack of data. However, results did not vary by age; if terminal decline played an important role we would expect a larger difference in cognitive decline at older ages. Finally, we were unable to assess whether results varied by race or ethnicity because these data were unavailable in SHARE. Future research should examine moderating effects of demographic and socioeconomic characteristics, explore results for other cognitive domains, and clarify the effect of smoking history, which we could not comprehensively examine because of missing data. Data sparsity also precluded examining between-country heterogeneity, which is another key area of future research, as effects of smoking cessation might differ between countries.

The findings of the present study align with small-scale smoking cessation trials (n=22–229) suggesting cognitive benefits in the 6–24 months following smoking cessation in middle-aged and older adults.[6,7] By contrast with one of these studies in which cognitive scores improved 6 months after smoking cessation,[7] we did not see an improvement in cognitive performance, but rather a reduction in the rate of decline. This difference is probably attributable to the younger age distribution in the previous study (mean age 45 years), given that memory and fluency decline generally begin to accelerate from age 60–65 years onwards,[24] and to the 2-year interval between cognitive assessments in the present study, which might not capture improvement in cognitive scores immediately after smoking cessation. The fact that we observed more rapid cognitive decline in the second half of follow-up when cognitive trajectories were centred at age 65 years (ie, participants were aged 65 years at $t = 0$) regardless of smoking cessation is also consistent with memory and fluency ageing trajectories.[24] Finally, our findings complement studies that show adults aged at least 65 years who quit smoking aged 44 years or younger had better cognitive scores than current smokers,[25] and that former and never smokers have a similar risk of dementia a decade or longer after quitting.[26] Smoking is thought to affect long-term cognitive health through its cardiovascular effects;[5] however, even for adults aged 65 years and older, excess risk of myocardial infarction, stroke, and cardiovascular death might be reduced 5 years after smoking cessation,[27] suggesting the worst cardiovascular effects might be partly reversible and providing a plausible explanation for the findings.

Preventive Alzheimer's disease strategies focus on improving overall cognitive trajectories by mitigating cognitive decline in the decades preceding dementia diagnosis, and thereby delaying onset of cognitive symptoms. Our results show that later-life smoking cessation is associated with a delay in cognitive decline corresponding to up to 3 years of cognitive ageing over 6 years. This benefit accumulates further over time. As a comparison, current Alzheimer's disease therapies delay progression of cognitive decline by around 5 months over a period of 18 months.[28] This research supports clinical practice and public health initiatives that encourage smoking cessation for older smokers to mitigate cognitive decline and potentially delay onset of dementia.

With the largest health consequences of smoking experienced by older people,[8] less than 10% of attempts to quit smoking succeeding after 1 year,[29] and a commitment of £15 million per year by the UK Government to fund national smoking-cessation campaigns,[30] identifying novel, compelling reasons that might motivate older smokers to attempt to quit is both clinically relevant and relevant to public health messaging. The present findings reiterate the negative effects of smoking on cognitive health and offer new evidence to support smoking cessation at any age.

Contributors

MB and JB contributed to conceptualisation. MB, JB, GDG, and FB contributed to the methodology. MB contributed to formal analysis. MB and AS contributed to data curation. MB wrote the original draft. All authors contributed to manuscript review and editing. MB contributed to visualisation. AS supervised the study and acquired funding. MB and AS accessed and verified the data. All authors had full access to the data and accept responsibility to submit for publication.

Declaration of interests

JB declares funding from Cancer Research UK and royalties from Wiley. All other authors declare no competing interests.

Data sharing

ELSA data are available to researchers after registration with the UK Data Service at https://beta.ukdataservice.ac.uk/datacatalogue/series/series?id=200011. SHARE data are accessible after registration with the SHARE project at the following addresses: Wave 1 (https://doi.org/10.6103/SHARE.w1.710), Wave 2 (https://doi.org/10.6103/SHARE.w2.710), Wave 4 (https://doi.org/10.6103/SHARE.w4.710), Wave 5 (https://doi.org/10.6103/SHARE.w5.710), Wave 6 (https://doi.org/10.6103/SHARE.w6.710), and Wave 7 (https://doi.org/10.6103/SHARE.w7.711 and (https://doi.org/10.6103/SHARE.w8cabeta.001). HRS data are available after registration at https://hrs.isr.umich.edu/.

Acknowledgments

ELSA is funded by the National Institute on Aging (R01AG017644), and by UK Government Departments coordinated by the National Institute for Health and Care Research. SHARE has received funding from the EU under grant agreements 101102412 and the EU's Horizon 2020 research and innovation programme under grant agreements 870628 and 101015924. HRS is funded by the National Institute on Aging (U01AG009740) and the Social Security Administration. ELSA Wave 1 received ethics approval from the London Multi-Centre Research Ethics Committee on Feb 7, 2002 (MREC/01/2/91). ELSA Wave 2 received ethics approval from the London Multi-Centre Research Ethics Committee on Aug 12, 2004 (MREC/04/2/006). ELSA Wave 3 received ethics approval from the London Multi-Centre Research Ethics Committee on Oct 27, 2005 (05/MRE02/63). ELSA Wave 4 received ethics approval from the National Hospital for Neurology and Neurosurgery & Institute of Neurology Joint Research Ethics Committee on Oct 12, 2007 (07/H0716/48). ELSA Wave 5 received ethics approval from the Berkshire Research Ethics Committee on Dec 21, 2009 (09/H0505/124). ELSA Wave 6 received ethics approval from the NRES Committee South Central–Berkshire on Nov 28, 2012 (11/SC/0374). ELSA Wave 7 received ethics approval from the NRES

Committee South Central–Berkshire on Nov 28, 2013 (13/SC/0532). ELSA Wave 8 received ethics approval from the South Central–Berkshire Research Ethics Committee on Sept 23, 2015 (15/SC/0526). ELSA Wave 9 received ethics approval from the South Central–Berkshire Research Ethics Committee on May 10, 2018 (17/SC/0588). Ethics approval of SHARE from the first to fourth waves was obtained from the Ethics Committee of the University of Mannheim. Most recently, in 2021, the Ethics Council of the Max Planck Society reviewed and approved the fourth and the consecutive waves of the SHARE project. Ethics approval for HRS was obtained from the University of Michigan Institutional Review Board. No further ethics approval was required for the present study.

References

1 Naghavi M, Ong KL, Aali A, et al, and the GBD 2021 Causes of Death Collaborators. Global burden of 288 causes of death and life expectancy decomposition in 204 countries and territories and 811 subnational locations, 1990-2021: a systematic analysis for the Global Burden of Disease Study 2021. *Lancet* 2024; **403:** 2100–32.

2 Ferrari AJ, Santomauro DF, Aali A, et al, and the GBD 2021 Diseases and Injuries Collaborators. Global incidence, prevalence, years lived with disability (YLDs), disability-adjusted life-years (DALYs), and healthy life expectancy (HALE) for 371 diseases and injuries in 204 countries and territories and 811 subnational locations, 1990-2021: a systematic analysis for the Global Burden of Disease Study 2021. *Lancet* 2024; **403:** 2133–61.

3 Dubois B, Hampel H, Feldman HH, et al, and the Proceedings of the Meeting of the International Working Group (IWG) and the American Alzheimer's Association on "The Preclinical State of AD"; July 23, 2015; Washington DC, USA. Preclinical Alzheimer's disease: Definition, natural history, and diagnostic criteria. *Alzheimers Dement* 2016; **12:** 292–323.

4 Livingston G, Huntley J, Liu KY, et al. Dementia prevention, intervention, and care: 2024 report of the Lancet standing Commission. *Lancet* 2024; **404:** 572–628.

5 Hahad O, Daiber A, Michal M, et al. Smoking and Neuropsychiatric Disease-Associations and Underlying Mechanisms. *Int J Mol Sci* 2021; **22:** 7272.

6 Almeida OP, Garrido GJ, Alfonso H, et al. 24-month effect of smoking cessation on cognitive function and brain structure in later life. *Neuroimage* 2011; **55:** 1480–89.

7 Noé-Díaz V, Salinas-Rivera E, Cruz-Pérez F, et al. Changes on executive functions before and after quitting smoking: Pilot study. *J Subst Use* 2018; **23:** 452–56.

8 Doolan DM, Froelicher ES. Smoking cessation interventions and older adults. *Prog Cardiovasc Nurs* 2008; **23:** 119–27.

9 Babb S, Malarcher A, Schauer G, Asman K, Jamal A. Quitting Smoking Among Adults - United States, 2000-2015. *MMWR Morb Mortal Wkly Rep* 2017; **65:** 1457–64.

10 Sonnega A, Faul JD, Ofstedal MB, Langa KM, Phillips JW, Weir DR. Cohort Profile: the Health and Retirement Study (HRS). *Int J Epidemiol* 2014; **43:** 576–85.

11 Steptoe A, Breeze E, Banks J, Nazroo J. Cohort profile: the English longitudinal study of ageing. *Int J Epidemiol* 2013; **42:** 1640–48.

12 Börsch-Supan A, Brandt M, Hunkler C, et al, and the SHARE Central Coordination Team. Data Resource Profile: the Survey of Health, Ageing and Retirement in Europe (SHARE). *Int J Epidemiol* 2013; **42:** 992–1001.

13 Payton NM, Marseglia A, Grande G, et al. Trajectories of cognitive decline and dementia development: A 12-year longitudinal study. *Alzheimers Dement* 2022.

14 Lamberty GJ, Kennedy CM, Flashman LA. Clinical utility of the CERAD word list memory test. *Appl Neuropsychol* 1995; **2:** 170–73.

15 Sebaldt R, Dalziel W, Massoud F, et al. Detection of cognitive impairment and dementia using the animal fluency test: the DECIDE study. *Can J Neurol Sci* 2009; **36:** 599–604.

16 Turvey CL, Wallace RB, Herzog R. A revised CES-D measure of depressive symptoms and a DSM-based measure of major depressive episodes in the elderly. *Int Psychogeriatr* 1999; **11:** 139–48.

17 Castro-Costa E, Dewey M, Stewart R, et al. Ascertaining late-life depressive symptoms in Europe: an evaluation of the survey version of the EURO-D scale in 10 nations. The SHARE project. *Int J Methods Psychiatr Res* 2008; **17:** 12–29.

18 Xue B, King M, Deindl C, Lacey R, Di Gessa G, McMunn A. Do Health and Well-Being Change Around the Transition to Informal Caring in Early Adulthood? A Longitudinal Comparison Between the United Kingdom and Germany. *J Adolesc Health* 2024; **74:** 885–91.

19 Lacey RE, Xue B, Di Gessa G, Lu W, McMunn A. Mental and physical health changes around transitions into unpaid caregiving in the UK: a longitudinal, propensity score analysis. *Lancet Public Health* 2024; **9:** e16–25.

20 Blackwell M, Iacus S, King G, Porro G. cem: Coarsened exact matching in Stata. *Stata J* 2009; **9:** 524–46.

21 Berrington A, Smith P, Sturgis P. An overview of methods for the analysis of panel data. 2006.

22 Wing C, Simon K, Bello-Gomez RA. Designing difference in difference studies: best practices for public health policy research. *Annu Rev Public Health* 2018; **39:** 453–69.

23 Oksanen T, Kivimäki M, Pentti J, Virtanen M, Klaukka T, Vahtera J. Self-report as an indicator of incident disease. *Ann Epidemiol* 2010; **20:** 547–54.

24 Singh-Manoux A, Kivimaki M, Glymour MM, et al. Timing of onset of cognitive decline: results from Whitehall II prospective cohort study. *BMJ* 2012; **344:** d7622.

25 Mons U, Schöttker B, Müller H, Kliegel M, Brenner H. History of lifetime smoking, smoking cessation and cognitive function in the elderly population. *Eur J Epidemiol* 2013; **28:** 823–31.

26 Deal JA, Power MC, Palta P, et al. Relationship of Cigarette Smoking and Time of Quitting with Incident Dementia and Cognitive Decline. *J Am Geriatr Soc* 2020; **68:** 337–45.

27 Gellert C, Schöttker B, Müller H, Holleczek B, Brenner H. Impact of smoking and quitting on cardiovascular outcomes and risk advancement periods among older adults. *Eur J Epidemiol* 2013; **28:** 649–58.

28 Knopman DS, Hershey L. Implications of the Approval of Lecanemab for Alzheimer Disease Patient Care: Incremental Step or Paradigm Shift? *Neurology* 2023; **101:** 610–20.

29 Jackson SE, McGowan JA, Ubhi HK, et al. Modelling continuous abstinence rates over time from clinical trials of pharmacological interventions for smoking cessation. *Addiction* 2019; **114:** 787–97.

30 Stopping the start: our new plan to create a smokefree generation. In: Department of Health & Social Care, editor. Online; 2023.