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Exposure to passive smoking and impairment in physical function in older people

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Availability of data and syntax for replication: The ELSA data are available for download from the UK Data Service website (https://discover.ukdataservice.ac.uk/catalogue/?sn=5050&type=Data%20catalogue). The syntax used in these analyses is available upon request from Paola Zaninotto (p.zaninotto@ucl.ac.uk).

To the Editor –

It is well documented that passive exposure to tobacco smoke (secondhand smoking) is a risk factor for cardiovascular disease.[1] Plausible mechanisms include plaque formation on the arterial wall resulting in decreased blood flow.[2] This disease process, plus other deleterious consequences of passive smoking – impaired lung and cognitive function[3] – may have implications for additional health outcomes, including physical capacity. In older adults, lower levels of indicators of physical capacity – walking speed, balance, and grip strength – appear to be linked to direct smoking,[4] but the impact of passive smoking, particularly when quantified objectively, has, to our knowledge, yet to be examined longitudinally.

The English Longitudinal Study of Ageing is an ongoing, open, prospective cohort study of a representative sample of men and women who were aged ≥50 years at baseline when living in private households in England.[5] Ethical approval for data collection was provided by the London Multicentre Research Ethics Committee. Study members had earlier participated in data collection in the Health Survey for England[6] (1998, 1999, 2001), from which they had been sampled, when salivary cotinine (ng/ml), a biomarker for recent exposure to secondhand smoke, was analyzed using gas chromatography (hp5890; Hewlett Packard, Palo Alto, USA) with a rapid liquid chromatography technique. A total of 6511 participants (3369 women) had data on salivary cotinine. In order to focus on study members whose only exposure to cigarette smoke was apparently indirect, we excluded from analyses 2170 who were self-declared current smokers plus 232 so-called deceivers who claimed to be ex-smokers or non-smokers but had salivary cotinine concentrations that belied this status (≥14.1 ng/ml).

At each of the six waves of data collection, respondents aged 60 years and over were asked to walk a distance of 8 feet (2.4 m) and back; speed was then calculated. During alternative waves, grip strength

(kg) was assessed during six trials (three per hand) using a hand-held dynamometer with the average of the maximum value on each hand used in our analyses.

Lower body strength was based on the capacity to rise from a chair with arms across the chest to a full standing position on five occasions for persons ≥70 years (ten occasions for persons <70 years). We evaluated static balance in three separate and progressively more difficult tests: in a feet side-by-side stand for 10 seconds (side-by-side stand); a preferred heel and toe side-by-side stand (semi-tandem) for 10 seconds; and a preferred heel in front of toe stand (full tandem) for 10 seconds if aged ≥70 years (30 seconds if aged <70 years). Failure on each test was denoted by an inability to fully complete the protocol. Covariates were captured using standard protocols.

A greater exposure to passive smoking, as indexed by a higher salivary cotinine level, was associated with a lower performance on tests of both gait speed and grip strength (Table). These effects were seen at all waves of data collection. People with higher levels of salivary cotinine were also somewhat more likely to fail to complete the balance and chair rise tests. When we modelled trajectories in each of the four physical function measures, however, there was no consistent association with cotinine. With the inevitable loss to follow-up, we tested if data imputation had an impact on our positive results for gait speed, an outcome which had the greatest degree of missing data. The same pattern of association remained.

Our main finding was that, in non-smokers, cotinine levels were modestly inversely related to performance on several tests of physical capacity. While our cotinine biomarker has the advantage of capturing all forms of exposure to passive smoking, some of which may be missed by self-report, a limitation is that its short half-life may mean it has less utility as an indicator of longer-term patterns.

References

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Table. Prospective relation of a unit increase in cotinine level with physical performance indicators in non-smokers in the English Longitudinal Study of Ageing

	Wave 1 (2002/3)	Wave 2 (2004/5)	Wave 3 (2006/7)	Wave 4 (2008/9)	Wave 5 (2010/1)	Wave 6 (2012/3)
Gait speed (m/s)						
Analytical sample	2478	2120	1850	1547	1368	1148
Beta coefficients	-0.013 (-0.021;-0.006)	-0.018 (-0.026;-0.009)	-0.018 (-0.026;-0.009)	-0.009 (-0.018;-0.001)	-0.013 (-0.024;-0.003)	-0.001 (-0.021; 0.001)
Grip strength (kg)						
Analytical sample		3352		2377		2024
Beta coefficients		-0.340 (-0.521; -0.158)		-0.301 (-0.531; -0.071)		-0.221 (-0.471; 0.028)
Chair rises (failure)						
Analytical sample		3046		2014		1753
Odds ratios		1.0 (0.9; 1.2)		1.2 (1.1; 1.4)		1.2 (0.9; 1.4)
Balance (failure)						
Analytical sample		3301		2320		1969
Odds ratios		1.1 (1.0; 1.2)		1.1 (1.0; 1.2)		1.0 (0.9; 1.1)

Effect estimates are adjusted for age, gender, social class, limiting longstanding illness, alcohol intake, and body mass index. Covariates were collected at baseline examination in the Health Survey for England. The relation of cotinine level with gait speed and grip strength was estimated using linear regression; the relation of cotinine level with the balance test and chair rises was summarised using logistic regression.