eTable 1. Unweighted multivariable-adjusted[a] difference in rate of cognitive change (over a 10-year interval) between current and never smokers, within age strata.

		Difference in rate of change: current smokers vs never smokers		
Age range	**N**	**Difference**	**SE[b]**	**95% confidence interval**
65 to 70 years	1083	-0.09	0.06	(-0.21, 0.03)
71 to 80 years	1717	-0.14	0.07	(-0.28, -0.05)
81 years and older	902	0.16	0.24	(-0.31, 0.63)

[a] Adjusted for age, sex, race, education, and alcohol consumption.

[b] SE: standard error.

eTable 2a. Sensitivity analyses: alternative attrition models and their fit to the data.

Attrition model		Overall fit: Bayesian information criterion (BIC)	Discrimination and calibration statistics: % discordant	*c* statistic	Hosmer-Lemeshow χ^2 test, *P* value
Selected model[a]	Death:	8095	21	0.79	0.5
	Non-death dropout:	5683	38	0.62	0.7
Variations on the selected model[b]					
Same covariates, but fitting a single censoring model, with no distinction between death and non-death dropout		10599	29	0.71	0.06
Use ordinal term for study cycle in place of indicator terms	Death:	8067	21	0.79	0.7
	Non-death dropout:	5736	42	0.57	0.7
Omit terms for social network score, Nagi disability score, and self-reported health	Death:	8384	24	0.76	0.3
	Non-death dropout:	5651	39	0.60	1.0
Add terms for baseline coronary heart disease and hypertension	Death:	8106	21	0.79	0.8
	Non-death dropout:	5697	38	0.61	0.4

eTable 2b. Sensitivity analyses: alternative attrition models, their weights, and the results of applying these weights to analyses of smoking and cognitive decline.[c]

Attrition model	Weights: Mean (SD)	Weights: Range	Change in cognitive score over 10 years among never smokers[d]	Difference in cognitive score change over 10 years: current smokers vs never smokers: Difference	SE[e]	95% CI[e]	Increase from unweighted result[f]	Excess years of cognitive aging among current smokers[g]
None	-	-	-0.53	-0.11	0.05	(-0.20, -0.02)	-	2.1
Selected model[a]								
Nonstabilized weights:	1.8 (2.4)	1.0-84.7	-0.70	-0.17	0.07	(-0.31, -0.02)	56%	2.4
Stabilized weights:	1.0 (0.3)	0.2-14.7	-0.82	-0.20	0.08	(-0.36, -0.04)	86%	2.5
Variations on the selected model[b]								
Same covariates, but fitting a single censoring model, with no distinction between death and non-death drop-out								
Nonstabilized weights:	1.8 (1.9)	1.0-46.0	-0.70	-0.16	0.07	(-0.29, -0.02)	44%	2.2
Stabilized weights:	1.0 (0.3)	0.3-12.5	-0.80	-0.16	0.07	(-0.29, -0.04)	52%	2.1
Use ordinal term for study cycle in place of indicator terms								
Nonstabilized weights:	1.8 (2.5)	1.0-85.1	-0.73	-0.17	0.07	(-0.32, -0.03)	60%	2.4
Stabilized weights:	1.0 (0.4)	0.2-16.5	-0.83	-0.22	0.09	(-0.40, -0.04)	102%	2.6
Omit terms for social network score, Nagi disability score, and self-reported health								
Nonstabilized weights:	1.8 (2.3)	1.0-90.1	-0.69	-0.14	0.07	(-0.27, -0.01)	30%	2.0
Stabilized weights:	1.0 (0.3)	0.3-9.8	-0.80	-0.19	0.06	(-0.31, -0.07)	74%	2.4
Add terms for baseline coronary heart disease and hypertension								
Nonstabilized weights:	1.8 (2.3)	1.0-89.9	-0.70	-0.16	0.07	(-0.30, -0.02)	49%	2.3
Stabilized weights:	1.0 (0.4)	0.2-13.9	-0.81	-0.20	0.08	(-0.36, -0.04)	85%	2.5

[a] Entailed two pooled logistic regression models, one predicting mortality and the other predicting non-death dropout conditional on not dying. Both included terms for baseline smoking status (current vs never), prior global cognitive score, baseline age (years), sex, African-American race, education (4 categories), prior social network score, prior alcohol intake, prior Nagi disability score, prior self-rated health, diabetes at baseline, interview cycle, and the cross-product between cycle and age.

[b] Results shown were not bootstrapped.

[c] All models of smoking and cognitive decline were adjusted for age, sex, race, education, and alcohol consumption.

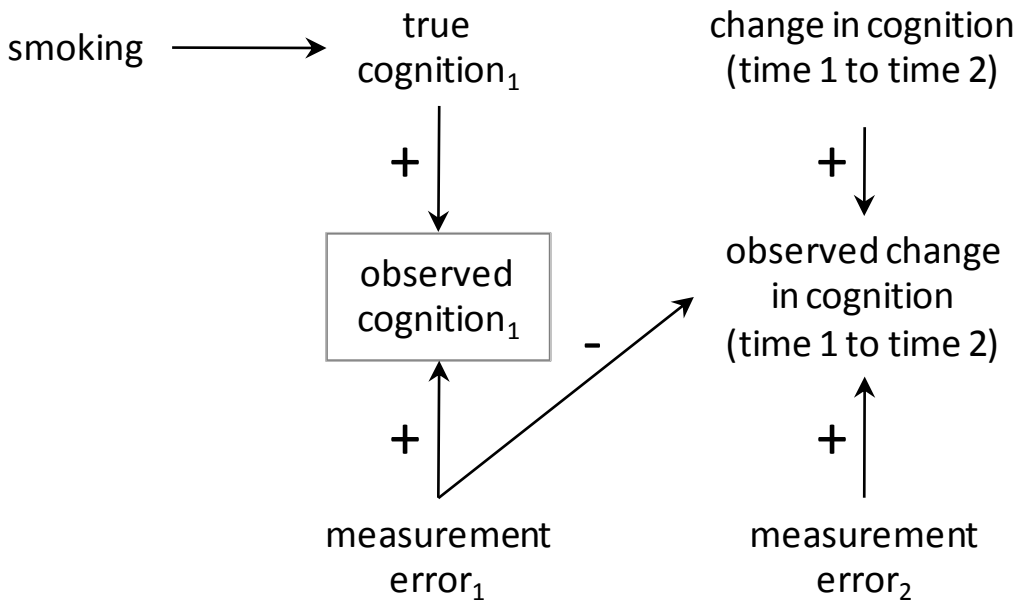
[d] Based on the model's parameter estimate for the "time" term, which is also the average rate of change among never smokers.

[d] SE: standard error; 95% CI: 95% confidence interval.

[f] Increase on an absolute scale, computed from estimates expressed to their nearest 0.001.

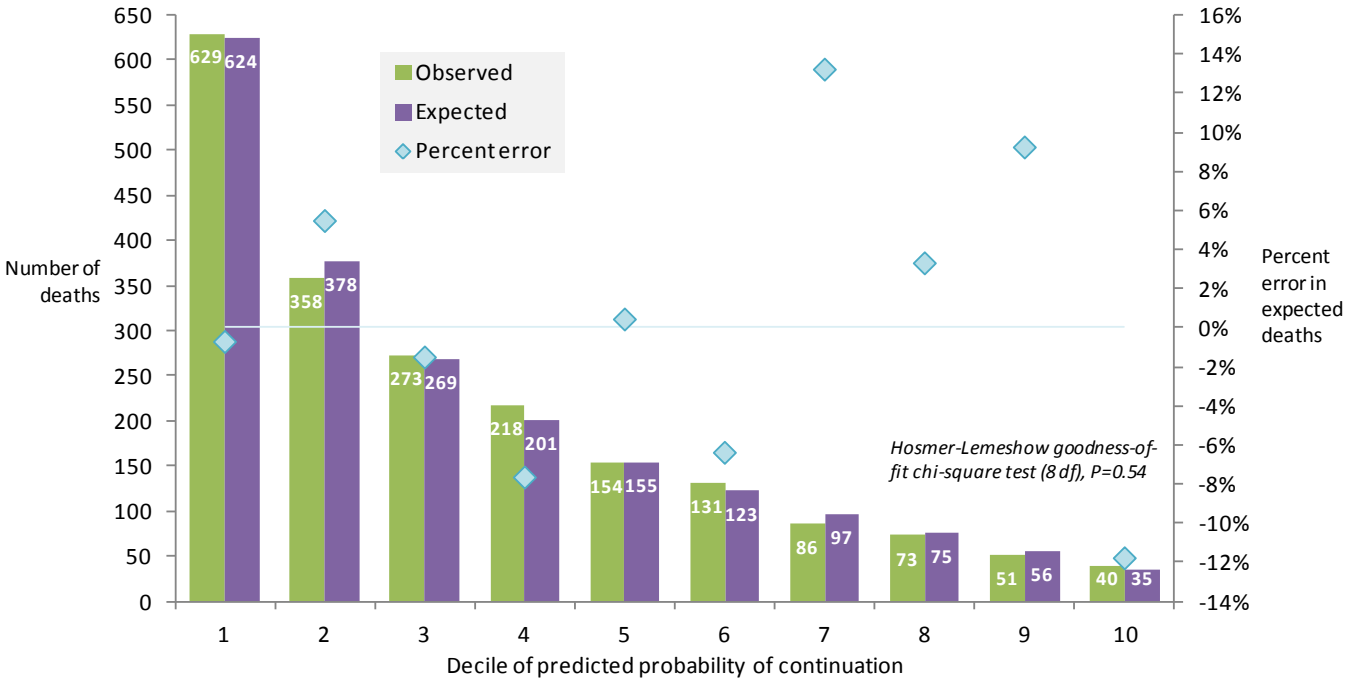
[g] Assuming that the rate of cognitive score change among never smokers represents “smoking-free cognitive aging,” we estimated the excess years of cognitive aging during a chronological period of 10 years among current smokers by dividing the difference between smokers’ and never smokers’ change in cognitive score over 10 years by the annual rate of change among never smokers. Estimates are specific to persons with reference group characteristics (except for smoking), specifically, 75-year-old white females with 9-12 years of education and no alcohol consumption.

eFigure 1. Directed acyclic graph (DAG) depicting bias in analyses of cognitive decline that are adjusted for baseline cognitive score. This DAG illustrates how adjustment for baseline cognitive score in analyses of cognitive change may yield biased findings. Measurements of cognition are subject to errors due to natural fluctuations in a participant's performance and errors associated with the instrument itself. As such, observed cognition is the sum of true underlying cognition and measurement error. Further, for any given cognitive score, participants with higher true cognition will tend to have more negative errors in their measured cognition. Through a simple mathematical derivation, it can be shown that observed change in cognition is worse (i.e., more negative) with larger error in the measurement of cognition at time 1 (e.g., persons with large positive errors at baseline will appear to decline more). Thus, conditioning on observed cognition at time 1 induces a potentially large inverse association between true cognition and measurement error at time 1 and, consequently, a strong upward bias in the association between true cognition at time 1 and observed change. If smoking is associated with baseline cognition, then the spuriously positive association between true baseline cognition and observed change magnifies the association between smoking and cognitive change in analyses that are baseline-adjusted. For example, if smokers have worse cognition at baseline, baseline-adjusted analyses will yield results that exaggerate smoking's adverse effect on cognitive change. For more detail, see Glymour MM, Weuve J, Berkman LF, Kawachi I, Robins JM. When is baseline adjustment useful in analyses of change? An example with education and cognitive change. *Am J Epidemiol* 2005;162:267-278.

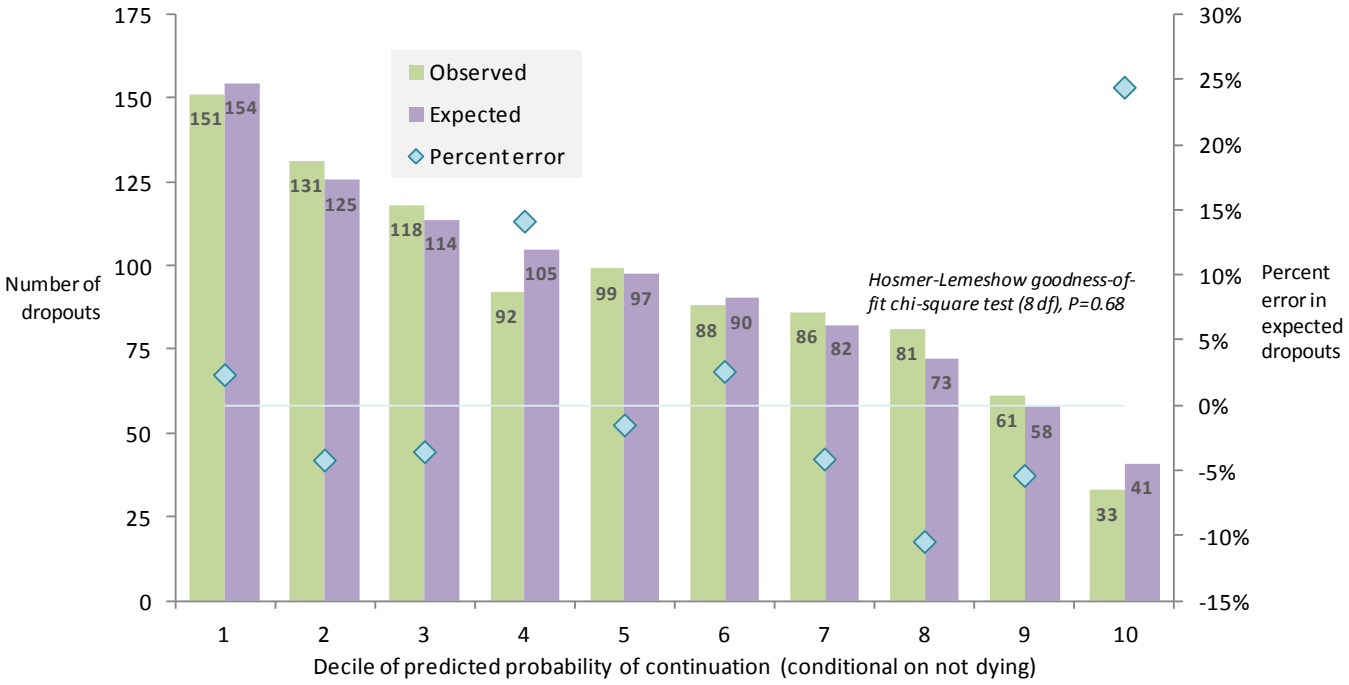


eFigure 2. Hosmer-Lemoshow goodness of fit assessment of attrition models.

Observed and expected number of deaths and percent error in expected deaths, by decile of predicted probability of continuation in the study (from mortality-specific attrition model).



Observed and expected number of non-death drop-outs and percent error in expected drop-outs, by decile of predicted probability of continuation in the study (from non-death dropout attrition model).



eFigure 3. Directed acyclic graph (DAG) depicting causal structure underlying attrition-related selection bias in the relation of smoking to cognitive decline. This DAG shows how attrition may bias findings on smoking and cognitive decline in the presence of an unmeasured genotype that reduces the risk of cognitive decline and extends survival and continued study participation. Conventional unweighted analyses of follow-up data are restricted to the group of participants who survive and continue in the study, a form of conditioning indicated by the box around survival/continuation. Continuing survivors who smoke will be more likely to have the efficient detoxifier genotypes, and the restriction to continuing survivors induces an downward bias in the association between smoking and cognitive decline, resulting in underestimates of harm or overestimates of protection. For an introduction to DAGs, see Glymour MM, Greenland S. Chapter 12: Causal diagrams. In: Rothman KJ, Greenland S, Lash TL, eds. *Modern Epidemiology, Third Edition*. New York: Wolters Kluwer, 2008: 183-209.