

Table: 48P Mendelian randomisation estimates of the associations between education attainment and risk of lung cancer overall and histologic types

Outcome	IVW method		MR-Egger		Weighted median method	
	OR (95% CI)	P value	OR (95% CI)	P value	OR (95% CI)	P value
Lung cancer overall	0.48 (0.34-0.66)	1.02e-05*	0.61 (0.12-3.17)	0.56	0.52 (0.35-0.77)	1.08e-03*
Adenocarcinoma	0.64 (0.41-1.00)	4.97e-02*	0.89 (0.09-8.48)	0.92	0.59 (0.32-1.08)	8.94e-02
Squamous cell carcinoma	0.41 (0.27-0.62)	2.57e-05*	0.32 (0.04-2.63)	0.29	0.55 (0.30-1.01)	5.24e-02

*: P value < 0.05; IVW: inverse-variance weighted; OR: odds ratio; CI: confidence interval.

48P Education and lung cancer: A Mendelian randomisation study

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Background: Education has been shown to be inversely associated with the incidence of lung cancer at several conventional observational studies. However, this association may be biased owing to the methodological limitations of traditional observational study-confounding, reverse causation, and measurement error. Therefore, we aimed to investigate whether more years spent in education is causally associated with risk of lung cancer through a two sample mendelian randomisation study.

Methods: The main analysis used publicly available genetic summary data from two large consortiums (International Lung Cancer Consortium (ILCCO) and Social Science Genetic Association Consortium (SSGAC)). Genetic variants used as instrumental variables for lung cancer and years of education were derived from two large genome wide association studies: ILCCO and SSGAC, respectively. Finally, genetic data from three additional consortia (TAG, GLGC, GIANT) were analyzed to investigate whether longer education can causally alter the common lung cancer risk factors. The exposure was the genetic predisposition to higher levels of education, measured by 73 SNPs from SSGAC. The primary outcome was the risk of lung cancer (11348 events in ILCCO). Secondary outcomes based on different histologic subtypes were also examined.

Results: Genetic predisposition towards 3.6 years of additional education was associated with a 52% lower risk of lung (odds ratio 0.48, 95% confidence interval 0.34 to 0.66; $p = 1.02 \times 10^{-5}$). Sensitivity analyses were consistent with a causal interpretation in which major bias from genetic pleiotropy was unlikely. The Mendelian randomisation assumptions did not seem to be violated. Genetic predisposition towards longer education was additionally associated with less smoking, lower body mass index, and a favorable blood lipid profile.

Conclusions: Our present mendelian randomisation study provided strong evidence to support that higher education attainment plays a causal role in lowering the risk of lung cancer. Furthermore, more work is needed to elucidate the potential mechanisms which mediate the association between education and lung cancer.

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