

The association between education and premature mortality in the Chinese population: a 10-year cohort study

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Summary

Background Recent studies have shown significant associations between education and premature mortality. However, the relationship differs across countries. We aimed to present the latest evidence on the educational inequalities in premature mortality in the Chinese population.

Methods We linked two databases, to establish a population-based, ten-year cohort spanning 2010 to 2020. Cox proportional hazard regression analyses adjusting for age, sex and urbanicity were conducted for all-cause mortality, and competing risk models were fitted for cause-specific mortality. We calculated population attributable fraction (PAF) using the hazard ratios (HRs) obtained by regression analyses. Additionally, we fitted models adjusting for risk factors and investigated the mediating effect of income, smoking, alcohol consumption and diets.

Findings Compared with individuals with upper secondary and above education, the HR for premature all-cause mortality for those with less than primary education was 1.93 (95% CI: 1.72–2.19). The HRs were the highest for deaths from respiratory diseases (HR = 3.09, 95% CI 1.82–5.27). The excess risk of premature mortality associated with low education was higher among women and urban population. The association of education remained significant after accounting for risk factors, and income was the main mediator, which accounted for 23.0% of mediation in men and 11.1% in women.

Interpretation The study's findings support the increased risk of premature mortality associated with low education, particularly in women and urban populations. The considerable number of deaths attributed to educational inequality underscores the necessity for more effective and targeted public health interventions.

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Keywords: Education; Premature mortality; Cohort study

Introduction

Education is a key social determinant of health. Many research have highlighted the salient relationship between education and health outcomes including life expectancy, healthy aging, cardio-cerebrovascular health, cancer as well as exposure to risk factors such as obesity, substance abuse, and lifestyle factors.^{1–8} In recent years, several large-scale cohort studies^{9–14} have demonstrated the impact of education on all-cause and cause-specific mortality at the population level. Although there is a

consistent negative association between education and all-cause, as well as cause-specific mortality, the magnitude of this association varies across countries depending on the epidemiological profiles and risk factors trends. The cross-countries variations underscore the fact that the relationship between education and mortality is not uniform. Country-specific examination of the connection is valuable.

Education has historically been a policy priority in China, acting as a key tool in social transformation and

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Research in context

Evidence before this study

A literature search was conducted in PubMed for studies published between January 1, 2005 and November 3, 2023 using the following search string: (“education” [Title] OR “educational” [Title] OR “socioeconomic status” [Title] OR “social determinants” [Title])) AND (mortality [MeSH]). Studies related to health education or regional socioeconomic factors were excluded.

The association between lower educational attainment and increased mortality was observed in studies in Cuba, England, Canada, Cyprus, and the US. While the strength of the association between education and all-cause mortality were comparable across countries, considerable variations were observed in cause-specific mortalities. And there have been few population-level longitudinal studies on the relationship between education and premature all-cause and cause-specific mortality in China.

Added value of this study

This study utilized national data from the China Chronic Disease and Risk Factor Surveillance (CCDRFS) and the National Mortality Surveillance System to establish a 10-year cohort. In addition to understanding the impact of education on premature all-cause mortality in the Chinese population, the analyses of cause-specific mortality revealed the varying risks of specific causes of death associated with low education

levels. We also calculated the population attributable fraction (PAF) to quantify the impact of low educational attainment on health and found that PAF could be as high as 57% depending on the cause, which showed the considerable educational inequality in premature mortality. By leveraging the wealth of sociodemographic and risk factors details from CCDRFS, our mediation analyses provided insights into the roles other factors played in mediating the relationship between education and premature mortality, and further highlighted the unique influence exerted by education.

Implications of all the available evidence

Consistent with previous studies, the current findings confirmed that education had a significant impact on all-cause premature mortality in the Chinese population. However, variations exist in cause-specific mortality. The risk of deaths from respiratory diseases was most strongly correlated with education level. Substantial sex differences were observed with education having a stronger impact on women. With nearly a quarter of the population still not finishing lower secondary education in China, there were a large number of deaths attributed to educational inequality. There is a need to continue the effort to provide universal education. Meanwhile, it is essential to expand public health intervention to promote lifestyle modifications and mitigate lifestyle-associated risks.

modernization. Quality and access to education have improved substantially in the past few decades.¹⁵ According to the Chinese Ministry of Education, the gross enrolment rate of upper secondary schools in China rose from 1.1% in 1949 to 88.8% in 2018. However, substantial educational inequality persists. Data from China’s 2020 census indicated that while 76.0% of the population aged 30 to 69 completed lower secondary school education, only 31.5% completed upper secondary school education. Moreover, there are considerable sex, regional, and urban-rural gaps in education across China.¹⁶

To date, there have been few comprehensive population-level longitudinal studies that examined the relationship between education and cause-specific premature mortality in China. A previous study explored the correlation between education and mortality at the national and regional levels.¹⁷ However, due to limitations of census data, the study fell short at evaluating cause-specific mortality and considering mediating effect of risk factors. A recent study based on the China-HEART project researched the association between education and all-cause as well as cardio-cerebrovascular disease-specific mortality.¹⁸ Despite the large national sample size, the median follow-up was only 3.5 years. In another national study, educational effect on health conditions was examined using a 7-year cohort.¹⁹

However, the outcome variables were based primarily on self-rated health measures and failed to address actual health outcomes and the occurrence of premature mortality.

In this study, we examined the relationship between education and premature all-cause and cause-specific mortality using a 10-year, population-based and nationwide cohort, to provide robust quantification of educational inequalities in premature mortality specific to the Chinese context.

Methods

Study design and participants

We used data from the China Chronic Disease and Risk Factor Surveillance (CCDRFS) conducted in 2010 and linked the individual death records to the National Mortality Surveillance System (NMSS). The CCDRFS is an ongoing national disease surveillance survey conducted by the National Centre for Chronic and Non-communicable Disease Control and Prevention. Details of the surveillance have been described previously.²⁰ Briefly, the CCDRFS 2010 was carried out at 162 disease surveillance points (DSP) covering all 31 provinces, autonomous regions, and municipalities directly governed by the central government of China. Evaluation showed that the selected DSPs had good

representativeness both in urban and rural areas.²¹ The participants were selected using a multi-stage stratified clustering random sampling method. In this survey, trained personnel reported sampling information, conducted face-to-face questionnaire interviews and physical measurements, collected blood and urine samples, and performed on-site haemoglobin tests and site-centralized blood glucose and lipids tests. The surveillance follows strict implementation and quality control protocol, providing information over time on risk factors and disease prevalence. The CCDRFS study was approved by the institutional review board of China CDC (approval number 201010). All participants consented in writing.

On the other hand, the NMSS is a comprehensive registry capturing nationwide death reports. It consists of over 605 Disease Surveillance Points (DSPs), located in all 31 provinces, municipalities, and autonomous regions, covering 323.8 million residents, approximately 24.3% of the total population of China. Additionally, the NMSS also includes a system for collecting death records from other regions of China, allowing deaths of most participants who migrate to other areas to be reported. The DSPs where CCDRFS2010 was conducted were consistent with NMSS, enabling the linkage of death outcomes for more than 90% of participants. Following the linkage, we confirmed and supplemented the death outcomes by contacting local collaborators. The registry records contain information such as death status, date of death, and the underlying causes of death encoded by ICD-10 codes. Details of NMSS can be found elsewhere.²²

In this study, we selected individuals aged 30 to 69 from the 2010 CCDRFS surveillance data and obtained their baseline information in 2010. We excluded participants younger than 30 years old because they were at the stage of education, job searching and marriage, during which socioeconomic factors like education, marital status and income were unstable. Additionally, mortality among this group was very low, particularly for cause-specific mortality. We identified their premature death records until 2020 from NMSS records, and formed a 10-year population-based cohort. Individuals' survival period concluded upon their death, and those who reached 70 years of age or were still alive in 2020 were censored.

Education

Individual education attainment data was obtained from the CCDRFS. In the CCDRFS questionnaire, respondents were asked about their highest education level achieved. Responses were classified into eight categories from “no formal school education” to “post-graduate and above.” There was no missingness for education so we didn't perform imputation. To ensure sufficient sample sizes in each segment and improved analytical robustness, we redefined these categories into

four segments: “less than primary school,” (failed to finish primary school) “primary school,” (finish primary school) “lower secondary school,” (finish middle school) and “upper secondary and above” (finish high school). [Supplementary Table S1](#) compared our categories with education systems of several countries. The “upper secondary and above” category served as the reference group for statistical comparisons.

Outcomes

The primary outcome of interest was premature mortality, which was defined as deaths occurring between the ages of 30 and 69 according to the World Health Organization criteria.²³ We set the upper age limit at 70 because comorbidity and misreporting tend to increase in population over 70 years old, which could decrease the reliability of death records. Causes of premature deaths were divided into cardio-cerebrovascular diseases (I21–I25, I60–I69), cancers (C00–C97), respiratory diseases (J00–J06, J10–J18, J20–J22, J30–J98), and other causes, based on ICD-10 codes ([Supplementary Table S2](#)). Death records from 2010 to 2020 were retrieved from the NMSS and linked to the CCDRFS database using personal details including unique ID numbers, or names, sex, residential addresses and other identifiers when ID numbers were incorrect.

Covariates

Covariates included age, sex, urban or rural residence, marital status, income, smoking status, alcohol consumption, body mass index (BMI), physical activity, diet, blood pressure, blood lipids, and blood glucose levels. No imputation was performed except for income because the missingness of each covariate was less than 5% except for income. The missingness of income was 14% so we performed multiple imputation using fully conditional specification method, forming 5 imputation sets. Income was defined as total household income divided by family size and was categorized into four levels by quintile (<3000, <7000, <13,000 and ≥13,000 RMB per year). Smoking status was characterized as “current smoking, everyday”, “current smoking, not everyday”, “past smoking” and “never smoking”. Alcohol consumption was characterized as no drinking, light drinking (less than two days a week) and frequent drinking (more than three days a week). BMI was classified into four levels according to the guidelines,²⁴ which were underweight (<18.5 kg/m²), normal (18.5 to <24.0 kg/m²), overweight (24.0 to <28.0 kg/m²) and obesity (≥28.0 kg/m²). The metabolic equivalent of physical activities (METs.h/w)²⁵ was calculated as the sum of time spent in various physical activities per week multiplied by corresponding metabolic equivalent, and was then divided into three levels: low (<260 METs.h/w), moderate (260 to <310 METs.h/w) and high (≥310 METs.h/w). Five dietary components were quantified with reference to the Dietary Guidelines for

Chinese Residents.²⁶ Red meat consumption over 100 g per day, salt over 6 g per day or oil over 30 g per day was regarded as excessive intake; vegetable consumption below 300 g per day or fruit below 200 g per day was regarded as insufficient intake. Blood pressure, blood lipid, and blood glucose were categorized according to conventional standards. Systolic blood pressure above 140 mmHg or diastolic blood pressure above 90 mmHg was regarded as hypertension. Any abnormality of total cholesterol (TC \geq 6.22 mmol/L), high-density lipoprotein cholesterol (HDL \leq 1.04 mmol/L), low-density lipoprotein cholesterol (LDL \geq 4.14 mmol/L), and triglycerides (TG \geq 2.26 mmol/L) was considered dyslipidemia. Based on fasting blood glucose (FBG) and postchallenge blood glucose (PBG) values, pre-diabetes was defined as $6.1 \leq$ FBG $<$ 7.0 mmol/L or $7.8 \leq$ PBG $<$ 11.1 mmol/L, and diabetes was defined as FBG \geq 7.0 mmol/L or PBG \geq 11.1 mmol/L.

Statistical analyses

Descriptive analyses were conducted to understand the general baseline characteristics of the cohort by level of education. For continuous variables, the mean and standard deviation were calculated. For categorical variables, percentages were calculated. All estimates, except for mean age, were age-standardized using the age distribution of all participants.

To examine the association between education level and premature all-cause and cause-specific mortality, we plotted the cumulative incidence function (CIF) plot, performed log-rank tests, and fitted Cox proportional hazards regression models for all-cause mortality and competing risk models for cause-specific mortality. An initial model was fitted to estimate hazard ratios (HRs) and 95% confidence interval (CI) by sex, adjusting for age and urbanicity. An alternative model was also fitted to examine HRs by urbanicity adjusting for age and sex. In order to test the statistical significance of the differences between sex and urbanicity, we introduced the interact terms in models. Using the HRs derived from the initial model, we calculated the population attributable fraction (PAF) of education. PAF represented the expected percentage reduction in premature mortality if the entire population experienced the mortality rates of the group with the highest level of education. PAF was calculated by sex using the formula below²⁷:

$$PAF = \frac{\sum_{i=1}^n P_i(HR_i - 1)}{\sum_{i=1}^n P_i(HR_i - 1) + 1}$$

where P_i was the proportion of the population with education level i , obtained from the sixth national population census in China, 2020. HR_i was the hazard ratio of the population with education level i compared to those with upper secondary and above education level. To estimate the number of premature deaths attributable to education inequality and avoid the effect of

COVID-19, we applied the PAF to all deaths between the ages of 30 and 69 years in China for the year 2019.

To study the association between education and mortality independent of risk factors, we fitted a series of models adjusting for marital status, income, BMI, smoking, alcohol consumption, diet, daily physical activities, blood pressure, blood lipids and blood glucose. We explored mediating effects of income, smoking, alcohol consumption and diets, which showed significant mediating effects between education and mortality. We conducted mediation analyses using the % MEDIANTE SAS macro (<https://ysph.yale.edu/cmips/research/software/mediate>).

We performed a series of sensitivity analyses. Firstly, considering the fact that many individuals had major chronic diseases at baseline, which might have resulted in changes in their lifestyle and increased mortality risk. We performed analyses excluding individuals with pre-existing myocardial infarction, stroke, COPD, asthma and cancers. Secondly, we fitted models using the death data from 2010 to 2019 to avoid the effect of COVID-19. Third, we included individuals aged 20 to 70 and reanalyzed the data. We followed STROBE checklist to display our study. All statistical analyses were performed using SAS (version 9.4). Graphical displays were created using R software (version 4.1.2). Significance tests were carried out with a significance level set at 0.05.

Role of the funding source

The funder of the study had no role in study design, data collection, data analyses, data interpretation, or writing of the report. The corresponding author had full access to all data in the study and had final responsibility for the decision to submit for publication.

Results

Baseline statistic

There were 98,658 participants in CDRFS2010. A total of 77,789 participants between 30 and 69 years old were included in this study. The average age of the study population was 48.52 (\pm 10.43), among which 34,945 (44.92%) were men and 42,844 (55.08%) were women. There were 19,349 (24.87%) individuals with less than primary education, 16,430 (21.12%) with primary education, 24,786 (31.86%) with lower secondary education and 17,224 (22.14%) with upper secondary and above education. As shown in [Table 1](#), among individuals with less than primary education, 70.3% was women. The mean age of individuals with upper secondary and above education was the lowest. And younger participants had greater proportion of upper secondary and above education ([Supplementary Fig. S1](#)). The proportion of individuals in the highest income quintiles increased steadily by educational attainment level. 49.2% of respondents in the upper secondary and above education level were in highest-income category

Baseline	Less than primary	Primary	Lower secondary	Upper secondary and above
Number of participants (n)	19,349	16,430	24,786	17,224
Proportion in total population (%)	24.87	21.12	31.86	22.14
Years of follow-up (person-year)	162,662	146,193	232,425	163,895
Men (n, %)	5748 (29.7)	7438 (45.3)	12,825 (51.7)	8934 (51.9)
Age (mean,s)	53.2 (10.2)	49.4 (10.6)	46.3 (9.8)	45.7 (9.4)
Urban (%)	23.8	31.2	49.8	76.8
Marital (%)				
Unmarried	3.1	1.7	1.4	2.0
Married	88.9	92.4	92.8	91.7
Divorced	0.9	1.1	1.8	3.0
Widowed	7.0	4.5	3.8	3.0
Others	0.2	0.20	0.3	0.4
Income per year (%)				
<3000 RMB	38.1	27.1	16.5	6.0
3000 to <7000 RMB	33.0	33.6	29.6	15.5
7000 to <13,000 RMB	18.7	23.9	28.4	27.9
≥13,000 RMB	10.2	15.4	25.5	50.7
Smoking status (%)				
Current smoking, everyday	18.0	25.7	29.0	24.7
Current smoking, not everyday	1.8	2.3	2.9	3.5
Past smoking	4.2	5.9	6.5	6.6
Never smoking	75.9	66.1	61.7	65.2
Alcohol consumption (%)				
Frequent drinking	11.6	14.5	16.0	15.1
Light drinking	15.7	16.5	21.3	30.9
No drinking	72.7	69.0	62.7	54.1
Physical activity (%)				
Low	16.3	19.9	25.3	41.1
Medium	41.3	41.9	44.4	43.4
High	42.4	38.2	30.3	15.5
BMI (%)				
Low weight	4.1	3.1	2.2	1.8
Normal	54.3	50.2	46.3	43.1
Overweight	24.3	26.9	29.6	31.8
Obesity	17.3	19.8	21.9	23.3
Diets				
Excessive intake of red meat (%)	22.2	23.2	25.1	31.3
Insufficient intake of vegetable (%)	48.6	43.1	40.9	39.2
Insufficient intake of fruit (%)	93.3	90.7	86.8	80.9
Excessive intake of salt (%)	81.9	78.4	73.4	65.4
Excessive intake of oil (%)	72.1	72.1	71.8	68.9
Chronic conditions				
Hypertension (%)	36.6	36.2	37.4	32.9
Dyslipidemia (%)	40.6	44.9	48.2	51.9
Pre-diabetes (%)	16.7	18.4	18.6	18.6
Diabetes (%)	7.8	9.1	10.8	11.4

All characteristics were adjusted by age except number of participants and age.

Table 1: Baseline characteristics of participants of four education levels.

compared to only 10.0% in the less than primary level (Supplementary Fig. S2).

The prevalence of smoking was slightly lower among individuals with higher levels of education attainment

(Table 1). The proportion of individuals with high levels of physical activity was 42.4% in individuals with less than primary education, compared to 15.5% in individuals with upper secondary and above education.

The proportion of overweight and obesity was 41.6% in individuals with less than primary education, and 55.1% in individuals with upper secondary and above education. Same trends were also shown in both men and women (Supplementary Fig. S3 and Table S3). Further analyses showed similar baseline trends in urban and rural population, (Supplementary Table S4).

Main analysis results

A total of 3082 premature deaths was observed within the 705,175 person-years of follow-up. The average follow-up duration was 9.07 years. The age-standardised all-cause premature mortality was negatively associated with education level, with higher mortality rate among those with lower level of education attainment (Fig. 1, Supplementary Table S5).

The CIF plot showed a significant negative association between education and mortality (Supplementary Fig. S4). The p value of log-rank test was less than 0.001. According to the similar increasing trends of different education levels, the proportional hazards assumption wasn't violated. The HR for premature all-cause mortality for individuals with less than primary education compared to those with upper secondary and above education was 1.93 (95% CI: 1.72–2.19). For cause-specific mortality, the HRs were 1.60 (1.28–2.01) for cardio-cerebrovascular diseases, 1.53 (1.24–1.90) for cancer, 3.09 (1.82–5.27) for respiratory and 2.76 (2.15–3.54) for all other causes (Fig. 2).

Variabilities were observed between men and women (Figs. 2 and 3). The all-cause mortality rate of men with less than primary education was 1.78 (95% CI: 1.52–2.09) times higher than those with upper secondary and above education, while 2.15 (95% CI: 1.74–2.67) for women. The p value of the interaction between education and sex was 0.030, which showed statistical significance of difference between men and women.

HRs for cause-specific mortality were similar. The largest differences were observed in respiratory-related mortality. Comparing individuals with less than primary education to those with upper secondary and above education, the HRs were 2.65 (1.44–4.88) among men and 5.26 (1.63–17.02) among women.

The effect of education on premature mortality also varied between urban and rural settings, with a more positive impact observed in urban areas (Fig. 2). While significant associations were found between education and each cause-specific mortality in urban areas, significant associations were only detected for respiratory and other causes-related mortalities in rural settings. The p value of the interaction between education and urban-rural was 0.018, which showed statistical significance of difference between urban and rural population.

Education-attributable deaths

Fig. 4 showed the PAF in percentage and the equivalent number of deaths in 2019. The PAF of low education on

all-cause premature mortality was 20.8% among men and 28.5% among women. It was equivalent to 457,979 premature deaths in men and 282,267 in women across the country which could have reduced if all people experienced the same mortality as those completing upper secondary education. With respect to cause-specific mortality, the PAF was relatively similar across causes among men, with the highest PAF observed in association with other causes (28.7%). Among women, PAF differed across causes, with the highest PAF observed in association with respiratory deaths (56.9%).

Effect of risk factors

After adjusting for income and marital status, for all-cause mortality, the HR of individuals with less than primary education was 1.70 (95% CI 1.49–1.94), slightly lower than unadjusted results. After adjusting for behaviour and metabolic factors, the HR increased to 1.71 (95% CI 1.49–1.95) (Fig. 5). Together, income, smoking, alcohol consumption and intake of vegetables and fruits accounted for 28.0% of the association between education and premature mortality in men, and 14.7% of the association in women. Income stood out as one of the most critical factors accounting for 23.0% of the relationship in men and 11.1% in women (Supplementary Table S6).

Sensitivity analysis results

All the sensitivity analyses showed similar trends with the main analyses after excluding individuals with pre-existing conditions (Supplementary Table S7), including individuals aged 20–70 (Supplementary Table S8), or using death data from 2010 to 2019 (Supplementary Table S9). All the results showed increased premature mortality risk associated with low education. The HRs were higher for deaths caused by respiratory diseases and other causes.

Discussion

In this retrospective cohort study, we utilized linked data from CCDRFS and NMSS, following 77,789 participants for a decade to examine the relationship between education attainment and premature mortality. Our results showed a strong inverse connection between education and all-cause premature mortality. For those with the lowest education, the hazard ratios were estimated to be 1.78 among men and 2.15 among women compared with individuals with the highest education, and 20.8% of premature all-cause deaths among men and 28.5% among women could be attributed to low education.

Our results were similar to previous studies in Cuba,¹⁰ US²⁸ and Italy.²⁹ Stephanie Ross et al.¹⁰ also found that the death risk of population with the lowest education level was 1.79 times that of those with the highest education, and the effect among women was more pronounced. Gianfranco²⁸ et al. found that

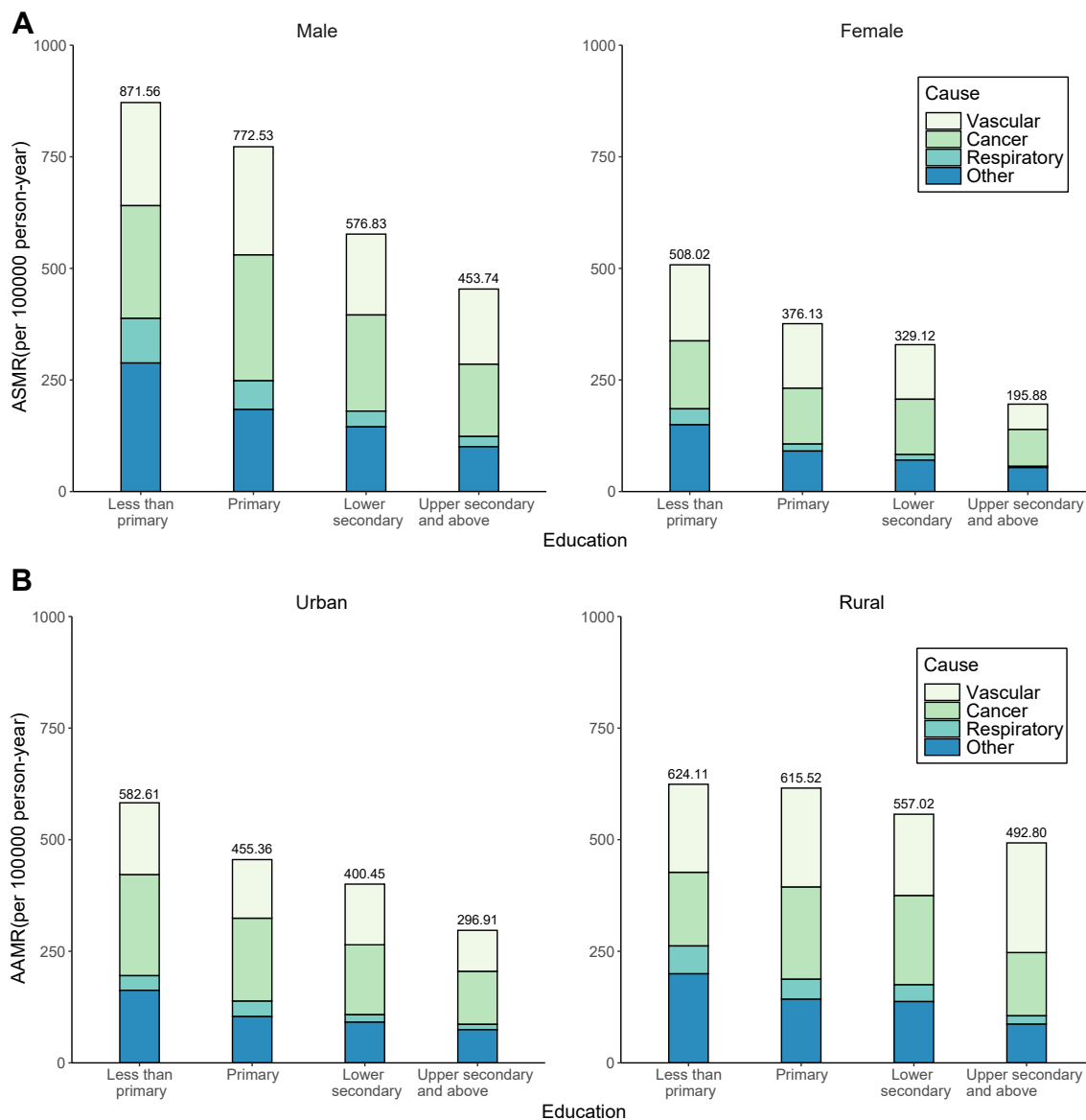


Fig. 1: Age-standardised all-cause and cause-specific mortality rates per 100,000 person-year across educational levels, by sex (A) and by urban-rural setting (B). ASMR: age-standardised mortality rates. The number represents the age-standardised mortality rates of all-cause death.

compared with individuals not completing primary school, the cancer mortality of those complete university had a HR of 0.57 among men and 0.84 among women. Najah²⁹ et al. showed compared to population with college and above education, the HR of those who didn't finish high school was 2.03. Our results were same or slightly higher than those reported in these studies.

The current study is unique in several aspects. Firstly, it provides the most up-to-date insights into the relationship between education and premature all-cause and cause-specific mortality within the Chinese context. The findings demonstrate the significant role education

plays in shaping China's current epidemiological landscape. Secondly, by merging information from two large national database, we conducted comprehensive, multivariate, and nationwide analyses. Thirdly, using the baseline characteristics data, we conducted specific sub-segment analyses to evaluate disparities between sexes and between urban-rural settings. The results expose the differential impact of education on premature mortality based on sex and location. Fourthly, the use of NMSS allowed us to gather granular data to assess cause-specific mortality, which has not been examined in prior studies in China. Finally, the large

Education	Total		Sex		Residence	
	Death(n)	HR(95%CI)	Male HR(95%CI)	Female HR(95%CI)	Urban HR(95%CI)	Rural HR(95%CI)
All causes						
Less than primary	1082	1.93(1.70,2.19)	1.78(1.52,2.09)	2.15(1.74,2.67)	2.26(1.89,2.70)	1.63(1.36,1.97)
Primary	745	1.59(1.40,1.80)	1.59(1.36,1.85)	1.65(1.32,2.07)	1.63(1.35,1.96)	1.41(1.17,1.71)
Lower secondary	851	1.35(1.20,1.52)	1.31(1.14,1.51)	1.46(1.18,1.82)	1.46(1.25,1.71)	1.18(0.97,1.42)
Upper secondary and above	404	1.00	1.00	1.00	1.00	1.00
Vascular						
Less than primary	349	1.60(1.28,2.01)	1.32(0.99,1.77)	2.23(1.49,3.35)	1.99(1.45,2.75)	1.29(0.94,1.77)
Primary	251	1.48(1.19,1.86)	1.28(0.97,1.69)	2.12(1.41,3.19)	1.38(0.98,1.96)	1.32(0.96,1.82)
Lower secondary	261	1.25(1.01,1.55)	1.14(0.88,1.48)	1.60(1.06,2.41)	1.48(1.11,1.97)	0.99(0.72,1.38)
Upper secondary and above	128	1.00	1.00	1.00	1.00	1.00
Cancer						
Less than primary	329	1.53(1.24,1.90)	1.47(1.11,1.95)	1.54(1.10,2.15)	2.01(1.51,2.68)	1.16(0.85,1.59)
Primary	263	1.49(1.20,1.83)	1.65(1.27,2.15)	1.23(0.86,1.76)	1.59(1.18,2.14)	1.23(0.90,1.68)
Lower secondary	315	1.32(1.09,1.61)	1.34(1.05,1.71)	1.30(0.93,1.80)	1.39(1.08,1.78)	1.13(0.83,1.55)
Upper secondary and above	157	1.00	1.00	1.00	1.00	1.00
Respiratory						
Less than primary	94	3.09(1.82,5.27)	2.65(1.44,4.88)	5.26(1.63,17.02)	3.59(1.63,7.90)	2.85(1.30,6.23)
Primary	47	1.80(1.03,3.15)	1.63(0.87,3.05)	2.50(0.70,8.90)	2.27(1.00,5.16)	1.64(0.73,3.69)
Lower secondary	48	1.50(0.88,2.56)	1.23(0.68,2.26)	2.87(0.83,9.90)	1.51(0.72,3.16)	1.51(0.67,3.44)
Upper secondary and above	19	1.00	1.00	1.00	1.00	1.00
Other causes						
Less than primary	310	2.76(2.15,3.54)	2.59(1.90,3.54)	2.96(1.92,4.56)	2.69(1.87,3.87)	2.69(1.84,3.93)
Primary	184	1.78(1.38,2.30)	1.83(1.35,2.48)	1.76(1.11,2.81)	1.85(1.29,2.67)	1.70(1.15,2.50)
Lower secondary	227	1.48(1.17,1.88)	1.49(1.12,1.98)	1.48(0.95,2.32)	1.53(1.12,2.09)	1.41(0.96,2.07)
Upper secondary and above	100	1.00	1.00	1.00	1.00	1.00

Fig. 2: Hazard ratio (HRs) and 95% CI for the risk of all-cause and cause-specific premature mortality by educational levels across sex and urbanicity. HR was calculated by Cox models for all-cause specific mortality and by competing risk models for cause-specific. Models was adjusted by age, sex and urbanicity.

population sample curated from the databases enabled us to compute population attributable fraction to quantify the burden of premature deaths associated with low education.

Respiratory diseases emerged as the cause that exhibited the strongest association with education in China. The cause to which education has the strongest impact is closely tied to the epidemiological profile of a

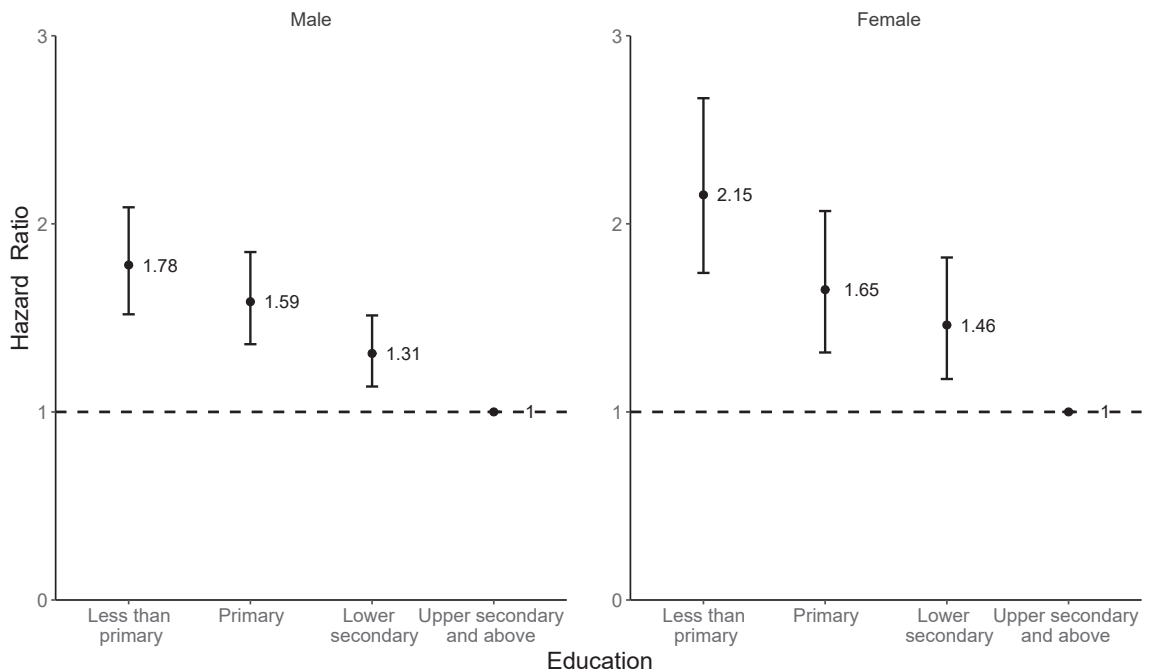


Fig. 3: Hazard ratio (HRs) and 95% CI for the risk of all-cause premature mortality by educational levels, stratified by sex. HR was adjusted by age and urban-rural and was displayed with plots, error bars and numbers.

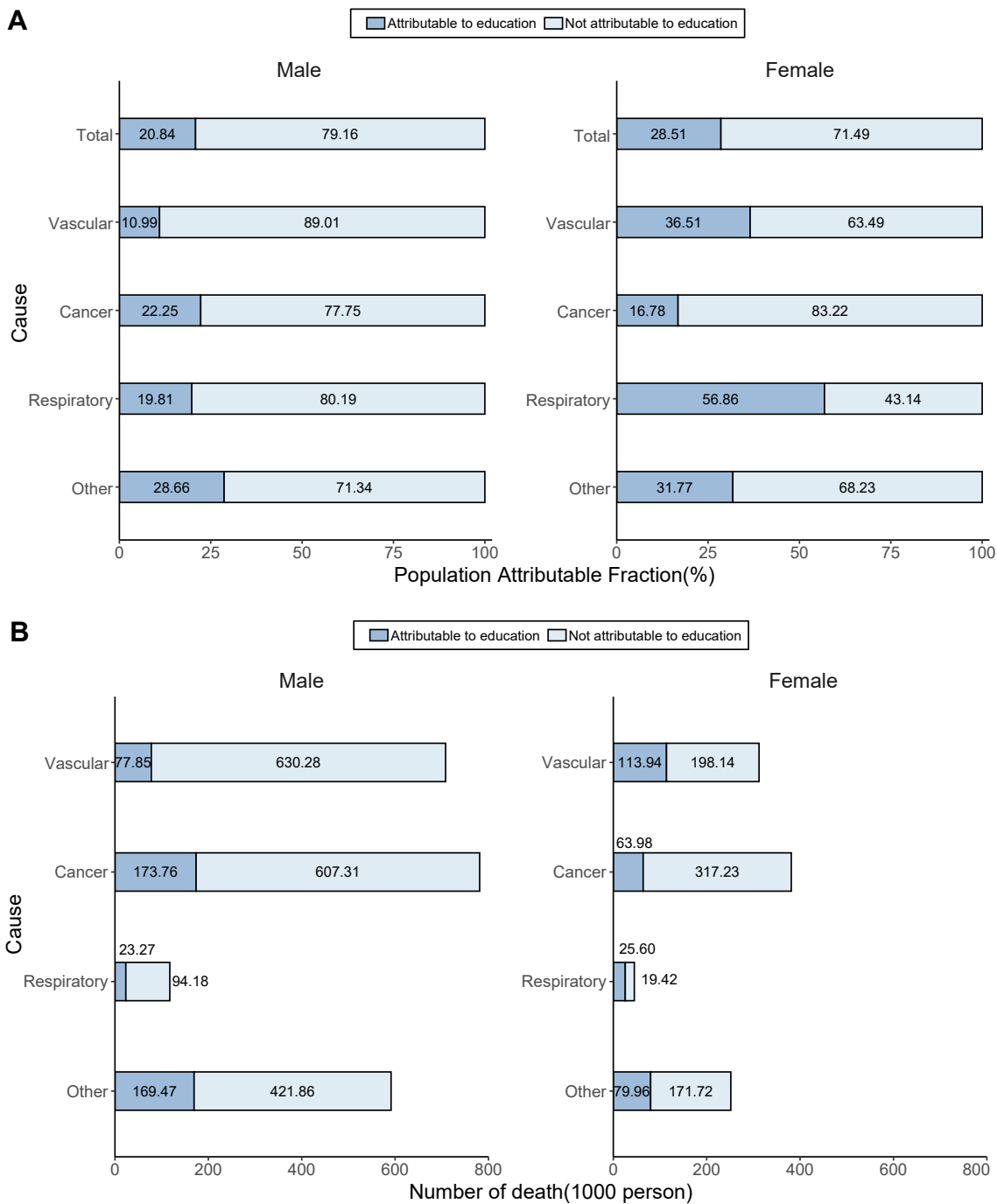


Fig. 4: The population fraction (A) and number in 2019 (B) of premature deaths attributable and not attributable to education, by sex and cause of death. The population fraction and number attributable to education represents the expect reduction in premature mortality if all people experienced the mortality of whom completed high school education.

country. In other countries,^{9–11} such as Cuba, education was found to exert a more substantial impact on vascular mortality in women and cancer mortality in men. In Mexico, education demonstrated a larger influence on

renal/acute diabetes mortality in both sexes. In England, COPD and liver disease were the top mortality causes which had stronger associations with education. The disease burden associated with respiratory conditions is

Education	Model1	HR(95%CI)	Model2	HR(95%CI)	Model3	HR(95%CI)
All causes						
Less than primary	■	1.70(1.49,1.94)	■	1.72(1.50,1.96)	■	1.71(1.49,1.95)
Primary	■	1.46(1.28,1.66)	■	1.48(1.30,1.69)	■	1.46(1.28,1.67)
Lower secondary	■	1.29(1.14,1.45)	■	1.30(1.15,1.47)	■	1.28(1.14,1.45)
Upper secondary and above	■	1.00	■	1.00	■	1.00
Vascular						
Less than primary	■	1.36(1.08,1.72)	■	1.38(1.09,1.76)	■	1.37(1.08,1.74)
Primary	■	1.32(1.05,1.66)	■	1.33(1.06,1.69)	■	1.30(1.03,1.65)
Lower secondary	■	1.17(0.94,1.45)	■	1.18(0.95,1.47)	■	1.13(0.91,1.41)
Upper secondary and above	■	1.00	■	1.00	■	1.00
Cancer						
Less than primary	■	1.45(1.16,1.82)	■	1.45(1.15,1.82)	■	1.47(1.16,1.85)
Primary	■	1.44(1.16,1.79)	■	1.44(1.16,1.79)	■	1.43(1.15,1.79)
Lower secondary	■	1.30(1.07,1.59)	■	1.31(1.07,1.60)	■	1.32(1.08,1.62)
Upper secondary and above	■	1.00	■	1.00	■	1.00
Respiratory						
Less than primary	■	2.28(1.33,3.90)	■	2.41(1.40,4.15)	■	2.19(1.25,3.86)
Primary	■	1.45(0.82,2.56)	■	1.51(0.86,2.67)	■	1.40(0.77,2.53)
Lower secondary	■	1.31(0.76,2.25)	■	1.32(0.77,2.27)	■	1.32(0.75,2.31)
Upper secondary and above	■	1.00	■	1.00	■	1.00
Other causes						
Less than primary	■	2.47(1.91,3.20)	■	2.43(1.87,3.17)	■	2.41(1.85,3.14)
Primary	■	1.65(1.27,2.14)	■	1.68(1.29,2.19)	■	1.67(1.28,2.19)
Lower secondary	■	1.42(1.11,1.80)	■	1.44(1.13,1.84)	■	1.41(1.10,1.81)
Upper secondary and above	■	1.00	■	1.00	■	1.00

Fig. 5: Hazard ratio (HRs) and 95% CI for the risk of all-cause and cause-specific premature mortality by educational levels. HR was calculated by Cox models for all-cause specific mortality and by competing risk models for cause-specific. Model 1 was adjusted by sex, age at baseline, urbanicity, marital status and income. Model 2 was adjusted by smoking status, alcohol intake, diet, physical activity based on model 1. Model 3 was adjusted by BMI, blood pressure, lipids and glucose based on model 2.

high in China. Upper and lower respiratory infections, for example, had resulted in 5 million DALYs in 2019.³⁰ Chronic respiratory diseases such as COPD was among the top three causes of death and resulted in 19.92 million DALYs in the same year.^{31,32} The strongest association with respiratory diseases might be explained by the exposure to tobacco and pollutants. A research found that people of low socioeconomic status had higher smoking rates, which was also found in our study, and were more likely to be exposed to occupational particulates and household and outdoor pollutants.³³

Although the mortality of men was higher than that of women, the excess risk of premature mortality associated with low education was higher among women than men, especially for respiratory-related mortality. The differences in sexes likely stem from varied exposures to risk factors. Despite higher education levels being associated with reduced risk behaviours,^{34,35} the overall prevalence of certain factors, especially smoking, remained high among men across all education levels.

The prevalence of obesity, hypertension, and diabetes increased with increased education in men but not in women. Additionally, the differences in knowledge, material conditions and other factors between men and women might also lead to this difference.^{36,37} For cause-specific mortality, there was a marked difference in the association between education and respiratory-related mortality between sexes. One hypothesis is that the women with lower education level are more likely to be limited to housework including cooking, and cooking with unclean energy source can lead to more risky respiratory outcomes. One study showed that lung cancer and COPD in women were associated with indoor biomass smoke.³⁸

Income, smoking, alcohol consumption, and intake of vegetables and fruits had different proportions of mediation between men and women. The difference in mediation of risk factors between men and women was mainly due to income, which produced a mediation effect of 23.0% in men and 11.1% in women. A previous study³⁹ found that the association between income and

mortality was stronger among men, and men were more vulnerable to income loss.

We found a stronger impact of education among urban population. One hypothesis to explain these urban-rural discrepancies is the differences in access to health services. Recent policies have substantially improved rural medical infrastructures.^{40–42} Such advancements might have equalized medical access across educational strata in rural settings. According to the health statistics yearbook, in 2019, the number of medical institutions in rural areas reached 795,534, while 212,045 in urban areas. However, 96.56% of rural medical institutions were primary medical institutions. Highly educated people in urban areas had better access to high-quality medical care. Another hypothesis is the geographical difference in the relationship between education and specific risk factors. For instance, while education appears to have minimal influence over smoking behaviours in rural areas, an inverse relationship between education and smoking was observed in urban areas.⁴³ Lastly, individuals with higher level of education tend to reside in urban areas. In our dataset, 70–80% of upper secondary school graduates resided in cities. Previous study has indicated that the concentration of highly educated individuals may foster a communal effect, creating a conducive environment which promotes awareness for better health.⁴⁴

Educational inequality accounted for about 20% of premature deaths of men and 30% of women, which referred to the proportion of deaths that could have been reduced if all people experienced the same mortality as those completing upper secondary education. The PAF of education in women was higher than that in men, especially for premature deaths caused by respiratory diseases. And the educational inequality accounted for 457,979 premature deaths in men and 282,276 in women, which indicated that reducing education inequality could bring considerable health benefits, both in men and women. Previous studies have shown that increasing the years of education and promoting educational equality in the population could decrease mortality. A Korean study⁴⁵ found that a consistent and sharp increase in educational attainment contributed to the decrease in PAFs of low education for mortality. And an empirical study⁴⁶ found that each additional year of education was observationally associated with a 14% lower mortality.

The negative association between education and premature mortality can be achieved in many ways. Firstly, education attainment is strongly related to socioeconomic factors such as career, economic condition, living environment, and others.⁴⁷ As our research showed, income contributes to the main mediation (23.0% in men and 11.1% in women). These socioeconomic factors can affect the access to public and medical services, exposure opportunities for risk factors and psychosocial indicators. Secondly, education can

influence behavioural factors. Highly educated individuals are more likely to adopt healthy lifestyles. In our results, smoking, alcohol consumption and intake of vegetables and fruits had significant mediating effects. However, there was a considerable proportion of mediation that could not be explained, indicating an effect of underlying factors. The heightened concern about one's health condition among highly educated people and the stress and feelings of worthlessness associated with low socioeconomic position¹¹ might be underlying mechanisms.

The Chinese government has been taking effort to improve the educational situation of population over the past few decades. However, due to regional and demographic differences in economic development, there is still a considerable inequality in education, which according to our results, caused an inequality in health. Therefore, we advocate for the following measures to reduce the mortality attributed to educational inequality. Firstly, policies should be continuously implemented to increase the number of years of education. This can include promoting the allocation of educational resources to underdeveloped areas, and improving the scale and quality of high school and above education. Secondly, more attention should be paid to the health problems of individuals with low education, particularly focusing on women and urban population. This can involve organizing health examinations and disease screenings, as well as increasing the proportion of medical insurance coverage. Thirdly, health education for individuals with low education should be strengthened to mitigate adverse behaviours, such as smoking, alcohol consumption and diets.

Our research had several limitations. Firstly, education was measured by self-reporting. The quality of education and actual learning were not captured. It has been documented that the quality of education varies across provinces and urban and rural settings.⁴⁸ The lack of robust capture of learning quality could lead to an underestimation of the actual effect of education. Secondly, the baseline characteristics of individuals were based on data from the 2010 CCDRFS. Certain risk factors might have changed. Thirdly, the death outcomes were obtained from NMSS. Because of underreporting, some deaths might not have been obtained, but almost all deaths could be linked. The lost follow up could potentially lead to an underestimation of the association, but in our results, a strong association between education and mortality was observed. Additionally, underreporting was primarily attributed to the quality of the local reporting system.⁴⁹ Therefore, the impact of personal characteristics on underreporting was limited, especially among people aged 30 to 70, and we had confirmed and implemented the deaths after linkage to NMSS. Hence, we could assume the missing was random across levels of education and the effects on results were limited. Fourthly, income was the sole

measure of individuals' socioeconomic status, and the income of original family might affect individuals' education. However, given that the main purpose of this study was not to assess the relative impact of mediating factors on the education-mortality relationship, income adequately served as a proxy of individuals' socioeconomic well-being. Moreover, since most individuals aged 30 to 70 had already left their original families, their current income better reflected the material conditions they had gained through education. Finally, our study period was limited to 10 years. Although there were 705,175 person-years, the total number of premature deaths recorded was 3082. The sample size for certain conditions, such as respiratory diseases was relatively low, which yielded relatively large confidence intervals reflecting higher uncertainty in those results.

In conclusion, we found increased premature mortality risk associated with low education, especially in women, and a considerable number of deaths attributed to educational inequality. As China continues to expand and enhance education equity and quality, efforts are necessary to strengthen public health interventions to improve education of the population, avoid adverse health outcomes among those with low education attainment and mitigate the negative impacts of risk factors such as smoking, alcohol consumption and diets. Similarity, the differences observed in urban and rural settings revealed the existence of a socioeconomic divide, which modifies the impact of education on mortality between the populations. Further studies are needed to examine the various hypotheses that might explain the underlying differences.

Contributors

JW was the first author and analysed the data and drafted the manuscript. MZ and MN designed this work and revised the manuscript and contributed equally to the correspondence work. ZZ contributed to data collection and study design. JY contributed to the data interpretation. JW, ZZ and MZ have accessed and verified the data, and MZ were responsible for the decision to submit the manuscript. All authors contributed to the development of the manuscript and approved the final draft. The corresponding authors attest that all listed authors meet authorship criteria and that no others meeting the criteria have been omitted.

Data sharing statement

The study data that underlie the results of this article will be available for investigators after approval by the Chinese Center for Disease Control and Prevention (Beijing, China). Please email the corresponding author for more information.

Declaration of interests

We declare no competing interests.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at <https://doi.org/10.1016/j.lanwpc.2024.101085>.

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