

Cause-contributions to Subnational Life Expectancy Sex Gaps:

Evidence by education levels from Australia

Authors:

Wen Su*

School of Demography, College of Arts and Social Sciences, Australian National University

Jennifer Welsh

National Centre for Epidemiology and Population Health, ANU College of Health and
Medicine, Australian National University

Rosemary J. Korda

National Centre for Epidemiology and Population Health, ANU College of Health and
Medicine, Australian National University

Vladimir Canudas-Romo

School of Demography, College of Arts and Social Sciences, Australian National University

*Corresponding Author: Wen Su (wen.su@anu.edu.au)

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Abstract

Life expectancy sex gap is a vital indicator comparing mortality difference between females and males, calculated as the difference between respective life expectancies. Among high-income countries, life expectancy has been narrowing before COVID-19 pandemic. While an abundance of studies focuses on national-level comparisons, few examine the life expectancy sex gap at the subnational level across different subpopulations. Subpopulations vary in their mortality and health profiles due to underlying structural factors related to socioeconomic status. Insufficient evidence exists to determine whether socioeconomic inequality extends to life expectancy sex gaps among subpopulations stratified by education level. Using whole-of-population linkage data from Australia during the period 2016-2019, we quantify the life expectancy sex gap by education level. We also disaggregate the life expectancy sex gap into contributions from various causes of death to gauge the influencing factors. We highlight a gradient in the life expectancy sex gap, with the university-educated population experiencing a smaller gap than their peers with lower education. We observed a large difference in contributions towards life expectancy sex gaps from external causes across education levels. Meanwhile, a pronounced portion of the difference is attributable to a combination of cardiovascular diseases, cancers, and respiratory diseases, particularly among individuals aged 60 to 85. We also note a persistent disadvantages for females at ages 25-60 from breast and gynaecologic cancers.

Introduction

Since 2006, female life expectancy has consistently exceeded male life expectancy worldwide (1). The life expectancy sex gap quantifies the magnitude and direction of the difference between female and male life expectancy, calculated as the difference between the two. In the past decades, the life expectancy sex gap in most high-income countries has begun to narrow. However, during the COVID-19 pandemic, the life expectancy sex gap briefly widened in most high-income countries (2). Understanding the female advantage in the life expectancy sex gap offers insights for pension planning and helps clarify biological differences between males and females.

Several studies have attempted to attribute the female advantage in the life expectancy sex gap to various influencing factors. An abundance of studies focuses on comparisons at the national level (3-5). Some studies have used subpopulations with specific characteristics to simulate comparisons in a natural experimental setting (6-10). However, few studies focus on the subnational level by examining the life expectancy sex gap across different subpopulations (11, 12). Subpopulations differ in their mortality and health profiles due to various underlying structural factors related to socioeconomic status. Male and female subpopulations stratified by socioeconomic status exhibit noticeable inequality, with those in better socioeconomic conditions having higher life expectancy. Meanwhile, insufficient evidence exists to determine whether this inequality by socioeconomic status extends to life expectancy sex gaps among subpopulations stratified by socioeconomic status.

This study quantifies the life expectancy sex gap by education level as a dimension of socioeconomic status. Additionally, we disaggregate the life expectancy sex gaps for different education levels into contributions from various causes of death.

Methods

Data

We conducted secondary data analysis using whole-of-population linkage data from Australia as part of the Person Level Integrated Data Asset (hereafter PLIDA) compiled by the Australian Bureau of Statistics (13). The data preparation data preparation process followed the methods of Welsh, Bishop (14) and Korda, Biddle (15). The PLIDA project links the Census 2016 information on education to the Death Register throughout the period 2016-2019 for the individual who is present within the Census 2016. Our study population included residents who were 25 or at least aged 25 during the period 2016-2019. Linkage was performed using a de-identified personal-level key, incorporating individual data from Medicare Enrolments, Social Security information, and Personal Income Tax records (15). Death Register data captured nearly all individuals who died during the study period, while the 2016 Census covered 94.8% of Australia's usual residents as of August 9, 2016 (16). For our study, educational attainment was derived from several questions in the 2016 Census, available for 85% of respondents. Missing data on individual education levels was imputed using a single imputation based on other covariates available in the 2016 Census.

Analysis

Following the procedures outlined above, we obtained mid-period population counts by education level for 2016-2019, as well as the estimated average number of deaths by group of causes for the same period, both stratified by 5-year age interval. We subsequently smoothed the population and cause of death data into 1-year age intervals using the Penalized-Composite Link Model (PCLM) developed by Rizzi et al. (17, 18). We constructed life tables by sex and education level using the standard life table method, with age-specific death rates calculated by dividing all-cause death counts by population counts at each age (19). We generated 95%

confidence intervals for female and male life expectancy, as well as for the life expectancy sex gap, using the life table bootstrap method (20, 21). The further details on the data treatment can be found in **Supplementary Material**.

This study employed demographic decomposition analysis to disaggregate age- and cause-specific contributions to the life expectancy sex gap for the Australian national population and across different education levels within Australia. Demographic decomposition analysis quantifies the components contributing to differences in the outcome indicator (e.g., life expectancy) in terms of the indicator's unit of measurement (years). We engaged the method developed by Vaupel and Canudas-Romo (22), which is a continuous-time equivalent to the discrete-time method developed by Arriaga. The methods separate contributions in terms of years of life attributable to different groups of causes of deaths across every age, with their sum equalling the life expectancy sex gap. The Vaupel & Canudas-Romo method and Arriaga methods only differ in the assumptions made about changes in the force of mortality. Therefore, the two methods will likely result in almost identical results. The Vaupel & Canudas-Romo method assumes a constant rate of change in the force of mortality, contrary to the Arriaga method, which assumes a linear change. As a result, the Vaupel & Canudas-Romo method produces smoother age-pattern results, especially when analysing single-year age intervals. A detailed description of the decomposition method could be found in **Supplementary Material**.

Results

Figure 1 presents the outlook of life expectancy sex gap across different education levels, as well as the life expectancy sex gap for the Australian national population. For university educated population, we are seeing a much smaller life expectancy sex gap (2.28 [1.99, 2.56] years), compared to those of secondary & equivalent and lower than secondary educated population. In the meantime, the university educated population is also enjoying a smaller life expectancy sex gap compared to the national average (3.79, [3.69, 3.88] years). Secondary & equivalent educated population (4.42 [4.26, 4.56] years) and lower than secondary educated population (4.67 [4.46, 4.89] years) have similar level of life expectancy sex gap, both lower than that of the Australian national level. The lower than secondary educated population has the highest life expectancy sex gap among all the education levels.

We subsequently looked at the cause-contributions across all ages to these life expectancy sex gaps we observed at the national level and across different education levels within the nation. These results are shown in Table 1. For cardiovascular diseases, contribution to life expectancy sex gap (hereafter simply contribution) for population with university education is lower than that of population with secondary & equivalent and lower than secondary education (0.46 [0.36, 0.58] years of mean absolute difference). The mean absolute difference in contributions for external causes between either lower than secondary education and university educated population is around 0.84 [0.81, 0.86] years, with a mean relative difference (ratio of mean contributions) being 3.4. Similar comparison can also be found in contributions from other cancers, only the largest mean difference is observed for 0.58 [0.38, 0.78] years (ratio of 1.98) between Secondary & equivalent and university educated population. These three groups of causes also accounted for around 70% of contributions towards each of the education-specific life expectancy sex gap (university 77.1%, secondary & equivalent 74.4%, lower than secondary 73.7%). Meanwhile, although having a moderate scale in contribution towards life

expectancy sex gap, lung cancer and respiratory diseases also show a gradient across different education levels, with university educated population having a much smaller contribution to their respective life expectancy sex gap compared to that of population with secondary & equivalent education (0.24 [0.16, 0.31] years) and population with lower than secondary education (0.32 [0.31, 0.33] years). For death from mental disorders, the population with university education shows an advantage towards males with negative values, while other two education levels show a female advantage like other groups of causes observed. For other causes, all education levels show similar contributions towards their respective life expectancy sex gap.

A breakdown of the age pattern of the contributions by causes for each education level, and in comparison, to the national population can be found in Figure 2. On top of this, we have also presented in Figure 3 a comparison of different cause contributions towards life expectancy sex gap across populations with different education levels. At each age, the contributions from various causes across different population with different education levels are compared against each other in Figure 3. For all-cause contributions, population with university education shows a more compacted age pattern compared to populations with other education levels (see Figure A2 in **Supplementary Materials**). For population with lower than secondary education, a bimodal distribution of the all-cause contribution is observed.

At younger to working ages, cardiovascular diseases, and more importantly external causes, dominate the contributions towards life expectancy sex gap for all education levels. Although for the population with the highest education, the contributions from these two causes towards life expectancy sex gap are smaller than their peer populations with lower education level (university 0.37 [0.16, 0.58] years, secondary & equivalent 1.01 [0.80, 1.22] years, lower than secondary 1.45 [0.97, 1.94] years)). Meanwhile, females have a disadvantage when looking at contributions from other cancers alone at ages 25-60 for all population separated by education

level. This phenomenon is especially visible across a long span of ages for university educated population.

At same time, contributions from the age group 60-85 account for a large proportion of the life expectancy sex gap across different population by education level (university 60.6%, secondary & equivalent 57.0%, lower than secondary 51.5%). For population with university education, contributions from different causes are smaller than those for populations with lower education (with 1.36 [0.23, 2.49] years). This is due to not only more concentrated contributions from different causes at much older ages for population with university education, but also similar or smaller contributions at every age from different causes, in comparison to other population with lower education (see Figure 3). An earlier onset of large contributions from cardiovascular diseases (also other cancers) within this age group for population with lower than secondary education shapes the bimodal all-cause age pattern we observed earlier.

Beyond age 85, contributions are similar across different education level. The negative contributions from mental conditions that serves male advantage, mentioned in the previous paragraph are mostly concentrated within this age group.

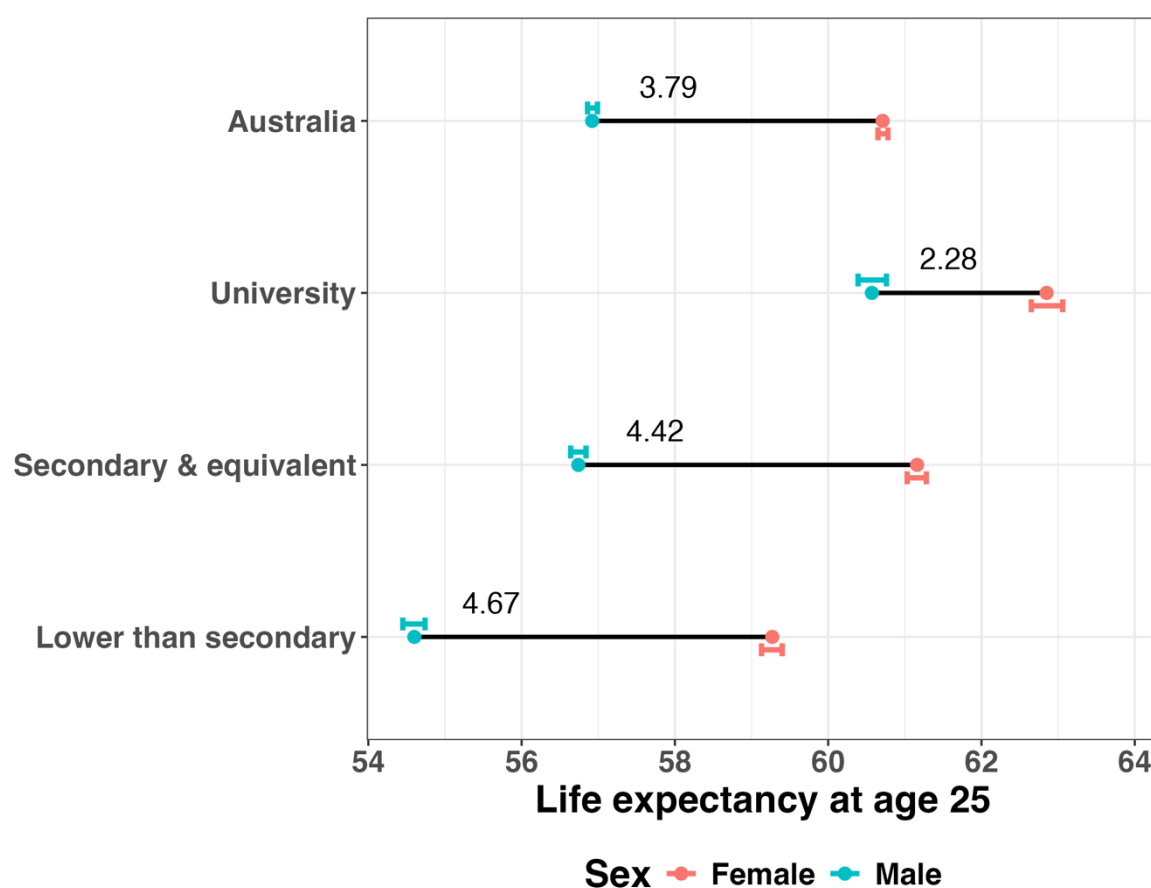
Highlight of Results

1. There is a gradient in the life expectancy sex gap across education levels. The population with a university education has a smaller life expectancy sex gap compared to their peers with lower education levels. In the population with a university education, the age pattern of all-cause contributions to the life expectancy sex gap is largely concentrated in older age groups. For populations with secondary or equivalent, as well as lower than secondary education, the age patterns are more spread out, with higher contributions from younger age groups.
2. There are clear differences in contributions from CVD and lung related diseases across different population subgroups by education levels. This might be linked with behaviour-related causes, mostly due to the socioeconomic determinants (influence of smoking; environmental factors, stress, occupational hazards) associated with different education levels. Similar outlook can be found with a large difference in external mortality across different education levels. Effective public health campaign reducing the risk of dying from lung cancer and respiratory diseases, which were known for having a direct link to smoking, as well as from external causes could further narrow the life expectancy sex gap in the future across different education levels and at the national level.
3. We observed negative contributions from other cancers serve towards female disadvantage around age 25-60. This female disadvantages from other cancers (mostly breast and gynaecologic cancers) are most visible within the population with university education. For populations with other education levels, we are seeing a less visible negative contribution. This is likely a result of counteraction to female disadvantages from males within these populations having similar or higher mortality from other cancers compared to females.
4. After accounting for major causes (e.g. cardiovascular diseases, lung cancer, external causes, etc.) that contributed to the life expectancy sex gap, we are seeing similar scale of

contributions from other causes across different education levels. This phenomenon might be attributed to males and females share similar risks of death among these underlying causes.

Figures

Figure 1. Life expectancy sex gap for the Australian national population and for each education level within Australia during the period 2016–2019.



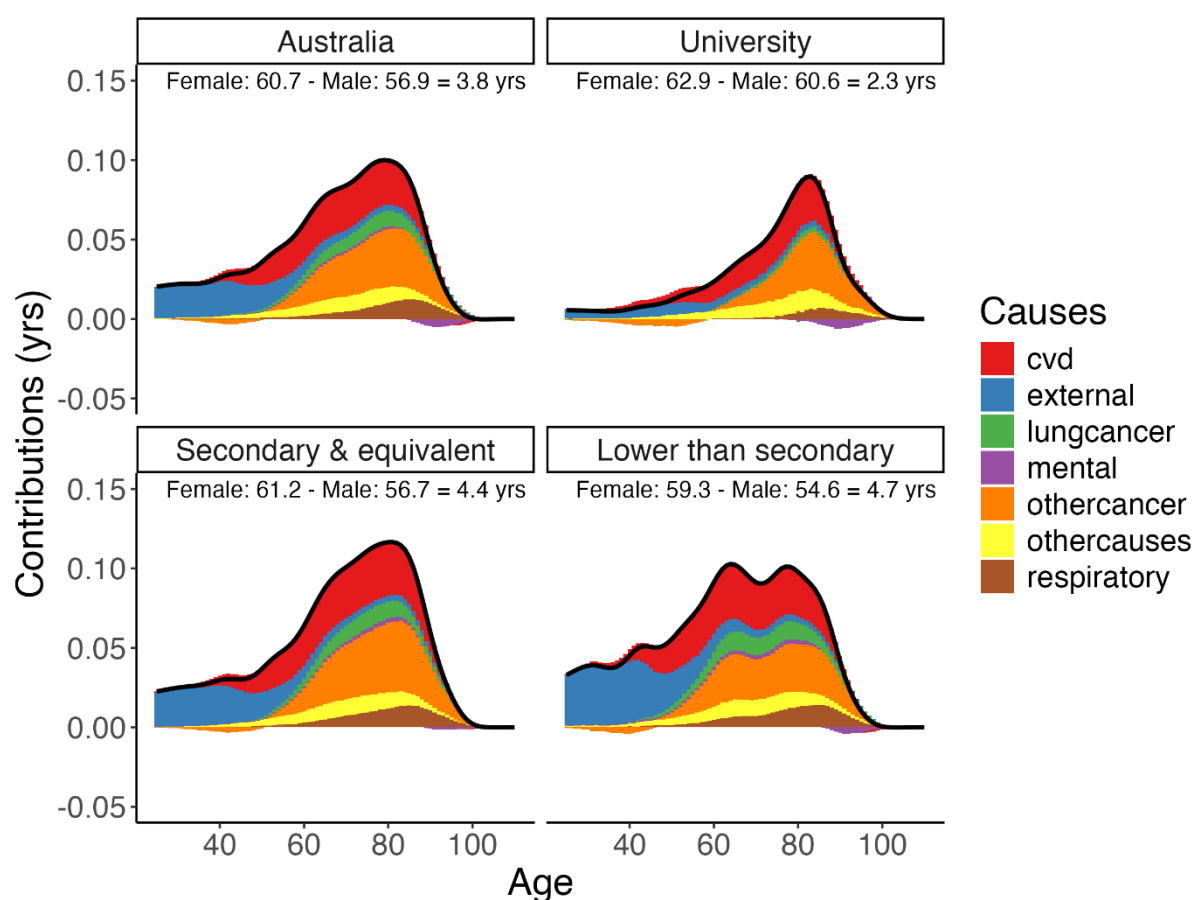
Notes: The coloured dot represents life expectancy at age 25 for either females (red) or males (green). The coloured bar next to the dots represents the 95% confidence interval for the life expectancy. The text represents the life expectancy sex gap calculated based on the mean value of the female and male life expectancy.

Table 1. Cause-specific contributions across all ages to the life expectancy sex gap for the Australian national population and for each education levels within Australia during the period 2016–2019.

<i>Causes</i>	<i>Australia</i>	<i>University</i>	<i>Secondary & Equivalent</i>	<i>Lower than Secondary</i>
<i>Total</i>	3.79 [3.69, 3.88]	2.28 [1.99, 2.56]	4.42 [4.26, 4.56]	4.67 [4.46, 4.89]
<i>Cardiovascular diseases</i>	1.11 [0.92, 1.31]	0.82 [0.29, 1.34]	1.28 [0.97, 1.59]	1.28 [0.87, 1.7]
<i>Other cancers</i>	0.98 [0.77, 1.19]	0.59 [0.06, 1.13]	1.17 [0.84, 1.51]	0.97 [0.56, 1.39]
<i>External causes</i>	0.77 [0.63, 0.91]	0.35 [0.04, 0.67]	0.84 [0.62, 1.06]	1.19 [0.85, 1.53]
<i>Lung cancer</i>	0.29 [0.18, 0.4]	0.07 [-0.17, 0.32]	0.31 [0.14, 0.48]	0.39 [0.16, 0.63]
<i>Respiratory diseases</i>	0.29 [0.17, 0.41]	0.12 [-0.18, 0.41]	0.35 [0.16, 0.54]	0.38 [0.12, 0.64]
<i>Mental disorders</i>	-0.01 [-0.09, 0.07]	-0.07 [-0.31, 0.18]	0.05 [-0.08, 0.18]	0.05 [-0.1, 0.21]
<i>Other causes</i>	0.35 [0.21, 0.49]	0.37 [-0.01, 0.73]	0.38 [0.16, 0.59]	0.39 [0.1, 0.67]

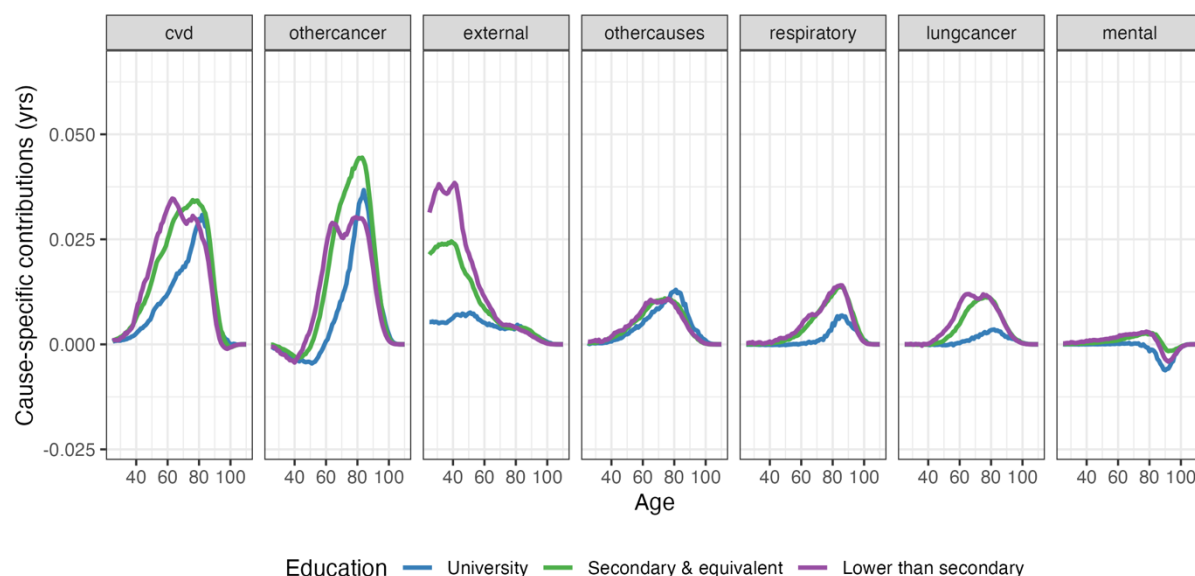
Notes: Cause groups within the table are arranged by the mean size of the cause-contributions for the Australian National level from the highest to the lowest. Values in brackets indicates the 95% confidence interval.

Figure 2. Age- and cause-specific contributions to the life expectancy sex gap for the Australian national population and for each education levels within Australia during the period 2016–2019.



Notes: The dark line represents the mean age-contributions towards life expectancy sex gap for each education levels and Australian national population. Different colours represent different cause categories. The cause categories are **cvd**: cardiovascular diseases, **external**: external causes, **lungcancer**: lung cancer, **mental**: mental disorders, **othercancer**: other cancers, **othercauses**: other causes, **respiratory**: respiratory diseases. A figure in age groups that contains the confidence intervals can be found in **Supplementary Materials**.

Figure 3. Comparison of the age- and cause-specific contributions to the life expectancy sex gap by education levels for the Australian national population and for each education levels within Australia during the period 2016–2019.



*Notes: Each panel presents the comparisons of the mean age- & cause- specific contributions towards life expectancy sex gap across different education levels and Australian national population. Different panels represent different cause categories. The panels are arranged by the scale of average contributions from highest (left) to lowest (right). The cause categories are **cvd**: cardiovascular diseases, **external**: external causes, **lungcancer**: lung cancer, **mental**: mental disorders, **othercancer**: other cancers, **othercauses**: other causes, **respiratory**: respiratory diseases. Figure that contains the confidence intervals can be found in **Supplementary Materials**.*

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Supplementary Material

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Supplementary Information on Data

Data Preparation

We fit Penalized Composite Link Model (PCLM) (see 1) to smooth the data into the shortest age interval possible (1 year) to suit our continuous-time decomposition. The PCLM smoothing assumes the data observed in the coarse age group (in our case the 5-year age group) is an indirect estimate of a latent and finer distribution of data. The PCLM makes modest assumptions of the underlying data and performs well compared to other smoothing techniques proposed in the past (2).

Our estimates resemble closely of those produced by Human Mortality Database (or HMD 3) at the national level. The difference between our estimates and that from HMD in terms of life expectancy at age 25 for females and males is around 0.2 years for the Australian national population.

The variable of sex in this study refers to the categorization of population traits in the 2016 Census. In the data linked by PLIDA projects, there were 1260 people out of the census Australian population who provided valid responses to sex/gender questions of “Other” aside from “Male” or “Female” (4). We acknowledge that differences in mortality can arise from cultural and behavioural traits, apart from the biological traits identified in the 2016 Census (5). Our data is therefore limited in this sense.

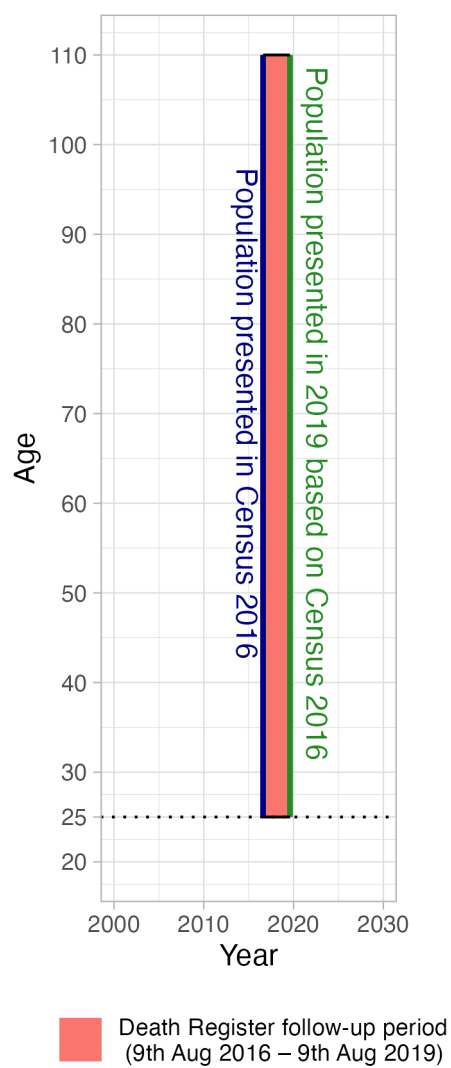
We used a bootstrap method to calculate the 95% confidence interval for the values of life expectancy for either males or females. We resample the all-cause number of deaths for 1000 times at each age and for each sex, resulting in 1000 life tables. We then based our calculations of the mean value of the life expectancy and the 95% confidence intervals for life expectancy, as well as those values for life expectancy sex gap on these resampled data. Confidence intervals constructed for cause-specific results are mentioned in the following section.

Education levels

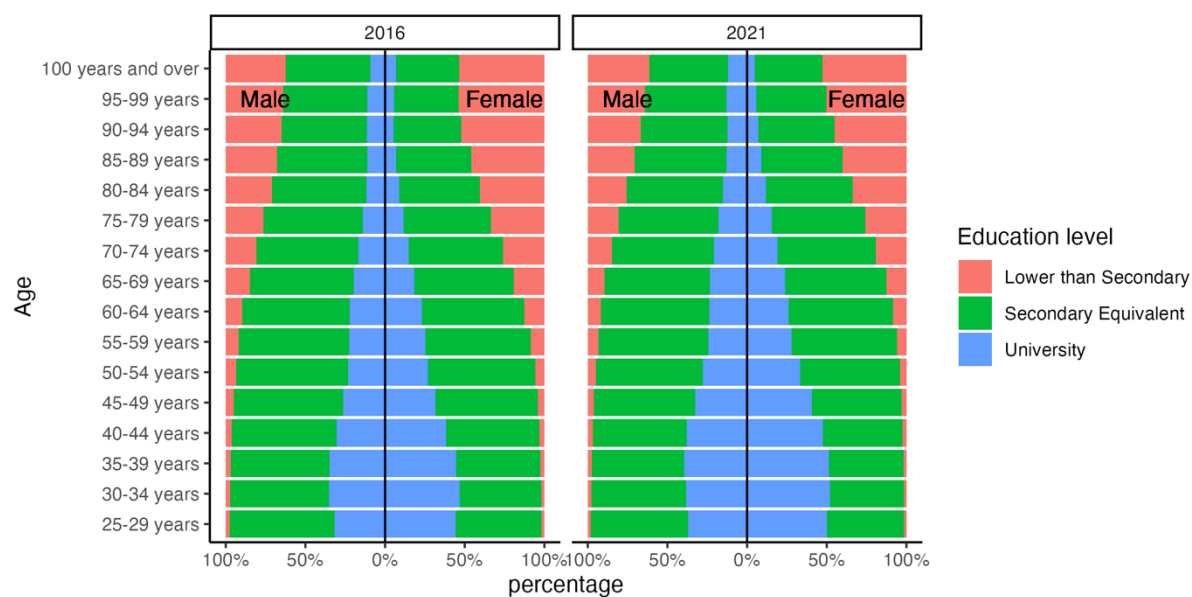
We derived the education levels in this study from the information in the Australian Census 2016 (6, 7). We matched the International Standard Classification of Education (8) to each level based on the definition and education qualification attained by individuals (Secondary, Post-Secondary, and Tertiary). Shorter labels were used as described in the right-last column.

Australian Census 2016 categorizations	International Standard Classification of Education (ISCED)	Categorization in this study
Bachelor's degree and up (Highest)	ISCED 5-8	University education
Other post-secondary education & finished year 12; Other post-secondary education & didn't finish year 12; No post-secondary education & finished year 12	ISCED 3-4	Secondary & equivalent
No post-secondary education & didn't finish year 12 (Lowest)	ISCED 0-2	Lower than secondary

Lexis surface for data coverage.



Comparison of education distribution between 2016 and 2021 for Australian National Population.



Notes: Each age group sums up to 100%. Education composition for males is on the left-hand side of the panel while composition for females is on the right-hand side.

Decomposition Methods Explained

We denote life expectancy at age 25 for a specific population during a given period as $e_{25}^i(s)$, with the notation s within the parenthesis representing either the male or female population. The notation i represents the subpopulation by education level we are examining in this study, being the population categorised by education levels within Australia, as well as the Australian national population. $e_{25}^i(s)$ is calculated as:

$$e_{25}^i(s) = \int_{25}^{\omega} \ell(x, s, i) dx. \quad (A1)$$

The life expectancy at age 25 for either females or males are calculated as taking the integral of (or sum across) the survival function $\ell(x, s, i)$ of a given education level i at each age x from age 25 to the highest age possible (denoted by ω). The survival function can also be related to the force of mortality μ from different causes, calculated as $\ell(x, s, i) = e^{-\int_{25}^x \sum_j \mu(a, s, i, j) da}$.

The decomposition method follows Vaupel and Canudas-Romo (9) which assumes a constant change of the force of mortality within an age interval between two periods. Let a dot on top of a variable (Newton's derivative notation (10)) denote change in a measure with respect to that variable. Here we look at $\dot{e}_{25}^i(s)$ or the change in life expectancy from the values of s observed for females to males for an education level i (similar assumptions of changes between populations can be seen in Su and Canudas-Romo (11), Canudas-Romo, Adair (12), Canudas-Romo and Guillot (13)). We will omit the notation s hereafter for simplicity. Assuming all functions used to calculate life expectancy change continuously as the values of s from females to males, the life expectancy sex gap (female-male) for a given education level i is given by:

$$\dot{e}_{25}^i = - \int_{25}^{\omega} \ell(x, i) e(x, i) \sum_j \dot{\mu}(x, i, j) dx.$$

(A2)

The notation $\ell(x, i)$ and $e(x, i)$ denotes survival function and remaining life expectancy at age x , respectively. The notation $\mu(x, i, j)$ represents the cause-specific force of mortality for age x , the education level i , and the cause j .

To simply explain equation A2, the life expectancy sex gap for an education level we observed for a given period is the sum of all differences from various cause-groups j , multiplied by a demographic weight reflecting the sensitivity of life expectancy changes to changes in force of mortality μ . The demographic weight $\ell(x, i)e(x, i)$ closely resembles the age-pattern of all-cause death distribution of the population in question, as shown in Aburto, Villavicencio (14).

We reply on the discrete approximation of the functions used in the decomposition when dealing with the continuous-time decomposition method. We assume the force of mortality is approximated by: $\mu(x, i) = 1 - e^{-m(x, i)}$. The notation $m(x, i)$ represents age-specific death rate from cause i within age x for female or male population. Let the notation v denote our interest variable (e.g. $e(x, i)$, $\mu(x, i, j)$). For approximating the derivative with respect to sex for a specific cause i under the continuous change assumption, we again use the equations developed in Vaupel and Canudas-Romo (9):

$$\dot{v}(x, i, j) = \log \left[\frac{v(x, female, i, j)}{v(x, male, i, j)} \right] \left(v(x, female, i, j) v(x, male, i, j) \right)^{\frac{1}{2}}.$$

(A3)

The confidence intervals for the cause-specific results from the decomposition method are calculated using the life table bootstrap method mentioned in the main text (12). For each iteration, we sample the number of deaths from different causes under a multinomial distribution for each age and sex. We then proceed to calculate the corresponding life table and

decomposition based on the resampled deaths counts. We repeat this process 1000 times. In this way, for every iteration we have a complete set of cause-specific decompositions towards life expectancy sex gap, preserving the constraints of adding to the total difference.

ICD-10 Causes Group

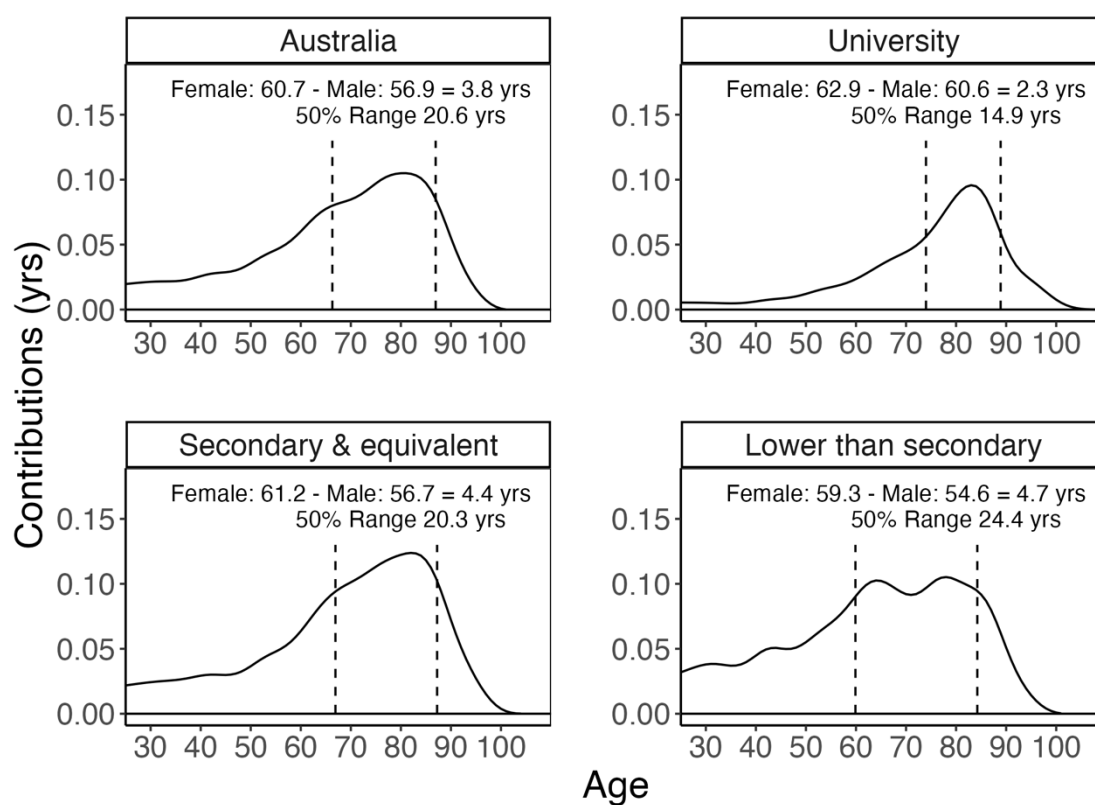
Table A1. Causes of death used and their respective International Classification of Diseases (ICD) codes from ICD 10.

Cause Groups	Short Name	ICD-10 Codes
Lung (and trachea, bronchus) cancers	lungcancer	C33, C34
Other cancers	othercancer	C00-25; C43-C97
Psychiatric conditions	mental	F01-F99
Cardiovascular Diseases	cvd	I00-I99
Respiratory Diseases	respiratory	J30-J98
All External Causes	external	V01-Y89
Other Causes	othercauses	Communicable, maternal, perinatal and nutritional conditions (A00-B99, D50-D53, D64.9, E00-E02, E40-E46, E50-E64, G00-G04, G14, H65-H66, J00-J22, N70-N73, O00-O99, P00-P96, U04, U07.1, U07.2, U09.9, U10.9); Other neoplasms (D00-D48); Diabetes mellitus and endocrine disorders (E10-E14, D55-D64 (minus D64.9), D65-D89, E03-E07, E15-E16, E20-E34, E65-E88); Neuro conditions (G06-G98 (minus G14), U07.0, X41, X42, X44, X45); Sense organ diseases (H00-H61, H68-H93); Digestive diseases (K20-K92); Genitourinary diseases (N00-N64, N75-N98). Skin diseases (L00-L98). Musculoskeletal diseases (M00-M99). Congenital anomalies (Q00-Q99). Oral conditions (K00-K14). Sudden infant death syndrome (R95). Ill-defined diseases (R00-R94, R96-R99)

Source: <https://platform.who.int/mortality>

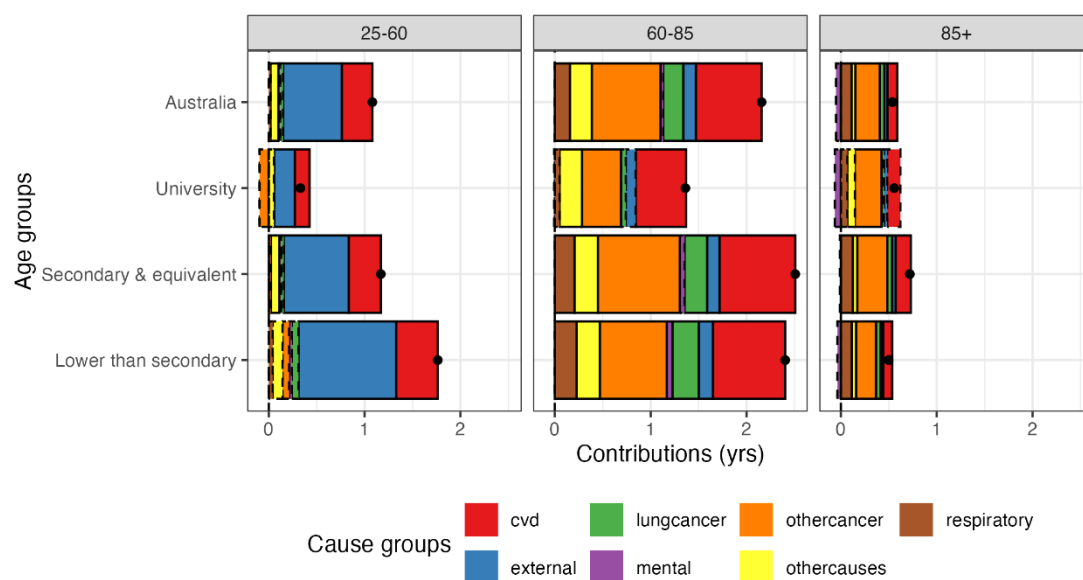
Supplementary Figures

Figure A1. Comparison of age-contributions to life expectancy sex gap for the Australian national population, as well as for each education levels within Australia during the period 2016–2019.



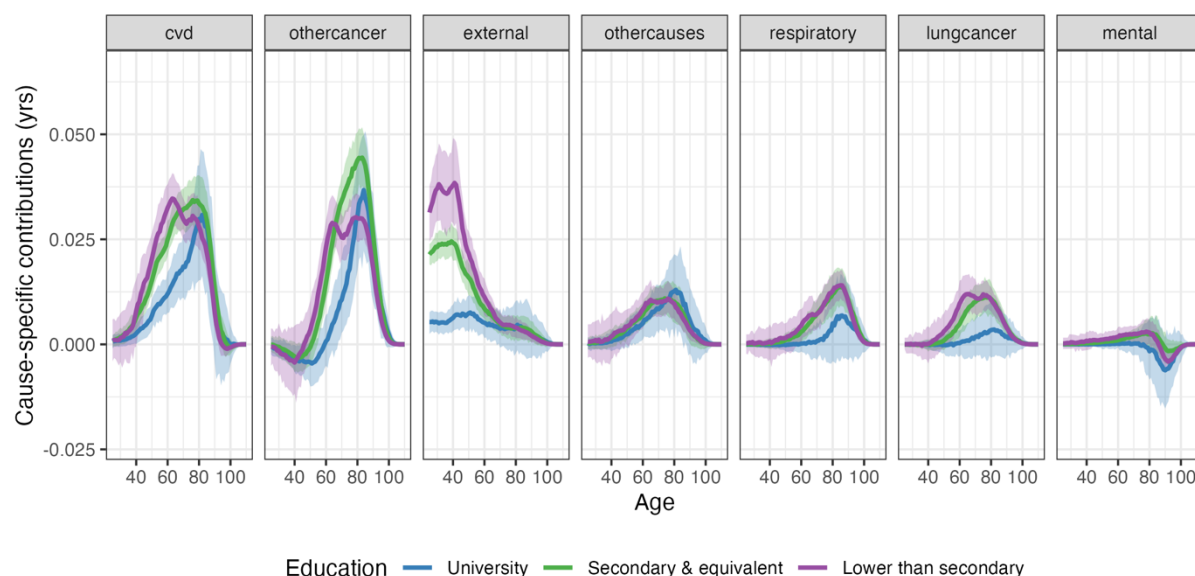
Notes: The first line of the caption represents the mean value of life expectancy sex gap. The second line represents the shortest age-interval of 50% of the contribution to life expectancy sex gap lies. The dot lines marked the lower (left) and upper (right) bounds of the shortest age-interval.

Figure A2. Age group- and cause-specific contributions to the life expectancy sex gap for the Australian national population and for each education levels within Australia during the period 2016–2019.



Notes: The component outlined by solid dark lines indicates that the lower bound of confidence interval for the age group- & cause-specific contributions to the life expectancy sex gap did not cross zero (statistically not significant), while the component being outlined by the dotted lines indicates that the lower bound of the confidence interval for the age group- & cause-specific contributions did cross zero (statistically significant).

Figure A3. Comparison of the age- and cause-specific contributions to the life expectancy sex gap by education levels for the Australian national population and for each education levels within Australia during the period 2016–2019.



Notes: Each panel presents the comparisons of the mean age- & cause- specific contributions (coloured lines), as well as the 95% confidence intervals of those contributions (coloured bands) towards life expectancy sex gap across different education levels and Australian national population. Different panels represent different cause categories. The cause categories are **cvd**: cardiovascular diseases, **external**: external causes, **lungcancer**: lung cancer, **mental**: mental disorders, **othercancer**: other cancers, **othercauses**: other causes, **respiratory**: respiratory diseases.

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