Decomposition Techniques for Social Epidemiology: Why and How

Danish Epidemiological Society Conference

Sam Harper

2026-09-22

McGill | Department of Epidemiology, Biostatistics and Occupational Health

Social Epidemiologic Aims

Descriptive

Existence of social differences in health

Etiologic

Causes of social differences in health

Interventions

Policies and programs to address causes

Enough already?

> Nevertheless, health inequalities are socially produced, and therefore, also potentially avoidable. However, effective political interventions require a scientific understanding of the causal mechanisms generating the strong and persistent correlations between social conditions and health outcomes.

Scandinavian Journal of Public Health
Volume 47, Issue 6, August 2019, Pages 593-597
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https://doi.org/10.1177/1403494819866416

Editorial

Social Inequalities in health: Challenges, knowledge gaps, key debates and the need for new data

Terje A. Eikemo and Emil Øversveen

How can we find out?

Decomposition: from description to explanation

What changed?

Explaining growing or shrinking gaps

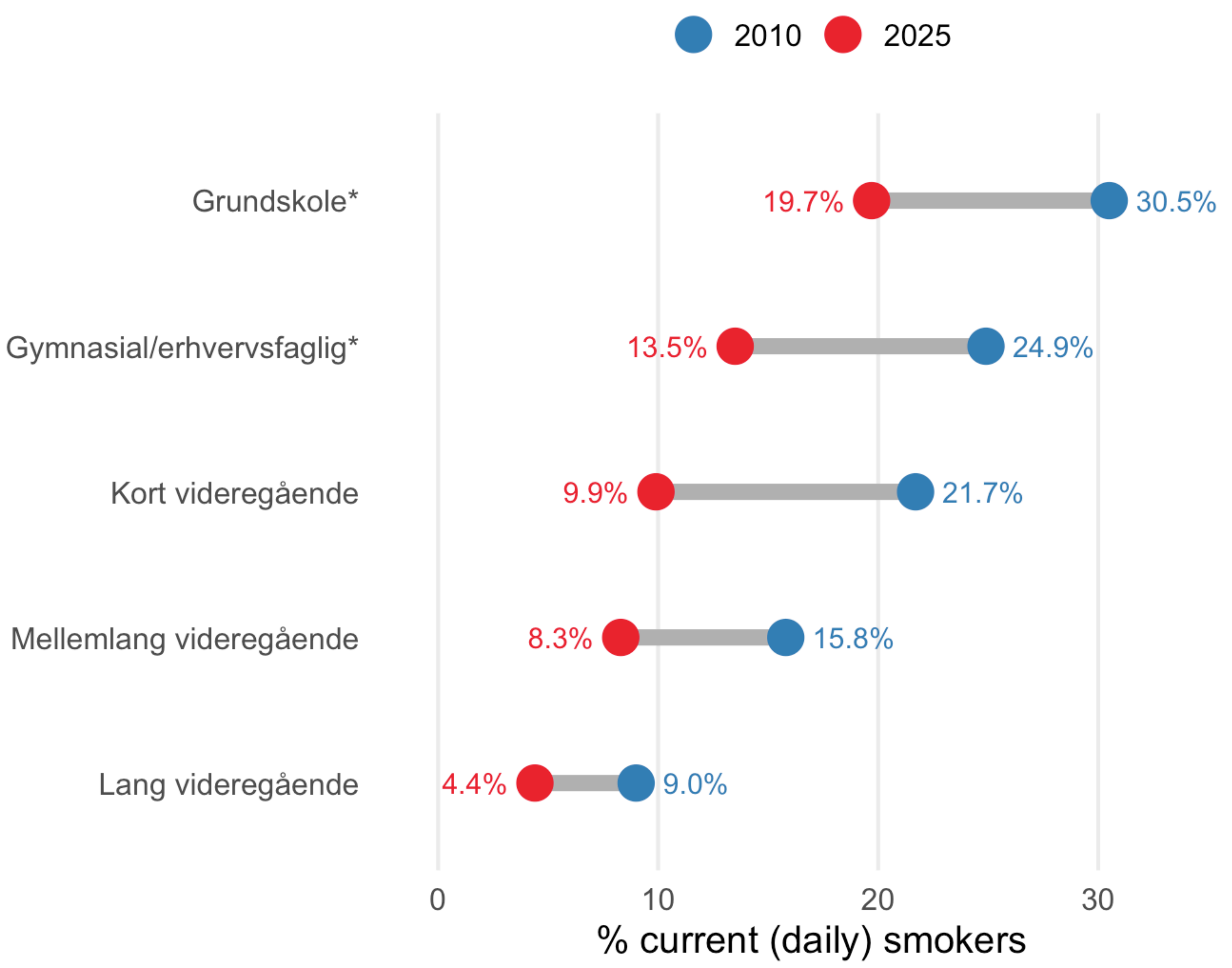
Which 'components'?

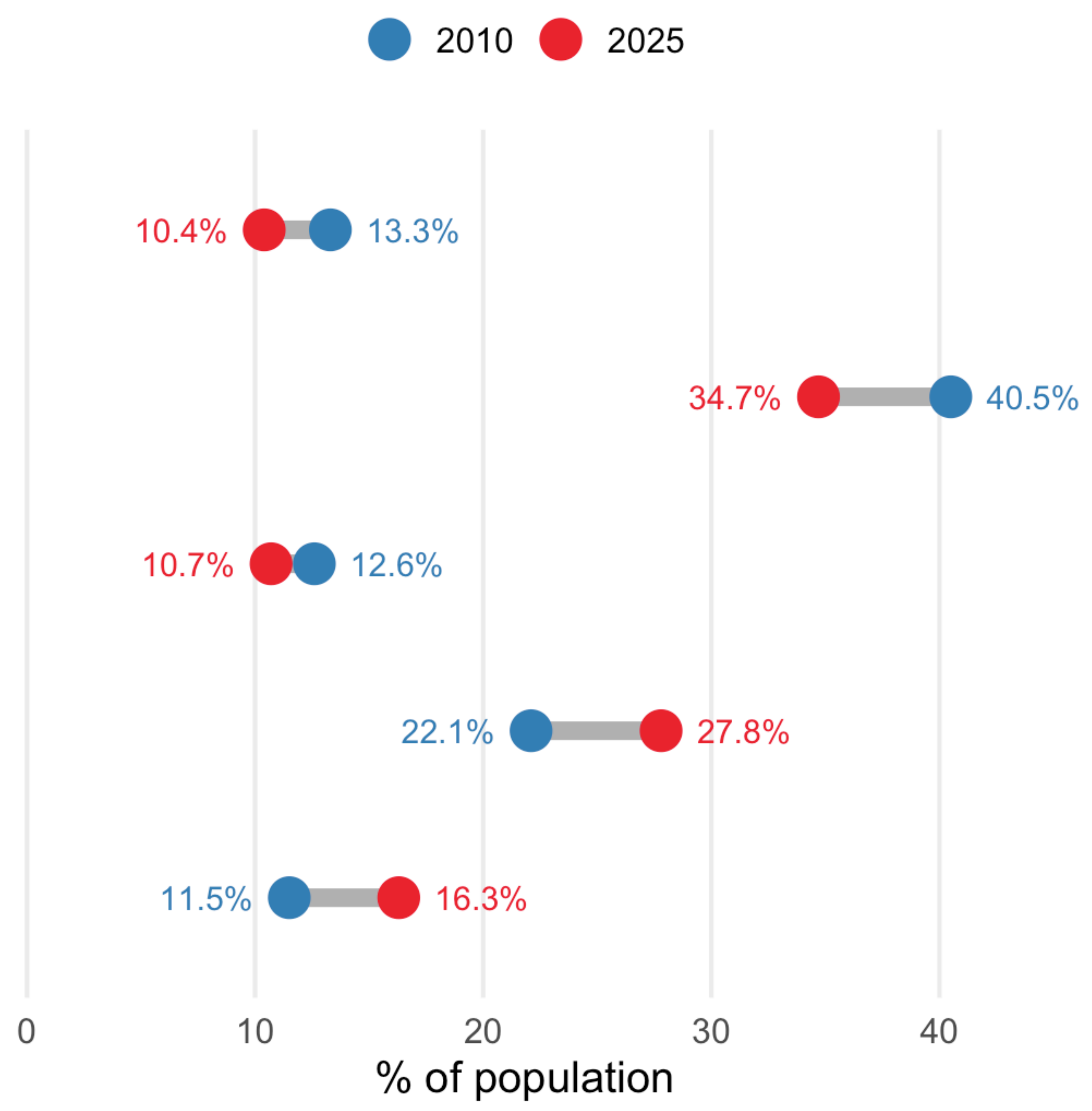
Bridge between descriptive and causal

What if?

Techniques often involve 'counterfactual' scenarios

Danish smoking has declined from 21% (2010) to 11% (2025)





Covered Today

1. Kitagawa's Decomposition
2. Oaxaca–Blinder Decomposition
3. Concentration Index Decomposition
4. Interventional Decomposition

1. Kitagawa's Decomposition

COMPONENTS OF A DIFFERENCE BETWEEN TWO RATES*

EVELYN M. KITAGAWA
University of Chicago and Scripps Foundation

WHEN comparing the incidence of some phenomenon in two or more groups, social researchers place much emphasis on the need for holding constant those related factors that would tend to distort the comparison. For example, before comparing the death rates for the residents of two areas, demographers frequently control the factors of differences between the areas in age, sex and race composition. A tech-

If standardization alters a difference between two rates, it should be possible … to break it up into components attributable to the various factors for which the data were standardized.

'Unpacking' the difference between crude rates of two groups

Potential consequences of ignoring composition

Impact of upward social mobility on population mortality: analysis with routine data

Richard F Heller, Patrick McElduff, Richard Edwards

> may give a distorted view of the overall level of health inequality due to social class differentials as it does not take into account the changing distribution of social class within the whole population.

> changes in the distribution of social class across the population are an important contributor to recent reductions in mortality

Group rate: $R_i = y_i/n_i$

Group share: $P_i = n_i/\sum_i n_i$

Crude rate: $\frac{\sum_{i=1}^I y_i}{\sum_{i=1}^I n_i} = \sum_{i=1}^I P_i R_i$

Standardized rate:

$\frac{\sum_{i=1}^I y_i}{\sum_{i=1}^I s_i} = \sum_{i=1}^I S_i R_i$

where S_i is the share of group i in a *standard* population ($\sum_i S_i = 1$)

Data for crude rates

Stratum	Pop	Count	Rate
1	n_i	y_i	r_i
$\vdots$	$\vdots$	$\vdots$	$\vdots$
I	N	Y	R

Age	Group A		Group B	
	Share	Rate	Share	Rate
Young	30%	10	50%	20
Old	70%	40	50%	30
Crude		**31**		**25**
Standardized to A		**31**		**27**

Standardization question:

What would the $A - B$ difference be *if B had A's age distribution*?

Kitagawa's key insight

> ...if the difference between two standardized rates is subtracted from the corresponding difference in crude rates...the result is a weighted average of differences between the composition of the two groups.

But what are the weights?

Crude $A - B$ difference = $\sum P_{Ai}R_{Ai} - \sum P_{Bi}R_{Bi}$

→ Reflects differences in both rates *and* composition

A-Standardized $A - B$ difference = $\sum P_{Ai}R_{Ai} - \sum P_{Ai}R_{Bi}$

→ Reflects *only* differences in rates (P_{Ai} is constant)

Crude - Standardized = $\sum R_{Bi}(P_{Ai} - P_{Bi})$

→ Differences in composition, *weighted* by Bs rates!

Applying the decomposition

$$\underbrace{\text{Rate}_A - \text{Rate}_B}_{\text{crude difference}} = \overbrace{\sum_i \left(\frac{R_{Ai} + R_{Bi}}{2}\right)(P_{Ai} - P_{Bi})}^{\text{composition effect}}$$

$$+ \underbrace{\sum_i \left(\frac{P_{Ai} + P_{Bi}}{2}\right)(R_{Ai} - R_{Bi})}_{\text{rate effect}}$$

R_{Ai} is the rate for group A in stratum i.

P_{Ai} is the i-stratum-specific weight in group A.

Age	Group A		Group B	
	Share	Rate	Share	Rate
Young	30%	10	50%	20
Old	70%	40	50%	30

Composition effect

Group	$\frac{R_{Ai}+R_{Bi}}{2}$	$P_{Ai} - P_{Bi}$	Contribution
Young	15	−0.20	−3.0
Old	35	+0.20	+7.0
Sum			**+4.0**

Rate effect

Group	$\frac{P_{Ai}+P_{Bi}}{2}$	$R_{Ai} - R_{Bi}$	Contribution
Young	0.40	−10	−4.0
Old	0.60	+10	+6.0
Sum			**+2.0**

Total = **+6.0** = crude gap (31 – 25) → 67% due to composition

Rate effect (+2) = standardized gap (avg reference) (28 – 26)

Kitagawa: does the reference group matter?

Reference	Composition	Rate	Total
A's rates	6.0	0.0	6.0
B's rates	2.0	4.0	6.0
Average (Kitagawa)	**4.0**	**2.0**	**6.0**

Crude gap = 31 – 25 = **6** in every row; only the split changes

The one-sided rows differ by the interaction, $\sum_i (P_{Ai} - P_{Bi})(R_{Ai} - R_{Bi})$ = 4.0, which the average splits evenly

What accounts for differences in neonatal mortality rates across European countries?

GA-specific rates *or* distribution?

Implications for interventions?

Original Investigation | Pediatrics

Neonatal Mortality Disparities by Gestational Age in European Countries

Victor Sartorius, MD; Marianne Philibert, MSc; Kari Klungsoyr, PhD; Jeannette Klimont, MSc; Katarzyna Szamotulska, PhD; Zeljka Drausnik, MPH; Petr Velebil, PhD; Laust Mortensen, PhD; Mika Gissler, PhD; Jeanne Fresson, MD; Jan Nijhuis, PhD; Wei-Hong Zhang, PhD; Karin Källén, PhD; Tonia A. Rihs, PhD; Vlad Tica, PhD; Ruth Matthews, PhD; Lucy Smith, PhD; Jennifer Zeitlin, DSc; for the Euro-Peristat Network

Abstract

IMPORTANCE There are wide disparities in neonatal mortality rates (NMRs, deaths <28 days of life after live birth per 1000 live births) between countries in Europe, indicating potential for improvement. Comparing country-specific patterns of births and deaths with countries with low mortality rates can facilitate the development of effective intervention strategies.

OBJECTIVE To investigate how these disparities are associated with the distribution of gestational age (GA) and GA-specific mortality rates.

DESIGN, SETTING, AND PARTICIPANTS This was a cross-sectional study of all live births in 14 participating European countries using routine data compiled by the Euro-Peristat Network. Live births with a GA of 22 weeks or higher from 2015 to 2020 were included. Data were analyzed from May to October 2023.

EXPOSURES GA at birth.

MAIN OUTCOMES AND MEASURES The study investigated excess neonatal mortality, defined as a rate difference relative to the pooled rate in the 3 countries with the lowest NMRs (Norway, Sweden, and Finland; hereafter termed the top 3). The Kitagawa method was used to divide this excess into the proportion explained by the GA distribution of births and by GA-specific mortality rates. A sensitivity analysis was conducted among births 24 weeks' GA or greater.

RESULTS There were 35 094 neonatal deaths among 15 123 428 live births for an overall NMR of 2.32 per 1000. The pooled NMR in the top 3 was 1.44 per 1000 (1937 of 1 342 528). Excess neonatal mortality compared with the top 3 ranged from 0.17 per 1000 in the Czech Republic to 1.82 per 1000 in Romania. Excess deaths were predominantly concentrated among births less than 28 weeks' GA (57.6% overall). Full-term births represented 22.7% of the excess deaths in Belgium, 17.8% in France, 40.6% in Romania and 17.3% in the United Kingdom. Heterogeneous patterns were observed when partitioning excess mortality into the proportion associated with the GA distribution vs GA-specific mortality. For example, these proportions were 9.2% and 90.8% in France, 58.4% and 41.6% in the United Kingdom, and 92.9% and 7.1% in Austria, respectively. These associations remained stable after removing births under 24 weeks' GA in most, but not all, countries.

CONCLUSIONS AND RELEVANCE This cohort study of 14 European countries found wide NMR disparities with varying patterns by GA. This knowledge is important for developing effective strategies to reduce neonatal mortality.

JAMA Network Open. 2024;7(8):e2424226. doi:10.1001/jamanetworkopen.2024.24226

Key Points

Question Are disparities in neonatal mortality between European countries associated with differences in the gestational age distribution of births or with higher mortality at specific gestational ages?

Findings In this cross-sectional study of over 15 million live births from 14 European countries, heterogeneous patterns were observed across countries and gestational age groups in the association of these 2 components with excess mortality in comparison with the 3 countries with the lowest neonatal mortality rates (Sweden, Norway, and Finland).

Meaning Stratifying neonatal mortality rates by gestational age can provide country-specific insights to inform preventive strategies to reduce neonatal mortality.

+ Supplemental content

Author affiliations and article information are listed at the end of this article.

Hiding in plain sight?

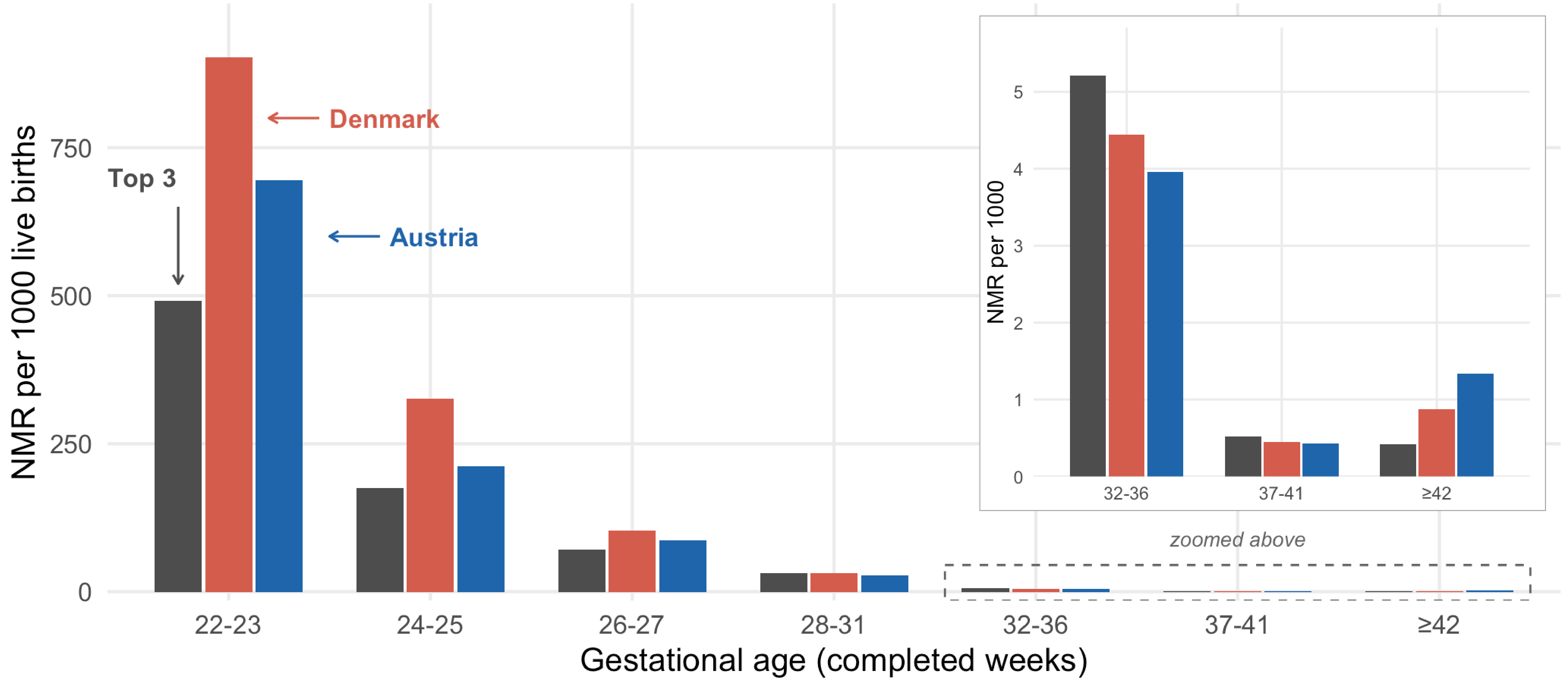
Both DK and AT similar and ~0.3 deaths per 1000 higher than "Top 3"

But *completely different* contributions to the difference

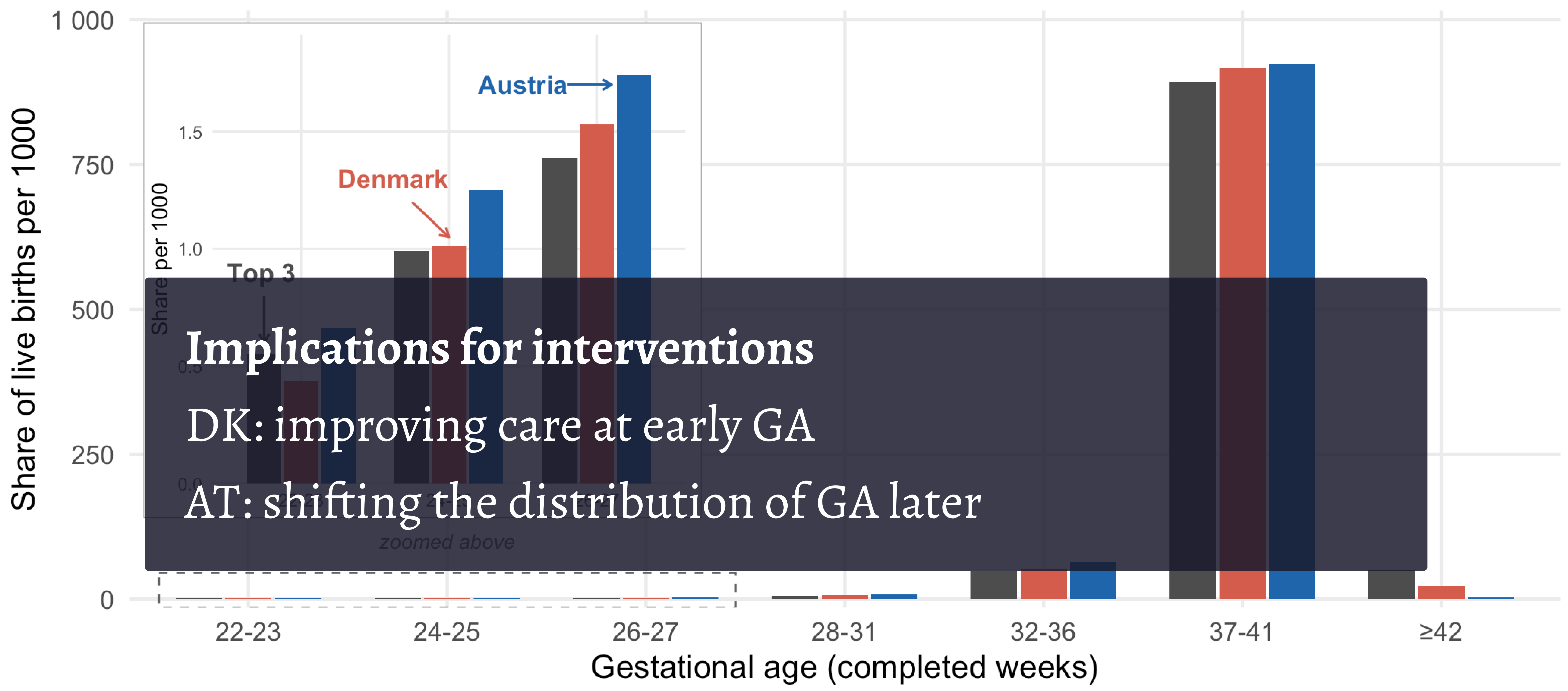
Table 3. Neonatal Mortality Rate (NMR) Differences for Each Country With Sweden, Norway, and Finland and the Kitagawa Decomposition Into the Differences Attributed to the Gestational Age (GA) Distribution and to GA-Specific NMRs Among Live Births at 22 Weeks or More and 24 Weeks or More of GA[a]

Country	NMR per 1000 live births	Difference from Finland, Norway, and Sweden	% of the Difference attributed to	
			GA distribution	GA-specific NMR
Live births ≥22 weeks of GA				
Finland, Norway, and Sweden	1.44	NA	NA	NA
Czech Republic	1.61	0.17	27.7	72.3
Austria	1.73	0.28	92.9	7.1
Denmark	1.74	0.30	0.0	100.0

Denmark has much worse rates at early GA



Austria more births at earlier GA



Implications for interventions

DK: improving care at early GA

AT: shifting the distribution of GA later

Summary: Kitagawa's decomposition

When to use

Comparing rates across groups or over time when their composition differs

Extensions

More than two groups or factors; three-part decomposition

Caveats

Not causal; results depend on the strata used and on the reference weights

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2. Oaxaca–Blinder Decomposition

Oaxaca-Blinder: Basic Idea

What explains average differences in outcomes? In a regression context:

1. Means

Differences in the prevalence of determinants of outcome

2. Coefficients

Differences in the coefficient of a given determinant on the outcome (i.e., effect measure modification)

Eur J Health Econ (2011) 12:17–28
DOI 10.1007/s10198-010-0220-z

ORIGINAL PAPER

Inequalities in the use of health services between immigrants and the native population in Spain: what is driving the differences?

Dolores Jiménez-Rubio · Cristina Hernández-Quevedo

Our findings show that unexplained factors associated to immigrant status determine to a great extent disparities in the probability of using hospital, specialist and emergency services of immigrants relative to Spaniards, while individual characteristics, in particular self-reported health and chronic conditions, are much more important in explaining the differences in the probability of using general practitioner services between immigrants and Spaniards

Origins

RESEARCH ARTICLE

Oaxaca-Blinder meets Kitagawa: What is the link?

Ronald L. Oaxaca[1,2,3☯], **Eva Sierminska**[1,2,3,4,5,6☯¤*]

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Studies by R. Oaxaca (1973) and Blinder (1973) applied regression-based decomposition methods to analyze the wage gap between men and women and between whites and blacks in the USA.

Focused on how much of wage gap was ‘explained’ by differences in observable characteristics

Recent attempts to reconcile O-B with Kitagawa (equivalent for binary outcome and categorical covariates)

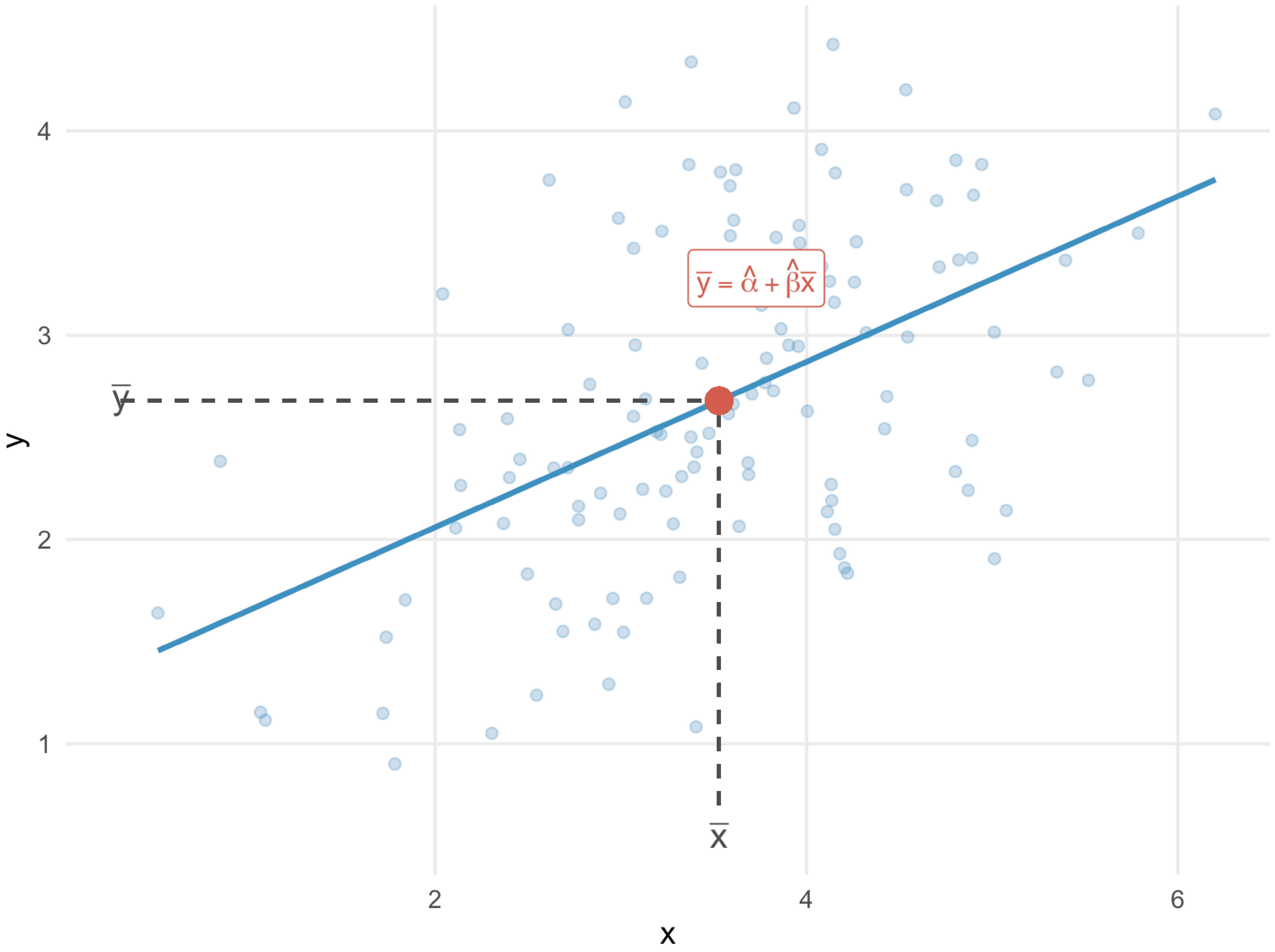
Brief note on interpretation

Decomposition methods are based on regression analyses, and thus all of the usual caveats about good specification apply.

If regressions are purely descriptive, they reveal the associations that characterize the health inequality. Then inequality is explained in a statistical sense but implications for policies to reduce inequality are limited.

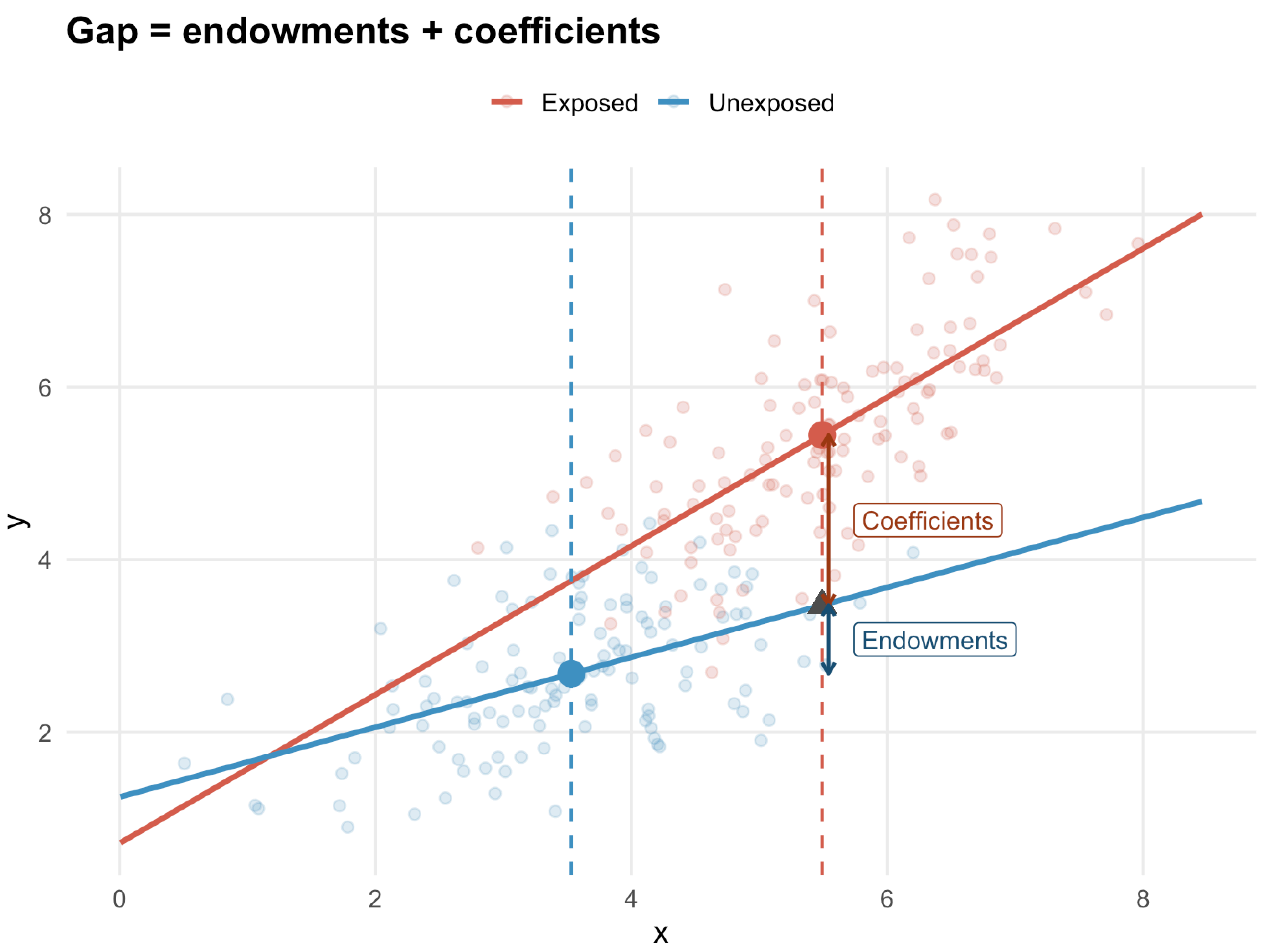
Why does this work?

OLS regression line always passes through $(\bar{x}, \bar{y})$ because residuals sum to zero.



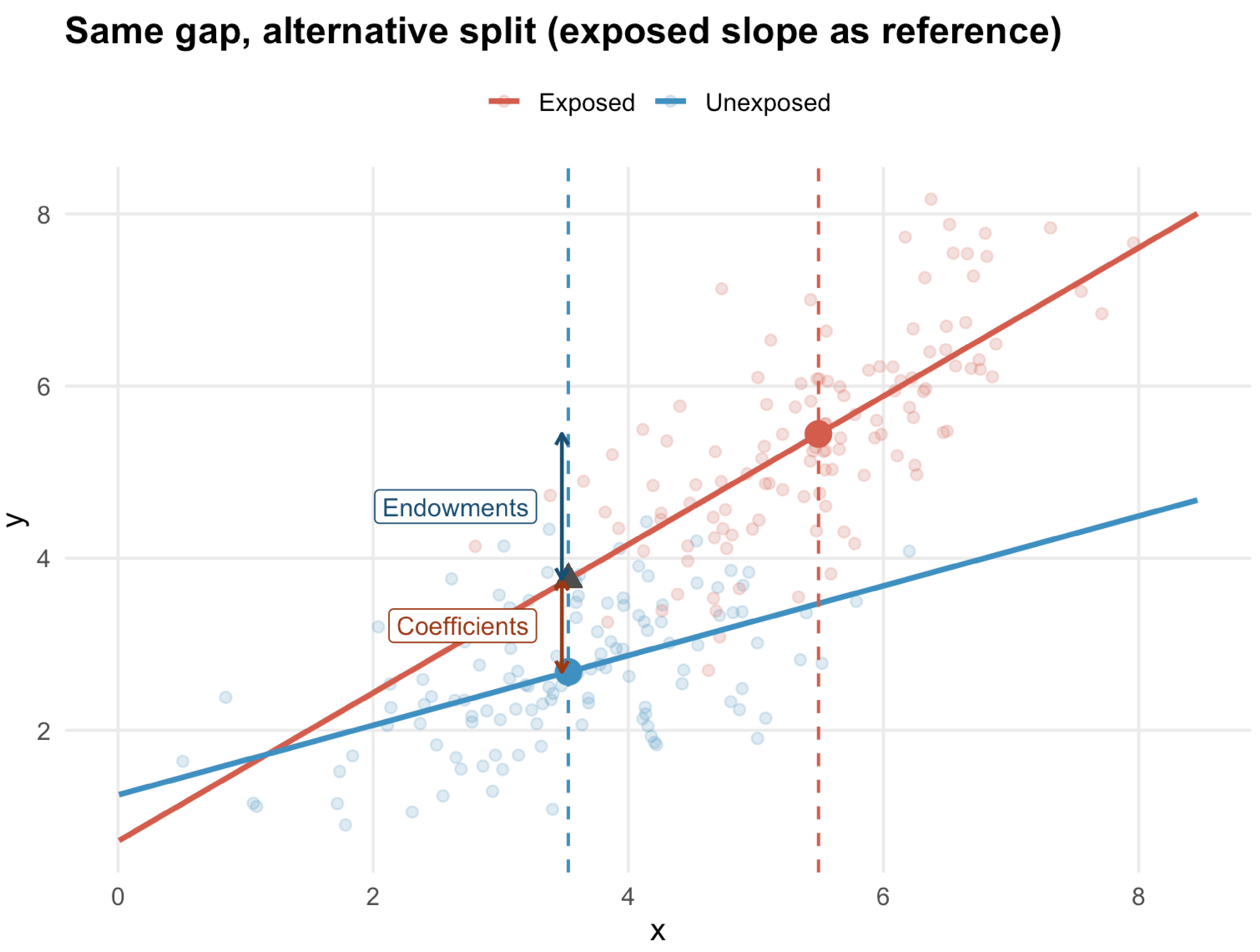
Why does this work?

The gap decomposes into **endowments** (different $\bar{x}$, same coefficients) and **coefficients** (same $\bar{x}$, different slopes/intercepts).



Equally valid to use the **exposed** group's slope

Same total gap, but different split



Example: Educational Differences in BMI in Italy

What is the average difference in body mass index (BMI) between those with low vs. high education?

How much is due to *differing determinants* of BMI (age, gender, smoking, drinking, income)?

Any residual difference is due to education differences in the *associations* between those risk factors and BMI — i.e., the coefficients differ.

Example data

European Social Survey Round 11, Italy ($n = 1743$)

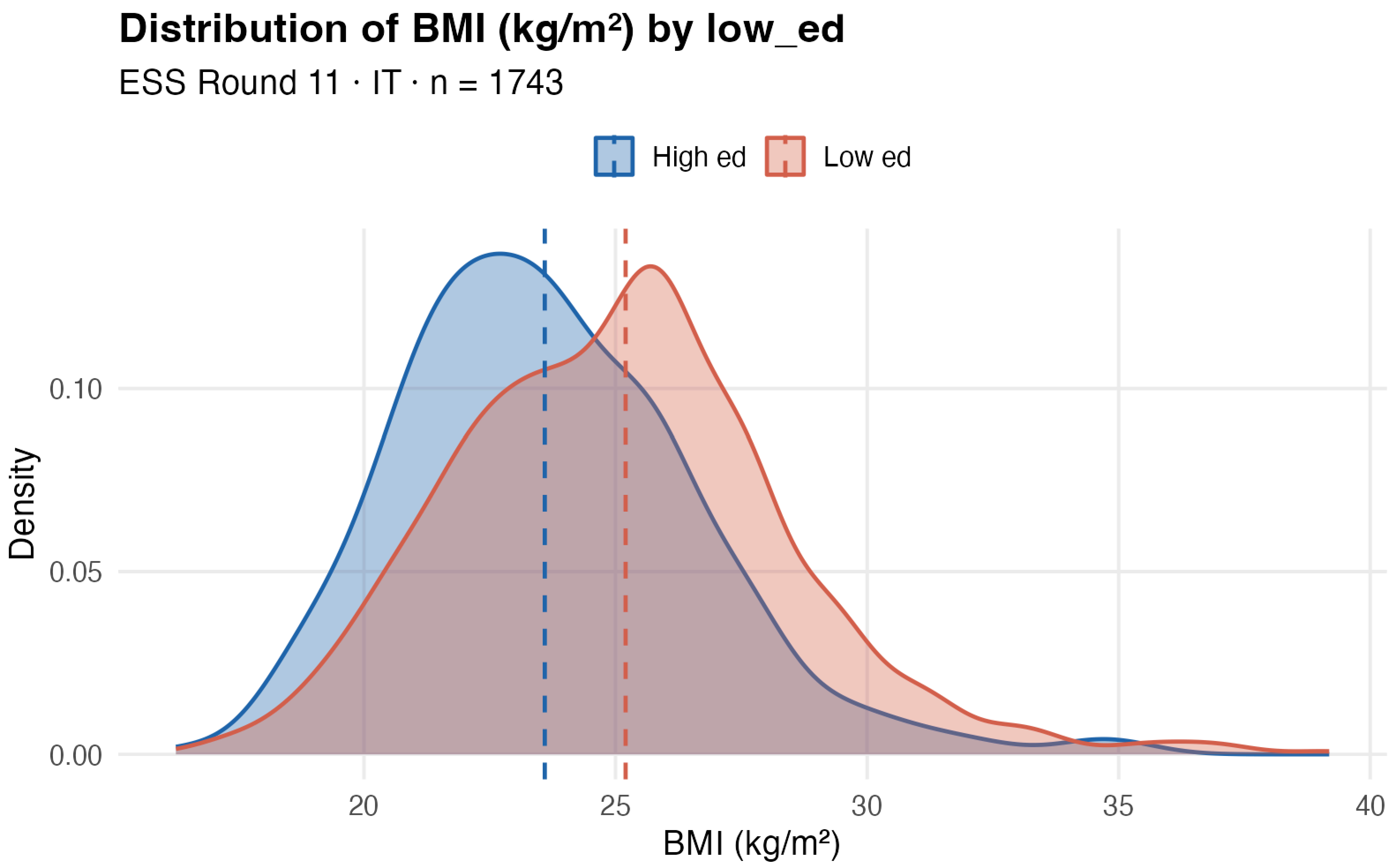
Body mass index as outcome (kg/m^2): `weight / height²`

Overall difference by education: High ed (ISCED 5–7) vs Low ed (ISCED 1–4)

Potential determinants (the Xs):

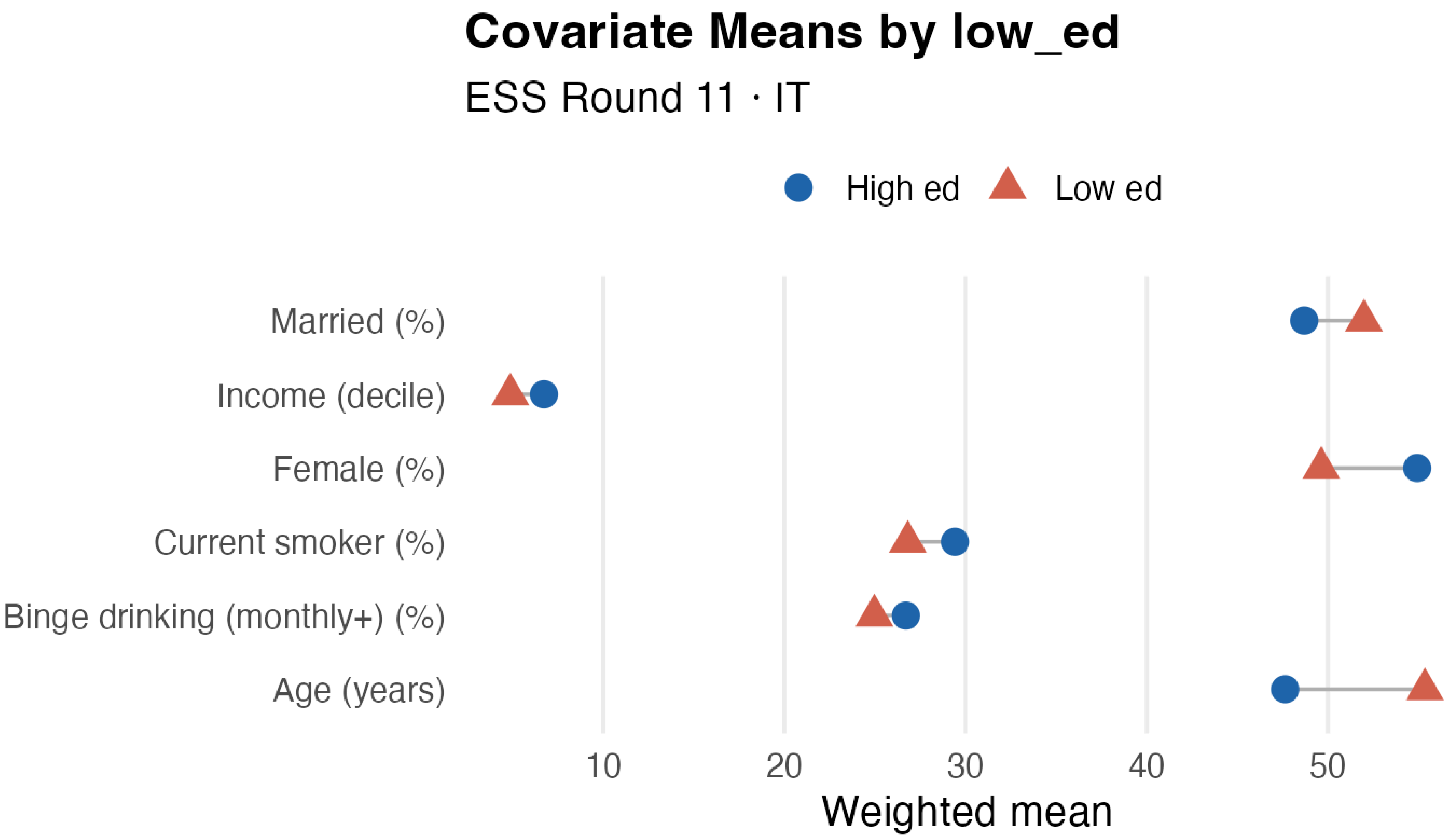
- age (years)
- gender (female = 1)
- smoking (1 = current smoker)
- binge drinking (1 = monthly or more)
- married (1 = currently married)
- income decile (1–10)

High ed BMI: 23.6, Low ed BMI: 25.2, Gap = 1.6



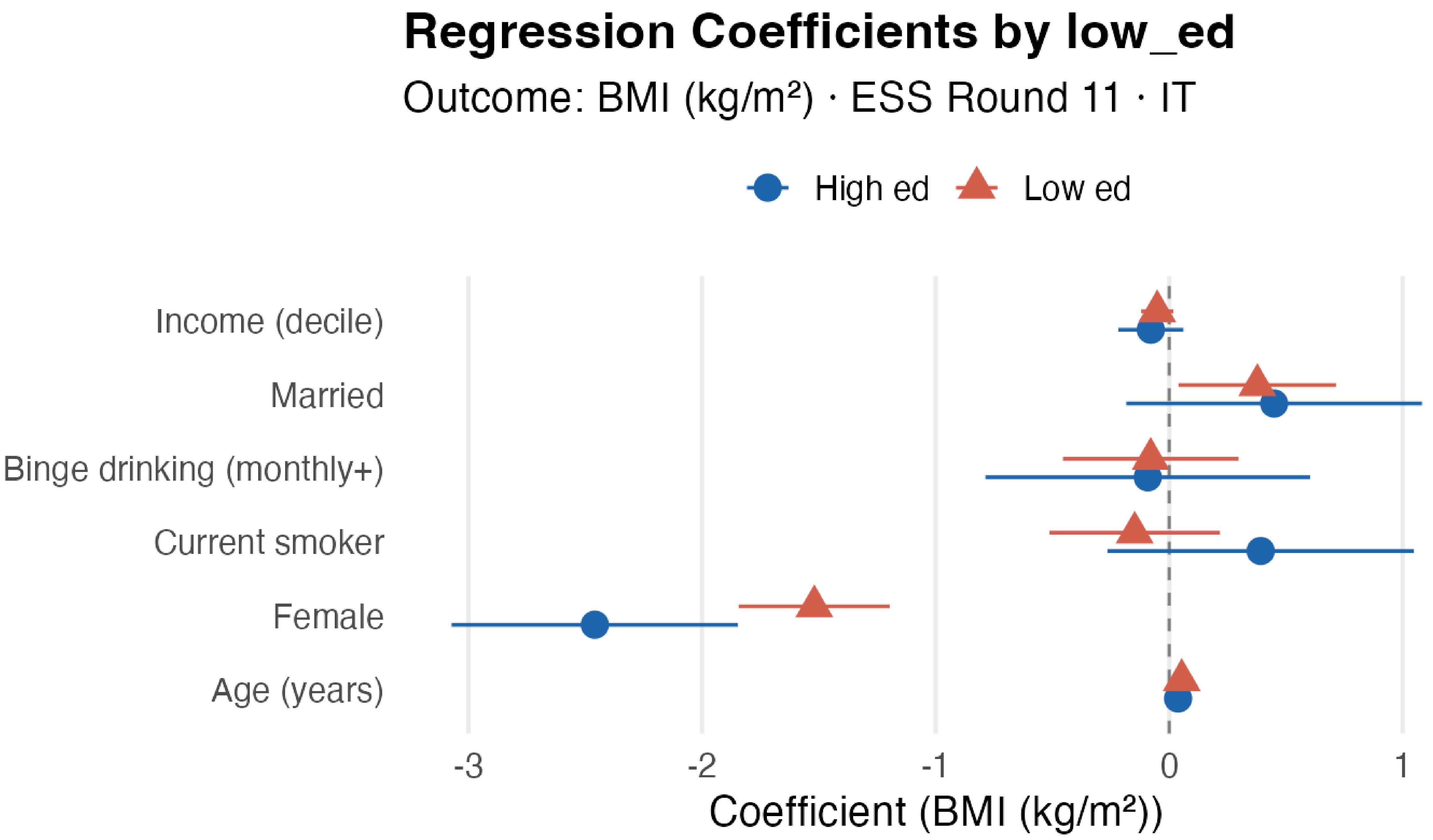
Differences in determinants

Differences in age, smoking, etc. could explain part of the BMI gap



Differences in coefficients

- Each covariate may have a different association with BMI by education
- Differences in *returns* form the unexplained component



Decomposition results: variable contributions

Overall gap = 1.61 kg/m². Endowments: 38%; Coefficients: 62%

	Endowments		Coefficients	
	Est	SE	Est	SE
Total	**0.608**	**0.108**	**1.030**	**0.259**
Age (years)	0.415	0.074	0.865	0.691
Female	0.080	0.051	0.466	0.181
Current smoker	0.004	0.008	-0.145	0.115
Binge drinking (monthly+)	0.001	0.007	0.003	0.104
Married	0.012	0.016	-0.037	0.208
Income (decile)	0.095	0.071	0.133	0.425
Intercept	—	—	-0.256	0.837

Summary: Endowments = 38%, Coefficients = 62%

	Endowments	
	Est	SE
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Female	0.080	0.051
Current smoker	0.004	0.008
Binge drinking (monthly+)	0.001	0.007
Married	0.012	0.016
Income (decile)	0.095	0.071

If the low-educated had the same covariate means as the high-educated (keeping the low-educated group's own coefficients), their BMI would be 0.608 kg/m^2 lower — closing 38% of the gap.

Most of this is due to older age among the low-educated, which predicts higher BMI.

Summary: Endowments = 38%, Coefficients = 62%

If the high-educated group's covariates had the same relationship with BMI as they do in the low-educated group, their BMI would be 1.030 higher, accounting for 62% of the gap.

Smoking is *negative*, since smoking predicts higher BMI among the high-educated and is more common among high educated.

	Coefficients	
	Est	SE
Total	**1.030**	**0.259**
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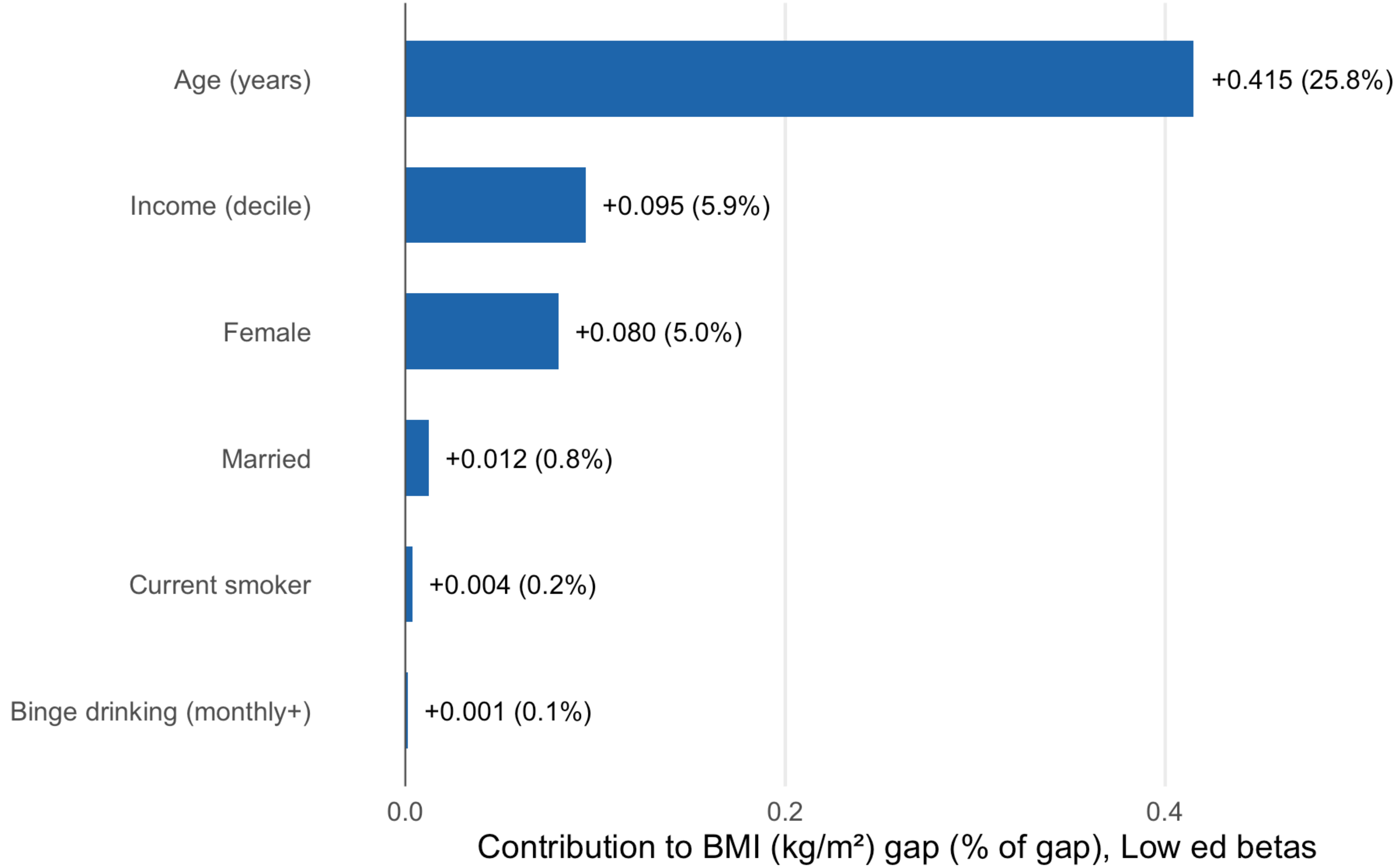
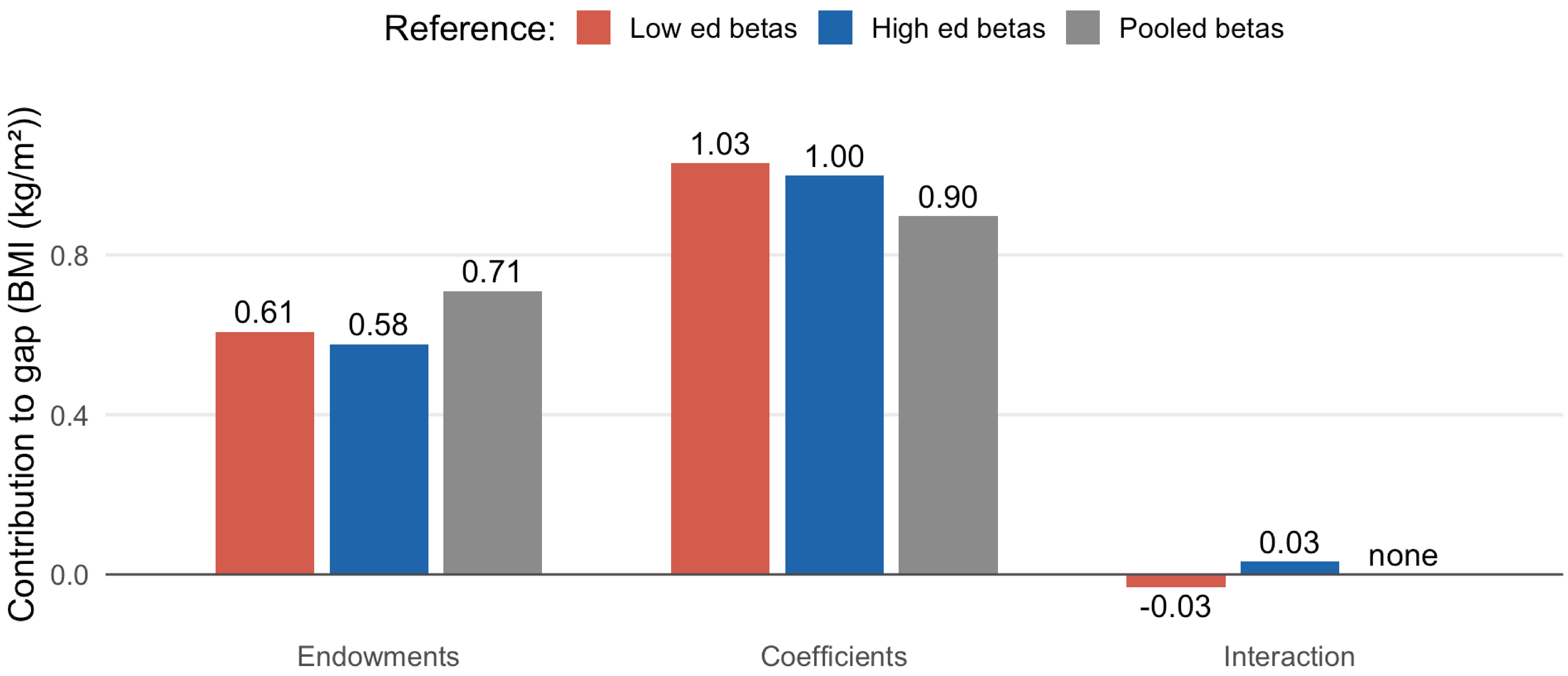
Income (decile)	0.133	0.425

Which covariates drive the endowments?

- Variables where groups differ *and* that predict BMI contribute most
- Income and age are the biggest drivers



Decomposition results: does the reference group matter?



Endowments account for **36–44%** of it; interaction is negligible

Summary: Oaxaca–Blinder decomposition

When to use

Explaining a gap in a mean between two groups by differences in covariates versus differences in their associations with the outcome

Extensions

Nonlinear outcomes (binary, counts); pooled reference coefficients; variable-by-variable contributions

Caveats

The split depends on the reference group and, for categorical covariates, the omitted category; associational, not causal

Covered Today

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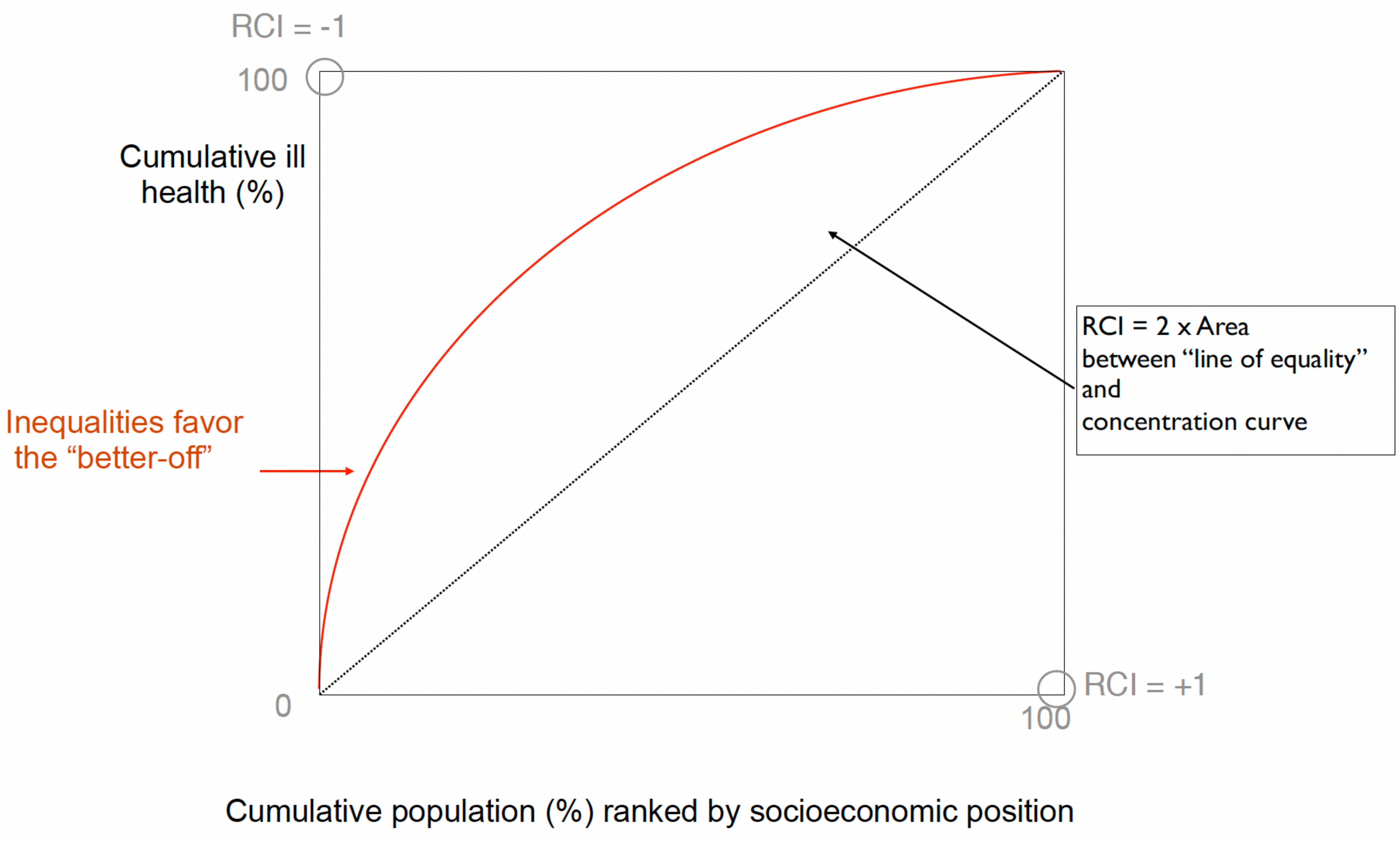
3. Concentration Index Decomposition

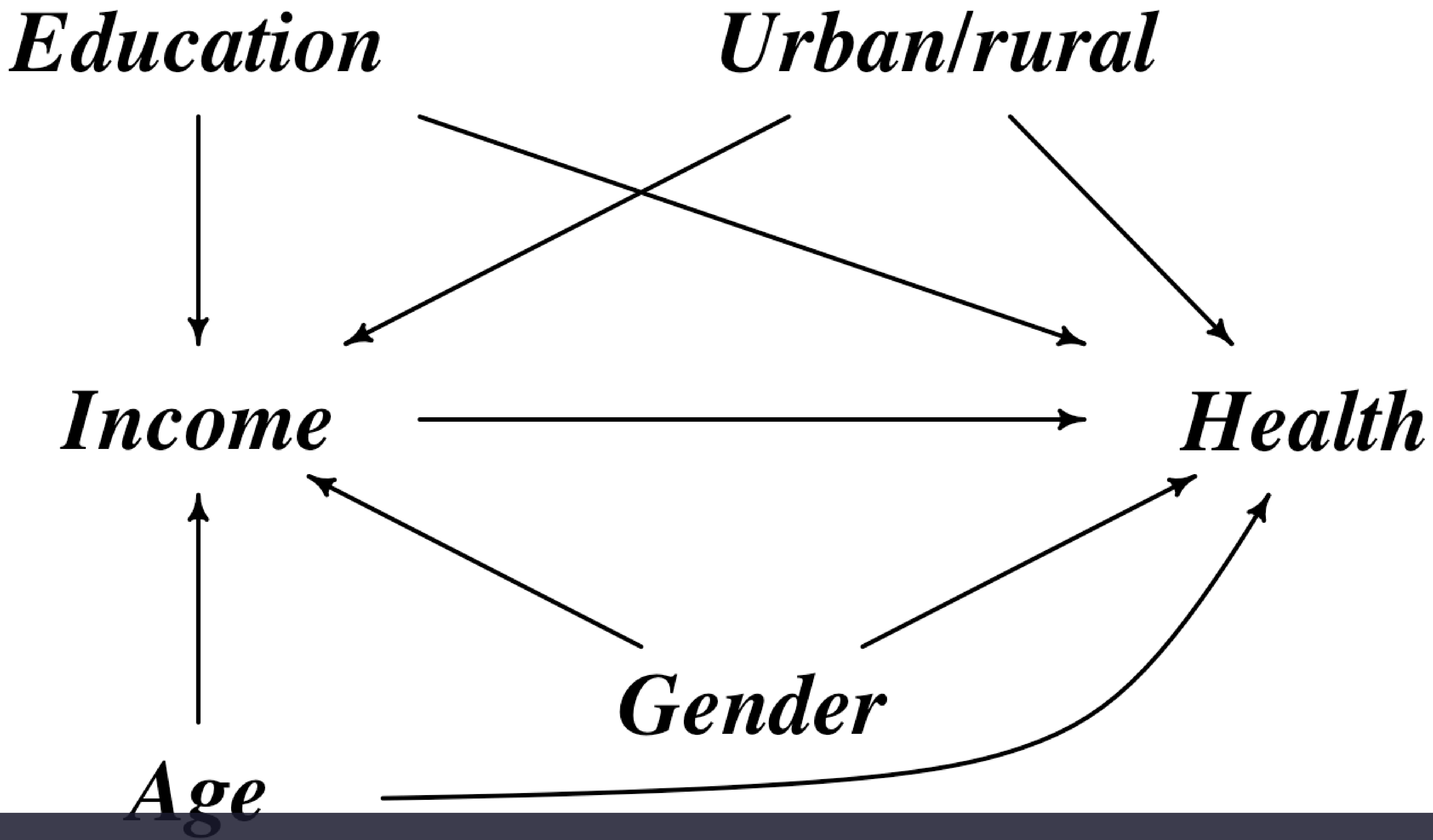
Relative Concentration Curve



We want to understand this:

Income ⟶ ***Health***

By evaluating something like this:



Research Question:

How much of inequality is due to other factors that are differential by x (income) and also affect y (health)?

Relative Concentration Index

$$RCI = \frac{2}{n\mu} \sum_{i=1}^{n} y_i R_i - 1$$

where

- μ is the mean of y_i (e.g., smoking);
- R_i is the fractional rank of the ith person in the socioeconomic (i.e., income) distribution.

Decomposition:

Develop a model for predicting y using several determinants, then plug back into the RCI equation

Estimates how much of the overall inequality in y is due to the association between income and other factors that predict health

RCI Decomposition

RCI is a function of health (y_i) and socioeconomic rank (R_i), i.e.

$$RCI = \frac{2}{n\mu}\sum_{i=1}^{n} y_i R_i - 1$$

Then write a regression expressing health (y_i) as a function of several k_i determinants (e.g., age, gender, urban/rural status):

$$y_i = \alpha + \sum \beta_x x_{k_i} + \epsilon_i$$

RCI Decomposition

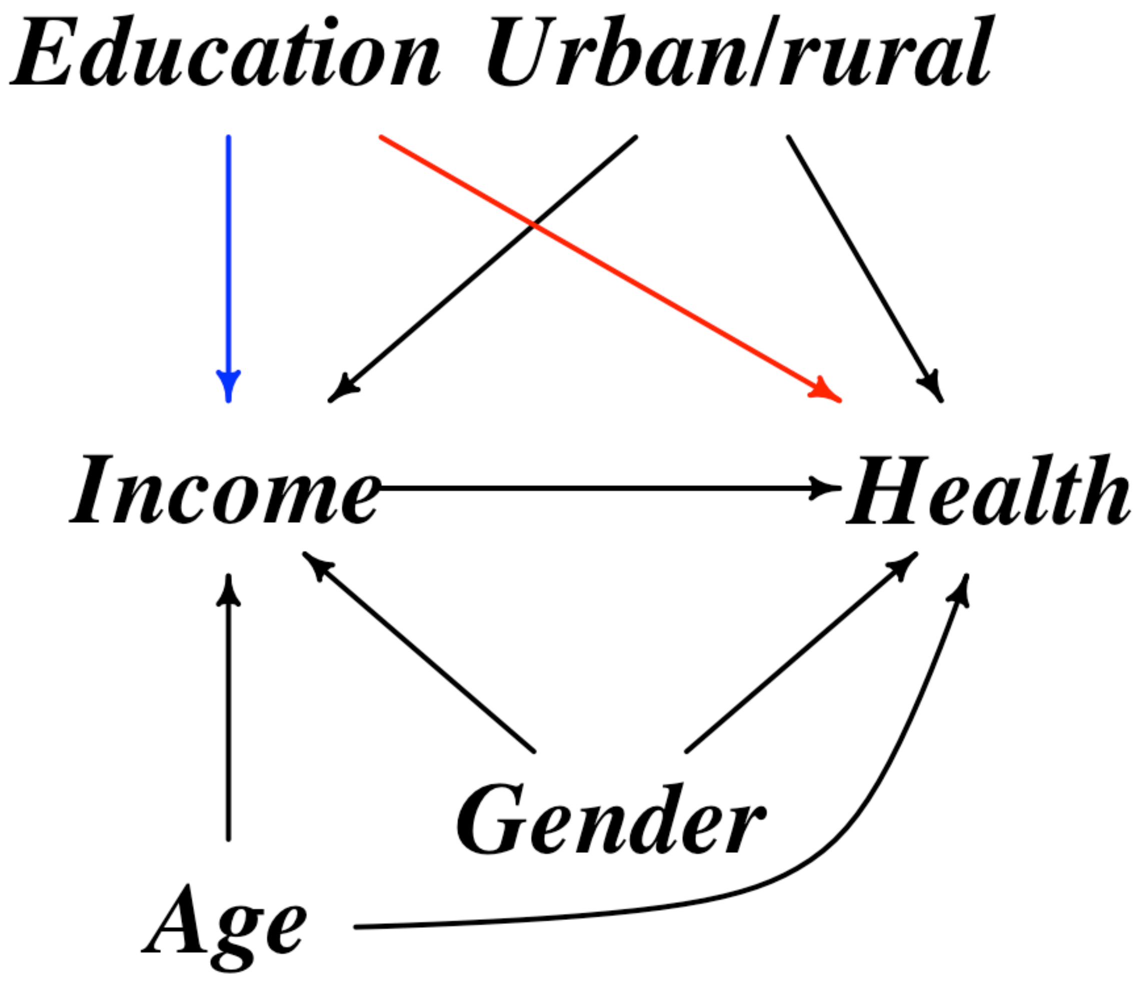
Now re-express RCI as:

$$RCI = \sum (\beta_k \bar{x}_k / \mu) RCI_k + gRCI_e / \mu$$

Where

- μ is the mean of y,
- $\bar{x}_k$ is the mean of determinant x_k,
- β_k is the regression coefficient for x_k, and
- RCI_k is the relative concentration index for x_k.

Two types of 'explained' components



Determinant impact depends on:

1. the strength of the relationship between each factor and income RCI_k
2. the strength of the relationship between each factor and health, and its prevalence in the population (elasticity) $\beta_k \bar{x}_k / \mu$

Procedure for decomposing the Concentration Index

1. Regress y ('health') on its determinants $(\beta_k x_k)$:

$$y_i = \alpha + \sum \beta_x x_{k_i} + \epsilon_i$$

2. Calculate means of y (μ) and all x_k determinants
3. Calculate (CI) for the health variable *and* for each determinant (CI_k). This means using each determinant x_k as the "outcome" and estimate a CI for age, CI for education, etc.

Procedure for decomposing the Concentration Index

4. Absolute contribution: for each x multiply 'elasticity' by its concentration index (CI_k):

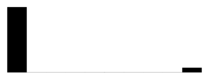
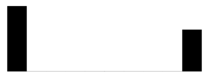
$$(\beta_k \bar{x}_k / \mu) RCI_k$$

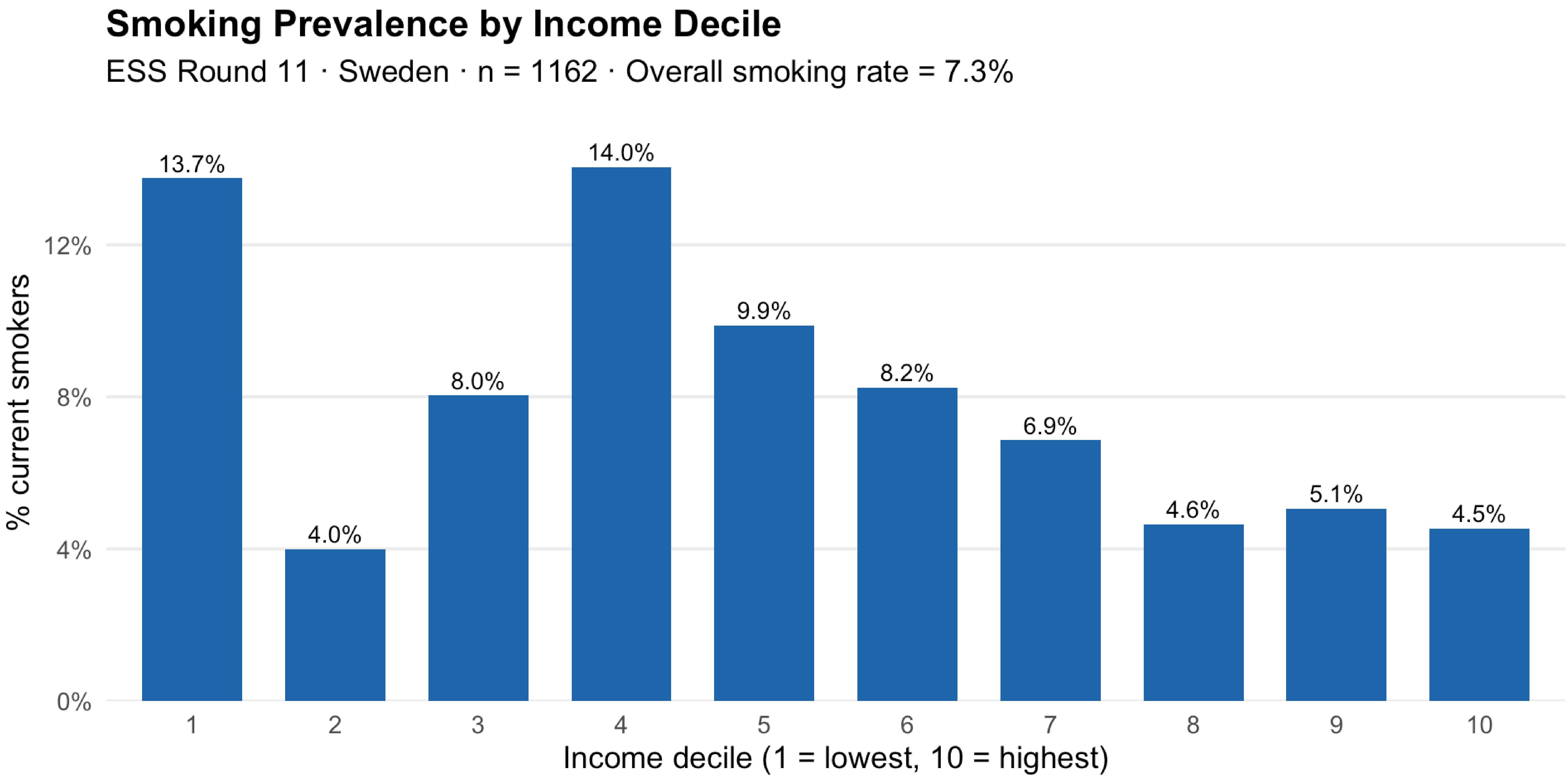
5. Calculate the % contribution of each determinant:

$$[(\beta_k \bar{x}_k / \mu) RCI_k] / RCI$$

Example: Decomposing Socioeconomic Inequality in Current Smoking

Data: European Social Survey Round 11 for Sweden

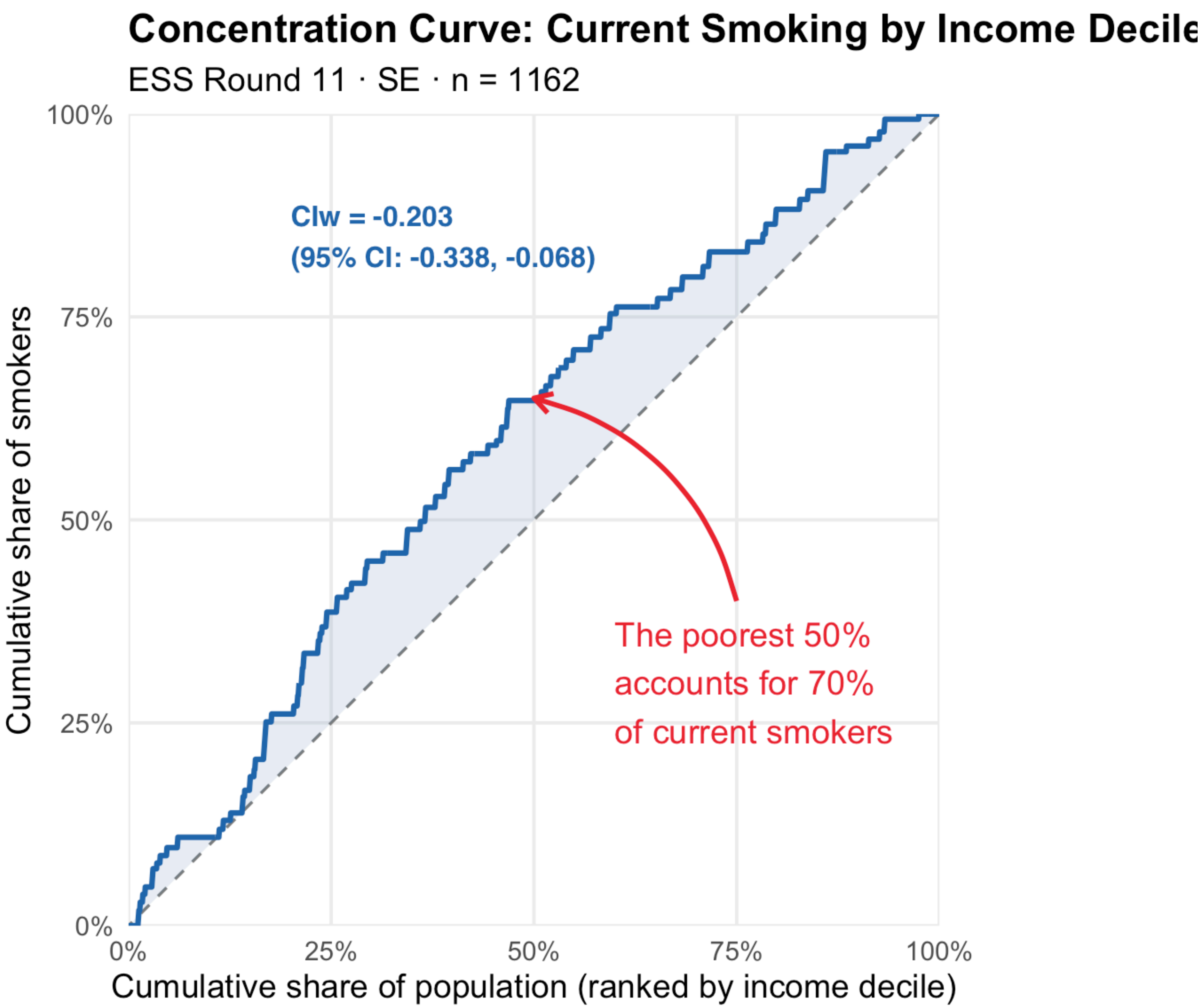
	Unique	Missing Pct.	Mean	SD	Min	Median	Max	Histogram
Current smoker	2	0	0.1	0.2	0.0	0.0	1.0	
Income decile	10	0	6.6	2.6	1.0	7.0	10.0	
Age (years)	76	0	54.4	18.6	15.0	56.0	90.0	
Low education (ISCED 1–4)	2	0	0.4	0.5	0.0	0.0	1.0	
Female	2	0	0.5	0.5	0.0	0.0	1.0	
Binge drinking (monthly+)	2	0	0.5	0.5	0.0	0.0	1.0	
Obese (BMI ≥ 30)	2	0	0.1	0.4	0.0	0.0	1.0	
Married	2	0	0.5	0.5	0.0	0.0	1.0	
Survey weight	132	0	1.0	0.5	0.3	0.8	4.0	


Smoking Prevalence by Income Decile
ESS Round 11 · Sweden · n = 1162 · Overall smoking rate = 7.3%
% current smokers
0%
4%
8%
12%
13.7%
4.0%
8.0%
14.0%
9.9%
8.2%
6.9%
4.6%
5.1%
4.5%
1
2
3
4
5
6
7
8
9
10
Income decile (1 = lowest, 10 = highest)

What are we explaining?

Overall CI = -0.20

Smoking *more concentrated* among the poor



Step 1: Predictors of current smoking

Variable	β	OR	95% CI	p
Age (years)	-0.001	0.999	(0.99, 1.01)	0.906
Female	0.141	1.152	(0.73, 1.83)	0.547
Low education (ISCED 1–4)	**0.359**	**1.432**	**(0.90, 2.27)**	**0.130**
Married	-0.989	0.372	(0.21, 0.65)	<0.001
Binge drinking (monthly+)	0.38	1.463	(0.92, 2.32)	0.107
Obese (BMI ≥ 30)	0.495	1.64	(0.90, 2.99)	0.106

Step 2: Estimating elasticity for education

$$RCI = \sum \underbrace{(\beta_k \bar{x}_k / \mu)}_{\text{elasticity}} RCI_k + gRCI_e / \mu$$

- $\beta_{\text{low education}}$ = 0.36 (OR = 1.4)
- Marginal effect: 0.024
- Mean low education: 0.452
- Mean smoking rate: 7.3%

Elasticity for low education is: (0.024 * 0.452 / .073) = 0.148

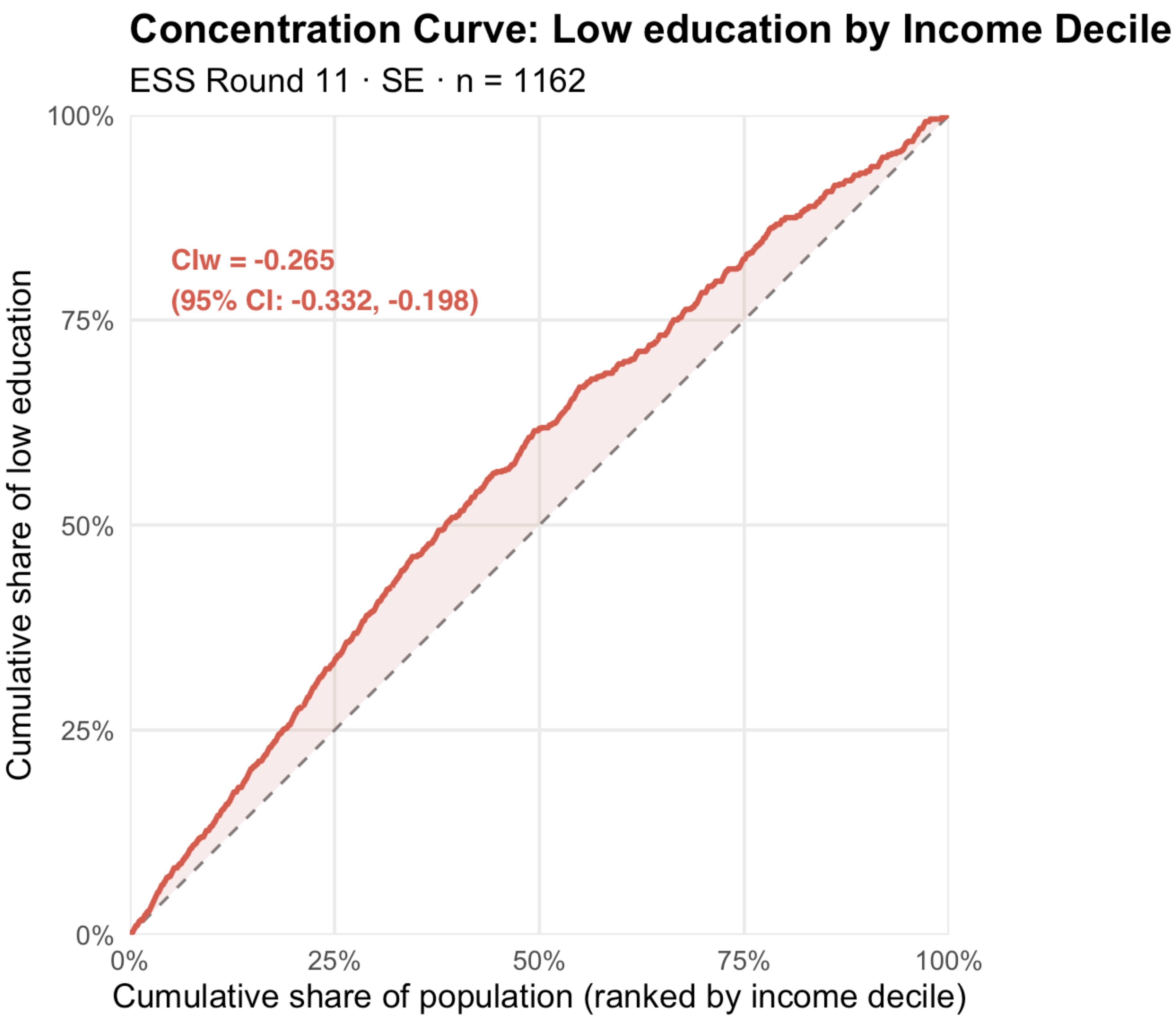
Interpretation: a 1% increase in low education increases smoking by 14.8% (not percentage points!).

What about the RCI for low education?

Step 3: RCI_k

Note the y-axis is cumulative share of *low education*

The poorest 50% account for roughly 60% of the share of low educated.



Step 4: Calculate contributions

Estimation for a specific factor: Low education

$$RCI = \sum \underbrace{(\beta_k \bar{x}_k / \mu)}_{\text{elasticity}} RCI_k + gRCI_e / \mu$$

The elasticity of smoking (prior slide) = 0.148

Now we have RCI_k for low education = -0.265

The contribution of low education:

$$\text{Elasticity} \times RCI_{ed} = 0.148 * -0.265 = -.039$$

Thus low education accounts for -.039/ -0.203 = 19% of the overall RCI

RCI = -0.203

(-) Binge drinking: ↑ smoking but more common at higher incomes

(+) Married: ↓ smoking but more common at higher incomes

			Contribution	
Variable	Elasticity	CI_k	Absolute	%
Age	-0.030	0.001	-0.000	0.0
Binge drinker	0.152	0.103	0.016	-7.7
Female	0.060	-0.031	-0.002	0.9
Obese	0.068	-0.013	-0.001	0.4
Low education	0.144	-0.265	-0.038	18.8
Married	-0.353	0.284	-0.100	49.4
Residual	—	—	-0.077	38.1

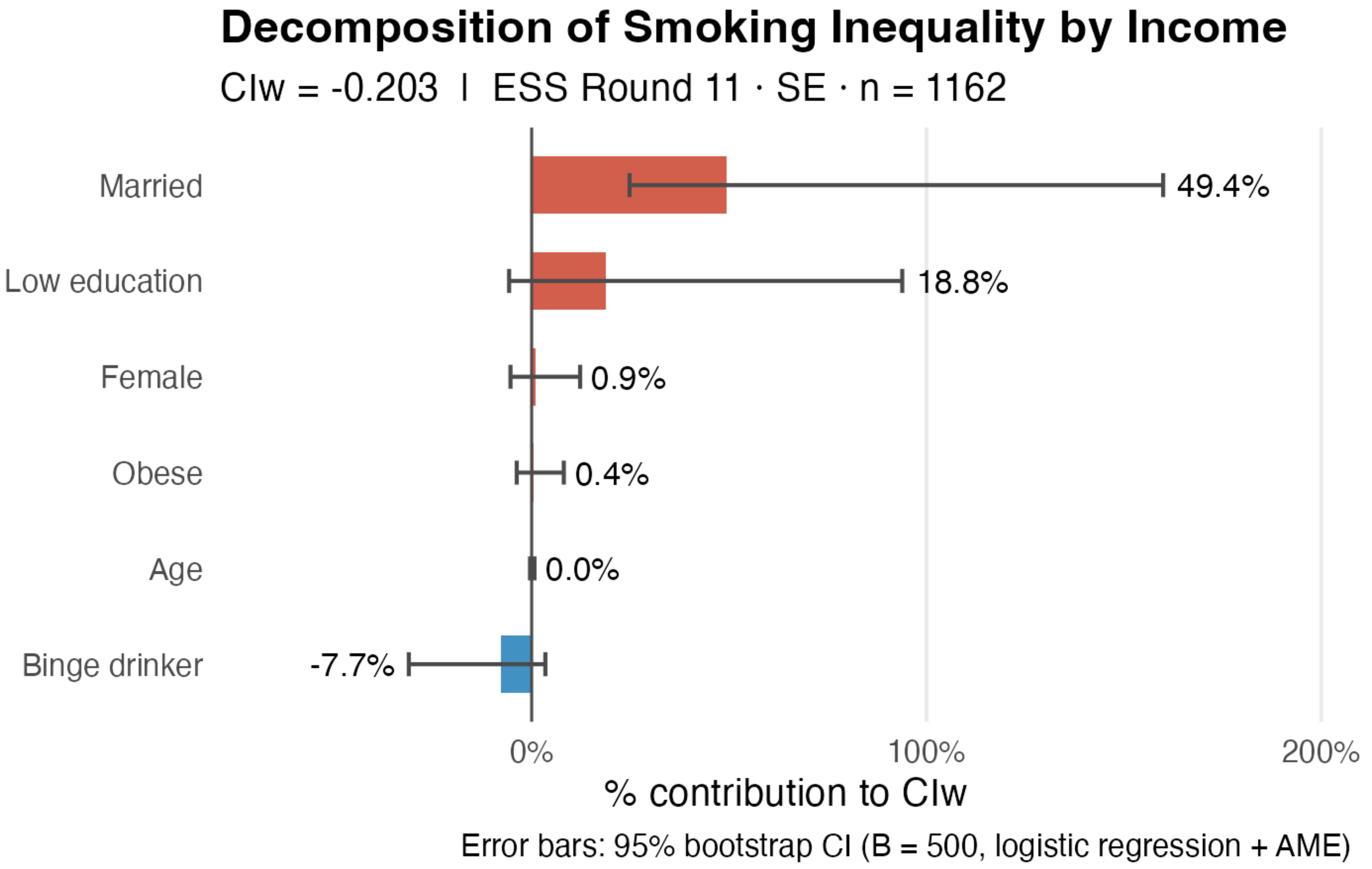
Logistic regression; marginal effects used as elasticity weights.

Uncertainty in the RCI Decomposition

Each contribution has sampling variability:

$$\underbrace{\frac{\hat{\beta}_k \bar{x}_k}{\hat{\mu}}}_{\text{elasticity}} \times \underbrace{\widehat{RCI}_k}_{\text{inequality in } x_k}$$

Can **bootstrap** the decomp B times to get empirical CIs.



Summary: Concentration index decomposition

When to use

Summarizing socioeconomic inequality in health with a single rank-based index and asking which determinants drive it

Extensions

Nonlinear models via marginal effects; structural-equation versions; bootstrap confidence intervals

Caveats

The explained share depends on which determinants enter; associations are predictive, not causal

Covered Today

1. **Kitagawa's Decomposition**
2. **Oaxaca–Blinder Decomposition**
3. **Concentration Index Decomposition**
4. **Interventional Decomposition**

4. Interventional Decomposition

Origins: Why ‘interventional’?

ORIGINAL ARTICLE

Decomposition Analysis to Identify Intervention Targets for Reducing Disparities

John W. Jackson,[a,b,c] *and Tyler J. VanderWeele*[c,d]

Abstract: There has been considerable interest in using decomposition methods in epidemiology (mediation analysis) and economics (Oaxaca–Blinder decomposition) to understand how health disparities arise and how they might change upon intervention. It has not been clear when estimates from the Oaxaca–Blinder decomposition can be interpreted causally because its implementation does not explicitly address potential confounding of target variables. While mediation analysis does explicitly adjust for confounders of target variables, it typically does so in a way that effectively entails equalizing confounders across racial groups, which may not reflect the intended intervention. Revisiting prior analyses in the National Longitudinal Survey of Youth on disparities in wages, unemployment, incarceration, and overall health with test scores, taken as a proxy for educational attainment, as a target intervention, we propose and demonstrate a novel decomposition that controls for confounders of test scores (e.g., measures of childhood socioeconomic status [SES]) while leaving their association with race intact. We compare this decomposition with others that use standardization (to equalize childhood SES [the confounders] alone), mediation analysis (to equalize test scores within levels of childhood SES), and one that equalizes both childhood SES and test scores. We also show how these decompositions, including our novel proposals, are equivalent to implementations of the Oaxaca–Blinder decomposition but provide a more formal causal interpretation for these decompositions.

Keywords: Causal inference, Health disparity, Inequity, Interventional effects, Mediation analysis, Path analysis, Path-specific effect, Oaxaca Blinder decomposition, Standardization

(*Epidemiology* 2018;29: 825–835)

Submitted March 17, 2017; accepted July 24, 2018.

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Data and Code Availability: https://github.com/jwjackson/SuppMaterials.

John Jackson was partly funded by the Alonzo Smythe Yerby Fellowship, and Tyler J. VanderWeele was funded by the National Institutes of health grant ES017876.

The authors report no conflicts of interest.

SDC Supplemental digital content is available through direct URL citations in the HTML and PDF versions of this article (www.epidem.com).

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ISSN: 1044-3983/18/2906-0825

DOI: 10.1097/EDE.0000000000000901

Health disparities are differences in health between socially advantaged versus disadvantaged groups that are considered unnecessary and unjust.[1] Although national and local efforts have sought to reduce and eliminate racial/ethnic disparities in health over the past few decades, they have often persisted.[2,3] Reducing racial/ethnic disparities requires that we understand how they arise and develop interventions to target the mechanisms that perpetuate them.[4]

Consider the seminal analyses by Fryer[5] in the National Longitudinal Survey of Youth (NLSY), patterned after analyses by Neal and Johnson,[6] which evaluated whether control for test score percentiles from the Armed Forces Qualifying Test, a measure of premarket skills, eliminated observed racial/ethnic disparities in wages, unemployment, incarceration, and health. Black–white differences in log wages decreased by 72%, and the disparity in self-reported health vanished. Though these analyses were originally motivated to statistically detect racial/ethnic discrimination, we are often tempted to interpret the results causally, that is, are these disparities driven by disparities in education? To answer this question, one would want to adjust for potential confounding by measures of familial socioeconomic status (SES) in childhood.[1,7] Otherwise, the disparity reductions might reflect the effect of equalizing childhood SES rather than just test scores. Such confounding, if present, could limit the value of these results for evidence-based policy to reduce disparities by targeting disparities in education.

More broadly, one might consider how disparities in wages and other outcomes would change by eliminating disparities in childhood SES versus disparities in test scores. While the queries involve ambitious, hypothetical interventions that could take many forms the results would help motivate and prioritize future work. Using the potential outcomes framework, one can examine how disparities might change by intervening on a target.[8] One might use standardization to examine how disparities might change upon equalizing the childhood SES distribution across race or use mediation analysis to examine how disparities might change upon equalizing the test score distribution across race among those with the same childhood SES.[9] One might even consider a joint intervention to equalize both childhood SES and test scores. But neither standardization nor mediation analysis examines how disparities in adult outcomes might change if we removed

Prior decompositions (e.g., Kitagawa, O-B) ‘descriptive’

Causal mediation challenges

Challenges with non-manipulable exposures

How much would gaps change if we intervened on a target?

Many recent developments

Reconciling the descriptive framework of KBO with causal inference.


ORIGINAL ARTICLE

Decomposition Analysis to Identify In[…] for Reducing Dispariti[…]

John W. Jackson,[a,b,c] and Tyler J. VanderW[…]

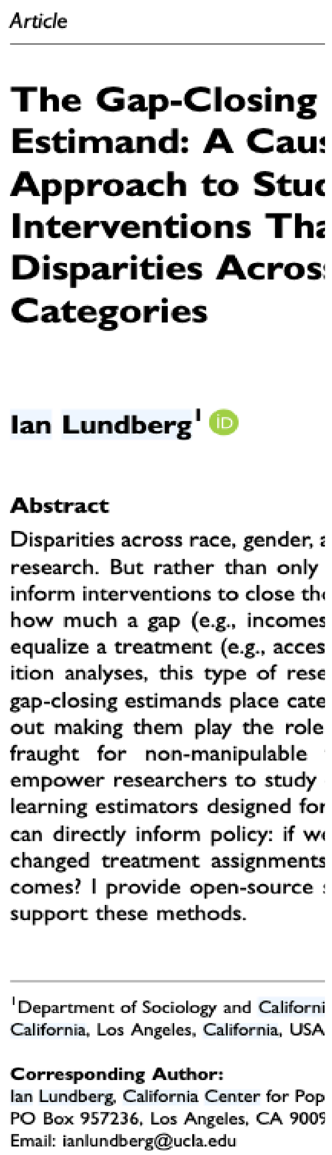
Epidemiology • Volume 29, Number 6, November 2018


ORIGINAL ARTICLE

Meaningful Causal Decompo[…] Health Equity Researc[…]

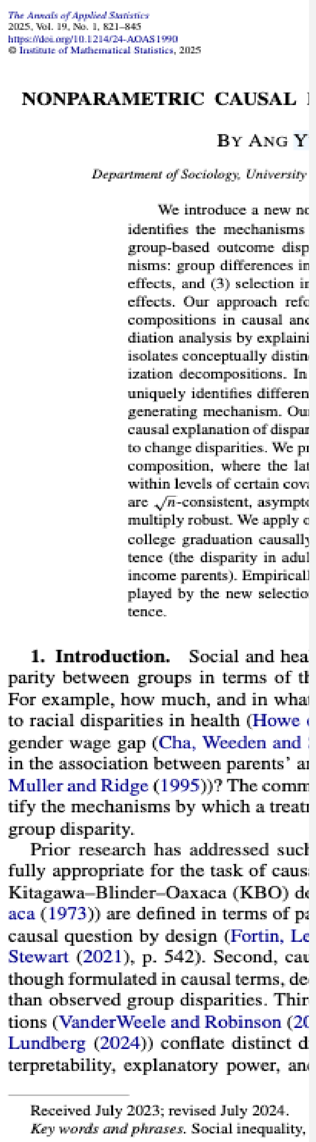
Definition, Identification, and Estimat[…] Weighting Framework

John W. Jackson[a,b,c,d,e]


Article

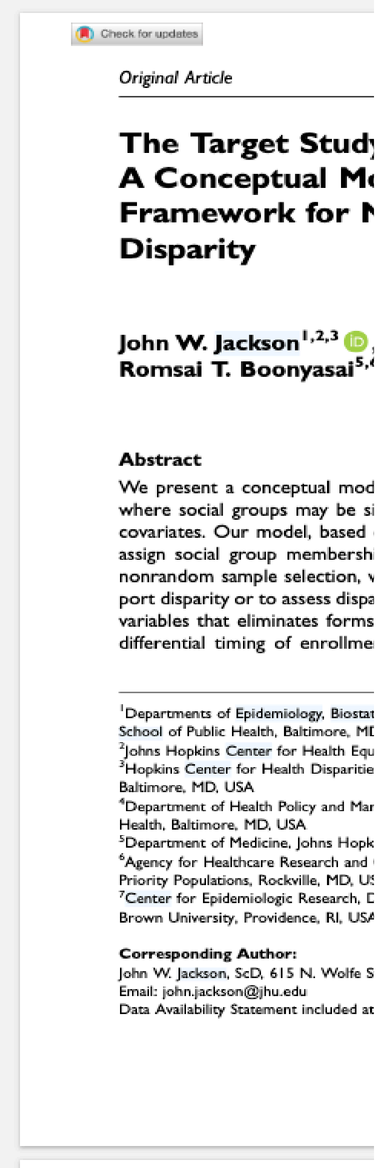
The Gap-Closing Estimand: A Caus[…] Approach to Stud[…] Interventions Tha[…] Disparities Acros[…] Categories

Ian Lundberg[1]


The Annals of Applied Statistics
2025, Vol. 19, No. 1, 821–845

NONPARAMETRIC CAUSAL […]

By Ang Y[…]


Original Article

The Target Stud[…] A Conceptual M[…] Framework for […] Disparity

John W. Jackson[1,2,3], Romsai T. Boonyasai[5,…]


Journal of the Royal Statistical Society Series A: Statistics in Society, 2026, 189, 805–833
https://doi.org/10.1093/jrsssa/qnaf008
Advance access publication 25 February 2025
Original Article

Disparity analysis: a tale of two approaches

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Abstract

To understand patterns of social inequality, social science research has typically relied on statistical models linking the conditional mean of an outcome variable to a set of explanatory factors. A prime example of this approach is the Kitagawa-Oaxaca-Blinder (KOB) method. By fitting two linear models separately for an advantaged group and a disadvantaged group, the KOB method decomposes the between-group outcome disparity into two parts: a part explained by group differences in background characteristics, and an unexplained part often dubbed 'residual inequality'. In this article, we explicate, contrast, and extend two distinct approaches to studying group disparities, which we term the descriptive approach, as epitomized by the KOB method and its variants, and the prescriptive approach, which focuses on how a disparity of interest would change under a hypothetical intervention to one or more manipulable treatments. For the descriptive approach, we propose a generalized nonparametric KOB decomposition that considers multiple explanatory variables sequentially. For the prescriptive approach, we introduce a variety of stylized interventions, such as lottery-type and affirmative-action-type interventions that close between-group gaps in treatment. We illustrate the two approaches to disparity analysis through an application to the Black-White gap in college completion rates in the U.S.

Keywords: causal inference, college completion gap, debiased machine learning, disparity analysis, interventional disparities, Kitagawa-Oaxaca-Blinder (KOB) decomposition

1 Introduction

Youth accident deaths: what would we intervene on?

Danish-origin adolescents die in accidents more often than immigrant-origin peers (IRR 1.7 vs first-generation)

Alcohol is a plausible pathway: 16% of fatal accidents; Danish-origin has 4x the odds of an alcohol-related death

But immigrant-origin families are poorer, so adjusting for income *widens* the gap (IRR 1.7 → 2.6)

What if Danish-origin youth drank like their immigrant-origin peers?

bmjmedicine

Check for updates

All cause and cause specific mortality in 15-24-year-olds in Denmark 2010 to 2022: nationwide study of socioeconomic predictors

Sofie Kruckow, Janne S Tolstrup

► Additional supplemental material is published online only. To view, please visit the journal online (https://doi.org/10.1136/bmjmed-2023-000685).

National Institute of Public Health, University of Southern Denmark, Copenhagen, Denmark

ABSTRACT

OBJECTIVE To assess inequalities in all cause and cause specific mortality in young people and if there are differences across gender and age groups.

DESIGN Nationwide cohort study of socioeconomic predictors.

SETTING Denmark, 1 January 2010 to 31 December 2022

association showing a higher mortality with lower levels of socioeconomic position. For instance, incidence rate ratios of all cause mortality related to parents' education was 2.3 (95% CI 2.0 to 2.7) for elementary level, 1.5 (1.3 to 1.6) for low, and 1.3 (1.1 to 1.4) for medium level as compared with high level. For deaths, incidence rate ratios of elementary education level compared with the most

Alcohol-related mortality in 15–24-year-olds in Denmark (2010–2019): a nationwide exploratory study of circumstances and socioeconomic predictors

Caroline Holt Udesen, Signe Skovgaard Hviid, Ulrik Becker, and Janne S. Tolstrup*

National Institute of Public Health, University of Southern Denmark, Studiestraede 6, DK-1455 Copenhagen, Denmark

Summary

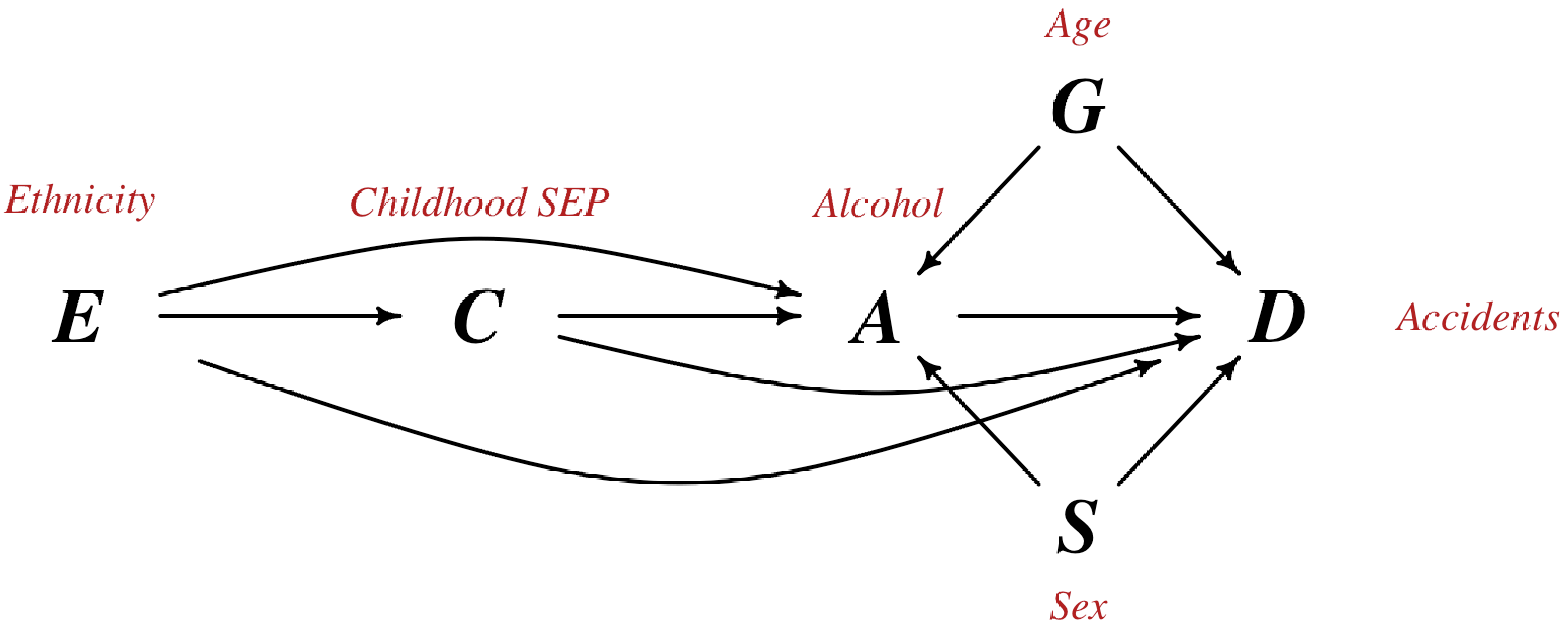
Background Adolescents and young adults aged 15–24 years are disproportionately affected by unnatural deaths, including accidents, suicide and interpersonal violence for which alcohol is a leading risk factor. We aimed to explore the extent of and circumstances surrounding alcohol-related deaths in young people aged 15–24 years and whether socioeconomic background and ethnicity differ in those who died due to alcohol-related causes as compared to the background population.

The Lancet Regional Health - Europe 2023;29: 100620

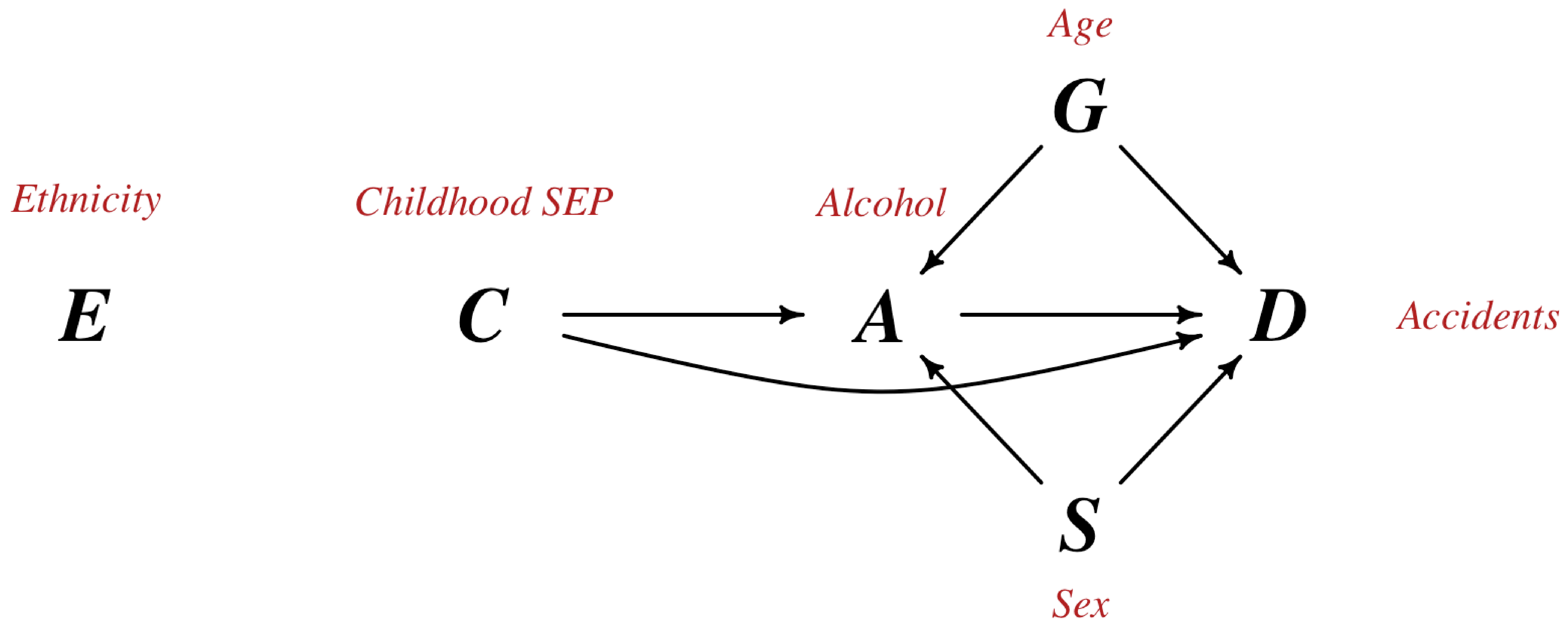
Published Online 29 March 2023

https://doi.org/10.1016/j.lanepe.2023.100620

Conceptualizing the DAG (incomplete)



Different from causal mediation (still valid)



Steps for pursuing interventional decomposition

1 Define the gap and intervention

Consider feasibility, implementation

2 Draw the DAG

Which covariates to adjust for (allowability)

3 Defend the assumptions

Exchangeability; positivity; consistency

4 Estimate the model

Outcome model, weighting, doubly robust

5 Compare factual and counterfactual

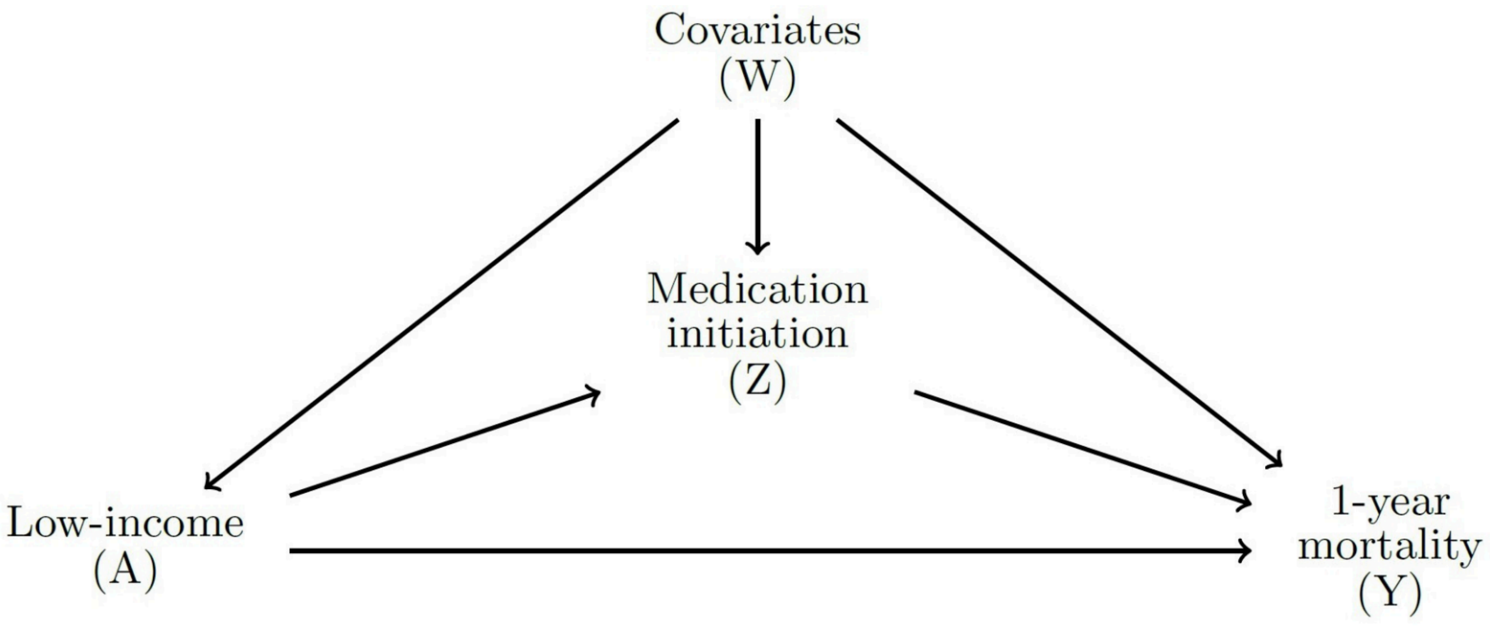
Consider effect scale, uncertainty

Recent example

Theory and methods

Estimating effects of targeted public health interventions using the interventional disparity indirect effect among the exposed

Amalie Lykkemark Møller,[1,2,3] Kathrine Kold Sørensen,[4] Nerissa Nance,[5,6] Julie Thietje Mortensen,[5] Thomas Alexander Gerds,[2] Christian Torp-Pedersen,[2,4] Helene Charlotte Wiese Rytgaard[2]



> we estimate the expected 1-year mortality among the low-income patients in a hypothetical world where they initiated medication as often as high-income patients…

Moller results

Parameter	1-year mortality with/without intervention	Risk difference (95%CI)
IDIE	without: 6.6% with: 6.2%	-0.4% (-0.6%;-0.1%)
IDIE-exposed	without: 10.3% with: 9.3%	-1.0% (-1.7%;-0.2%)

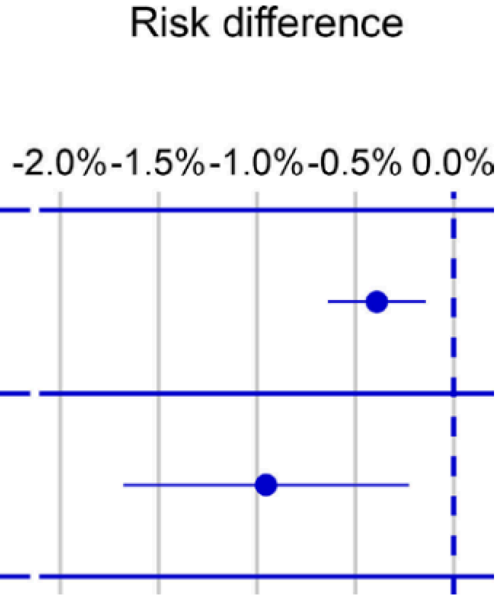


Figure 3 Comparison of the interventional disparity indirect effect (IDIE) and the interventional disparity indirect effect among the exposed (IDIE-exposed) in the study of medication initiation among heart failure patients. We studied a potential barrier-removing intervention under which low-income heart failure patients become as likely as similar high-income patients to initiate treatment. Patients with missing information on the educational level were excluded (n=50). IDIE, interventional disparity indirect effect; IDIE-exposed, interventional disparity indirect effect among the exposed.

IDIE-exposed is the expected reduction in 1-year mortality among low-income heart failure patients in a hypothetical world where they initiated medication with the same probability as observed among similar high-income patients, rather than their observed probability.

Summary: Interventional decomposition

When to use

Asking what would happen to a gap under a hypothetical intervention on a mediator (e.g., drinking)

Extensions

Gap-closing estimand; weighting framework; effects among the disadvantaged; nonparametric estimation

Caveats

Needs causal assumptions (no unmeasured confounding, none affected by the exposure) and a well-defined intervention; what counts as 'explained' is a normative choice

Summary

Various decomposition techniques exist that may be useful for analyzing social determinants of health:

- Regression-based decomposition of Concentration Index
- Oaxaca decomposition of mean health between groups

All of these techniques make assumptions that need to be evaluated in the course of analysis.

When used properly, decomposition techniques can help to provide key evidence on why health inequalities

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Backup

Backup: Kitagawa's three-component version

$$\underbrace{\text{Rate}_A - \text{Rate}_B}_{\text{crude difference}} = \overbrace{\sum_i R_{Bi}(P_{Ai} - P_{Bi})}^{\text{composition}} + \overbrace{\sum_i P_{Bi}(R_{Ai} - R_{Bi})}^{\text{rate}} + \overbrace{\sum_i (R_{Ai} - R_{Bi})(P_{Ai} - P_{Bi})}^{\text{interaction}}$$

Composition

Group	R_{Bi}	$P_{Ai} - P_{Bi}$	Contrib.
Young	20	−0.20	−4.0
Old	30	+0.20	+6.0
Sum			**+2.0**

Rate

Group	P_{Bi}	$R_{Ai} - R_{Bi}$	Contrib.
Young	0.50	−10	−5.0
Old	0.50	+10	+5.0
Sum			**0.0**

Interaction

Group	$R_{Ai} - R_{Bi}$	$P_{Ai} - P_{Bi}$	Contrib.
Young	−10	−0.20	+2.0
Old	+10	+0.20	+2.0
Sum			**+4.0**

Total = +2.0 + 0.0 + 4.0 = **+6.0** = crude gap (31 – 25)

Add the interaction to composition: +6.0 and 0.0 (A's rates). Add it to rate: +2.0 and +4.0 (B's rates).

Overall contributions are *net*