The consequences of consequentialism

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DEPARTMENT OF EPIDEMIOLOGY

Columbia University
MAILMAN SCHOOL
OF PUBLIC HEALTH

1. Motivations
2. A disciplinary definition
3. A call for recalibration
4. The consequences of consequentialism
5. Controversial consequential thoughts
6. Other consequences, not discussed

1. **Motivations**
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a. Population health; we can do better

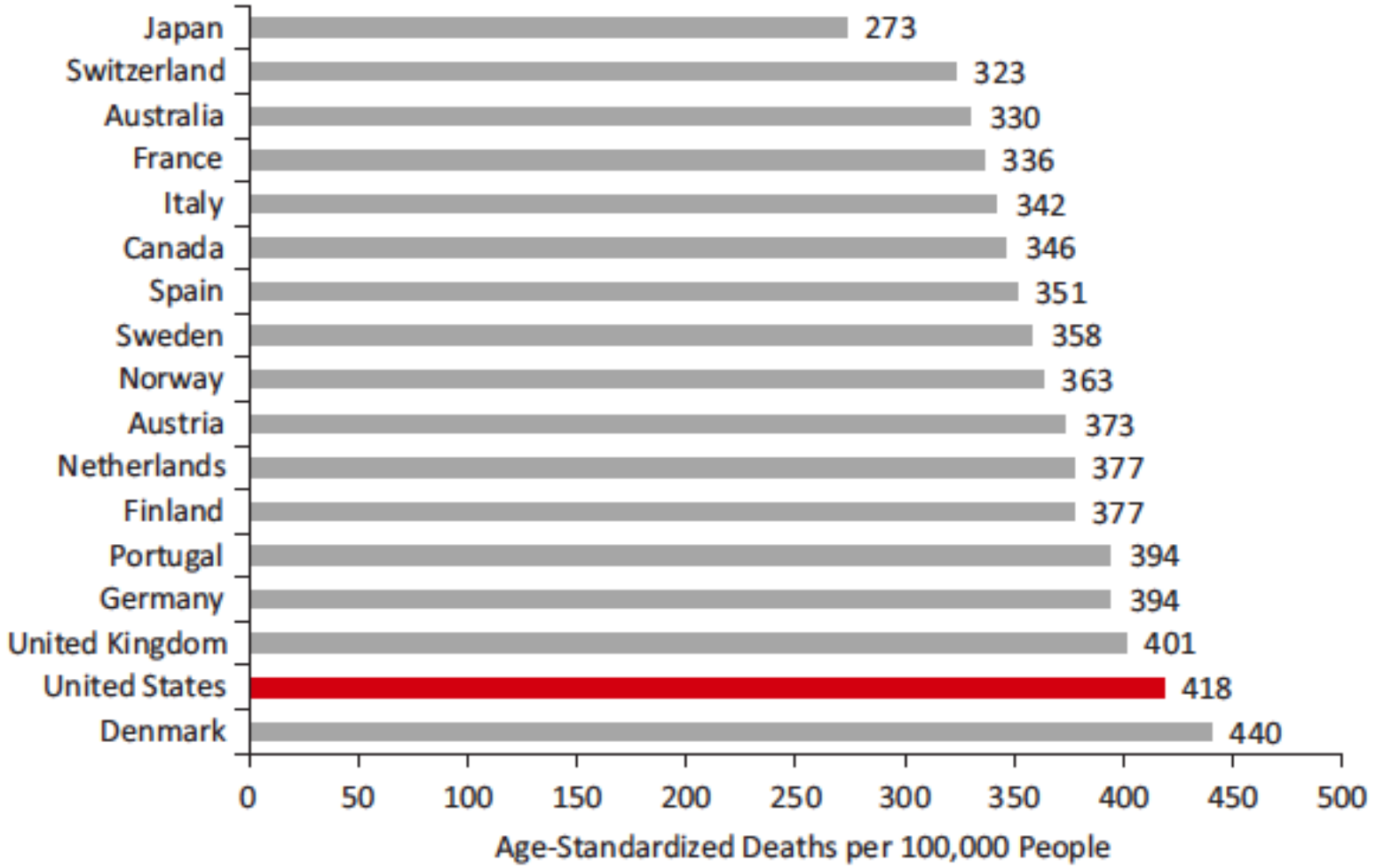


FIGURE 1-1 Mortality from noncommunicable diseases in 17 peer countries, 2008.
SOURCE: Data from World Health Organization (2011a, Table 3).

US Health in International Perspective. Shorter lives, poorer health. S Woolf, L Aron, eds. NRC and IOM. 2012.

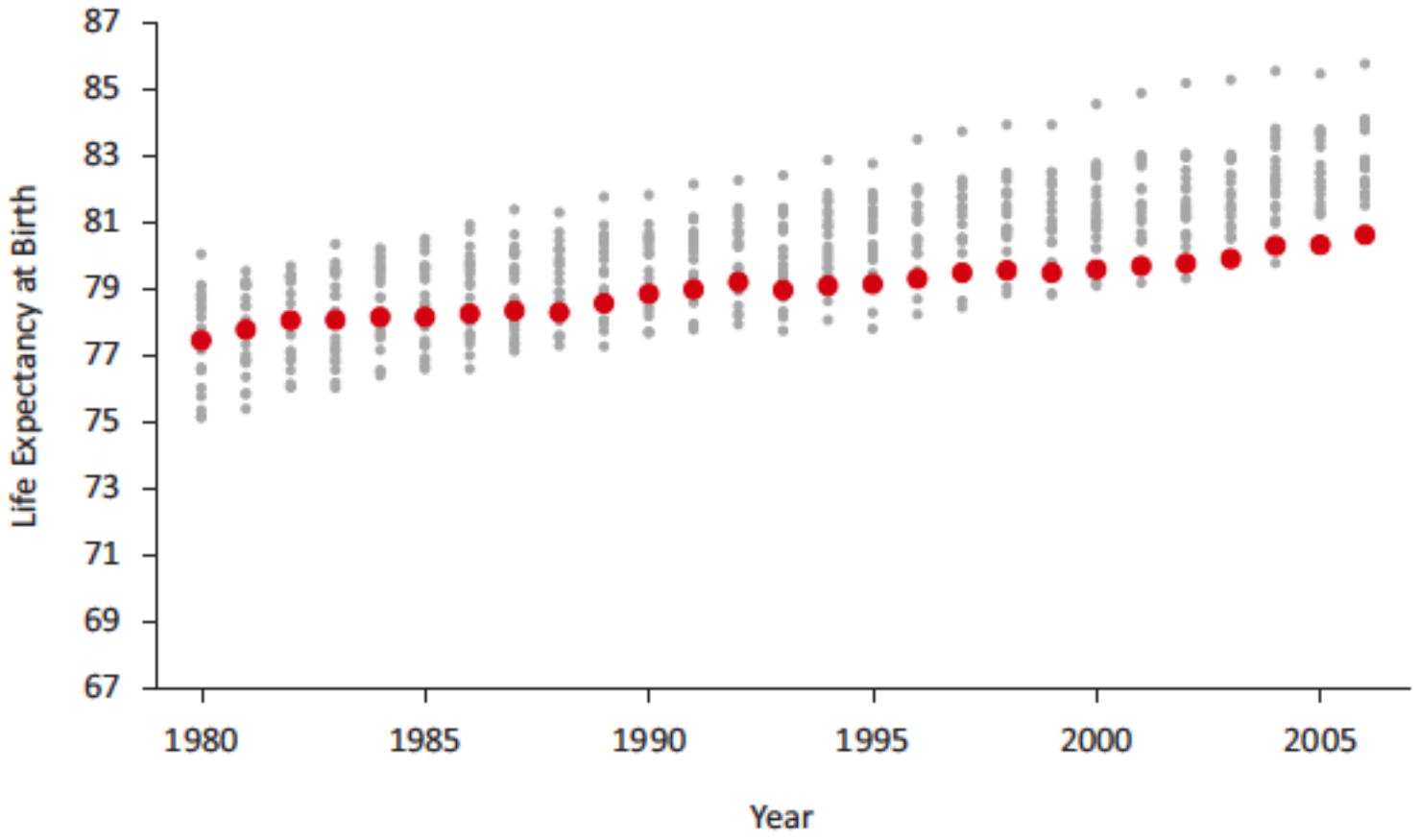


FIGURE 1-6 U.S. female life expectancy at birth relative to 21 other high-income countries, 1980-2006.

NOTES: Red circles depict newborn life expectancy in the United States. Grey circles depict life expectancy values for Australia, Austria, Belgium, Canada, Denmark, Finland, France, Iceland, Ireland, Italy, Japan, Luxembourg, the Netherlands, New Zealand, Norway, Portugal, Spain, Sweden, Switzerland, the United Kingdom, and West Germany.

SOURCE: National Research Council (2011, Figure 1-4).

US Health in International Perspective. Shorter lives, poorer health. S Woolf, L Aron, eds. NRC and IOM. 2012.

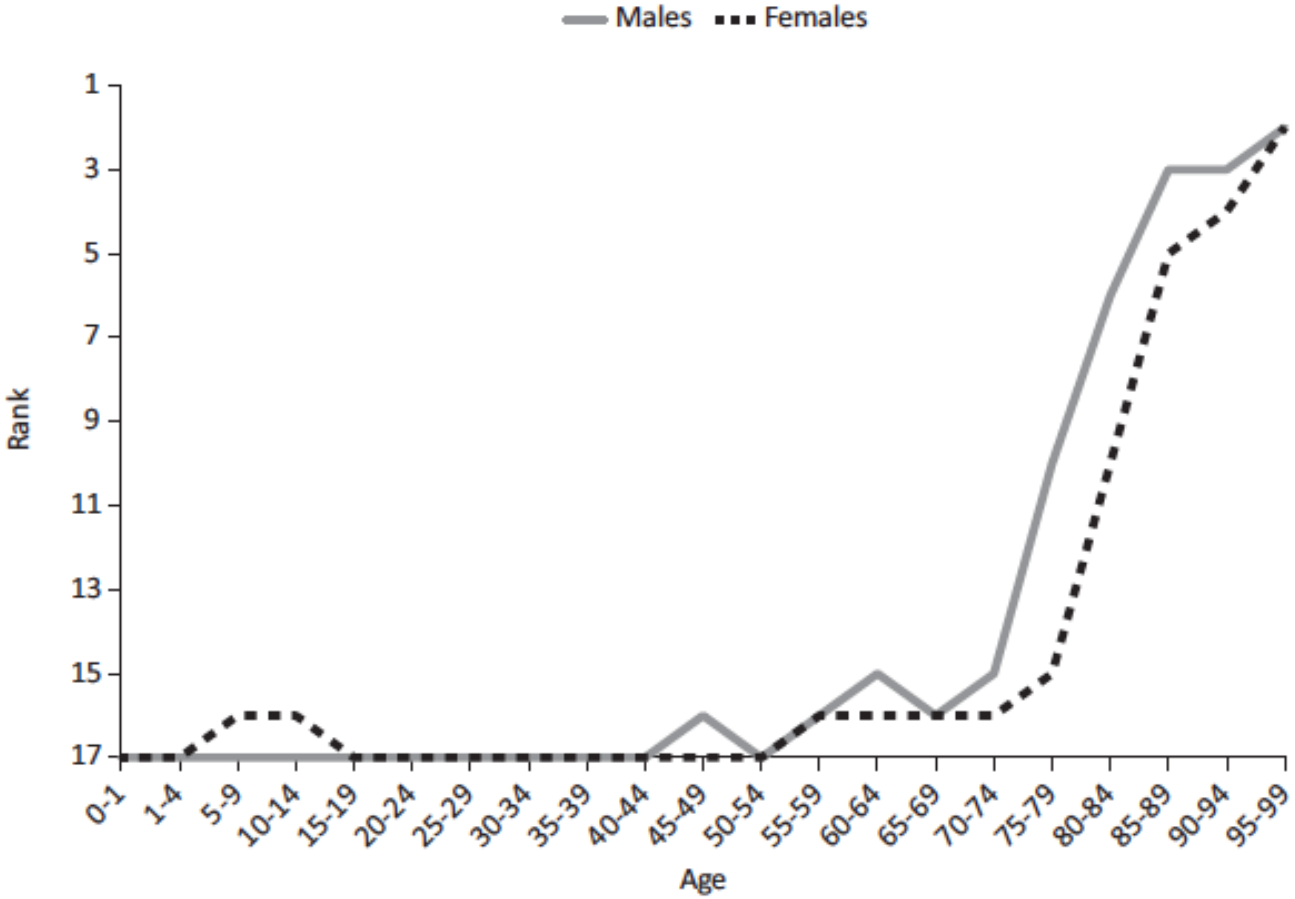
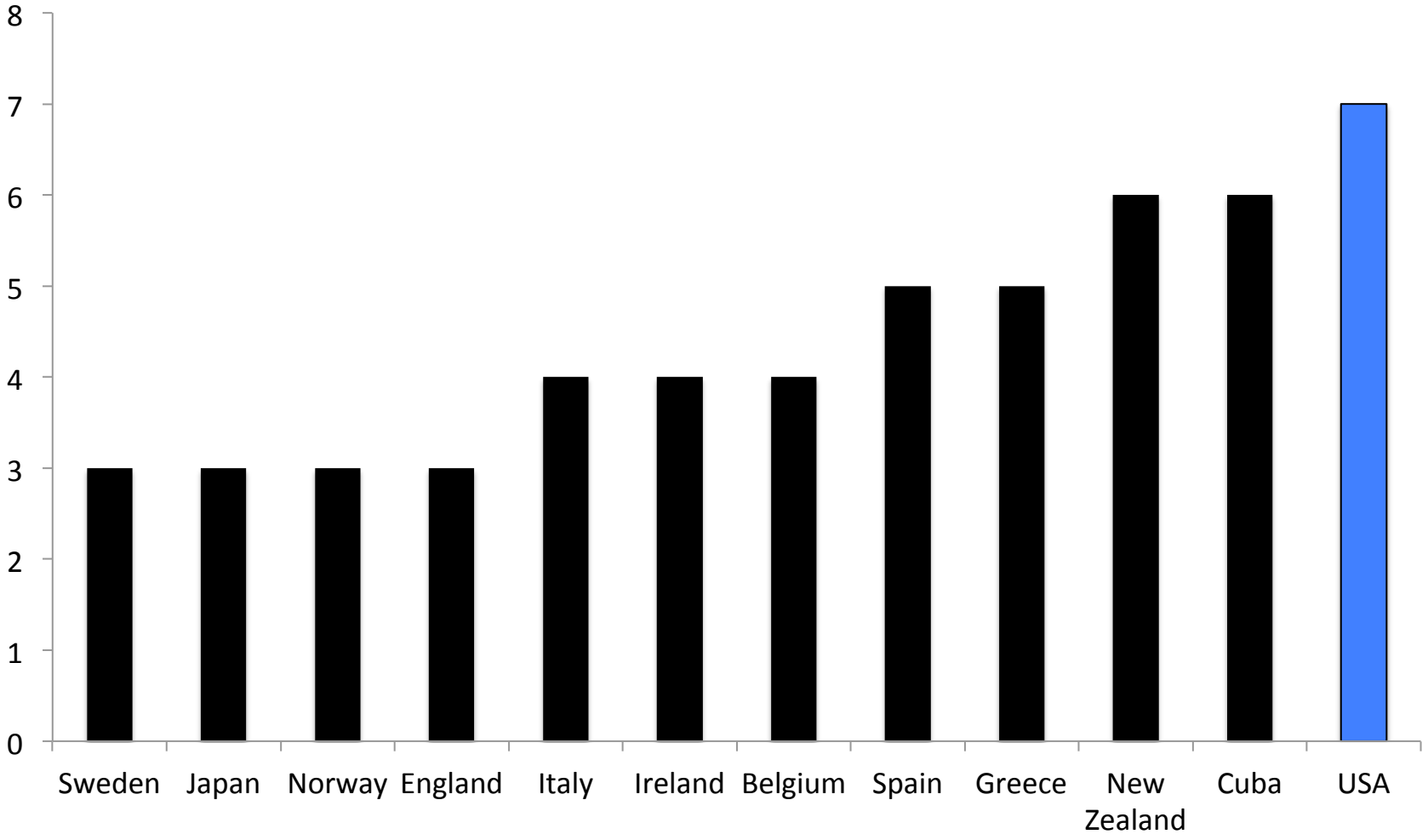


FIGURE 1-9 Ranking of U.S. mortality rates, by age group, among 17 peer countries, 2006-2008.

NOTES: The top rank is number 1, indicating the lowest death rate, and the bottom rank is number 17, indicating the highest death rate. Rankings are based on all-cause mortality rates for 2006-2008. Data for this figure were drawn from (1) the Human Mortality Database, 2011, University of California, Berkeley (USA), and Max Planck Institute for Demographic Research (Germany), available at http://www.mortality.org or http://www.humanmortality.de (data downloaded July 18, 2011) and (2) Arias, Elizabeth, 2011, United States Life Tables, 2007. *National Vital Statistics Reports, 59*(9), Hyattsville, MD: National Center for Health Statistics.

SOURCE: Adapted from Ho and Preston (2011, Figure 1).

Under five mortality rates per 1,000 live births in selected countries



Mortality rate, under-5 (per 1,000 live births). The World Bank Data, 2012. United Nations Inter-agency Group for Child Mortality Estimation (UNICEF, WHO, World Bank, UN DESA Population Division). <http://data.worldbank.org/indicator/SH.DYN.MORT> Accessed October 29, 2013.

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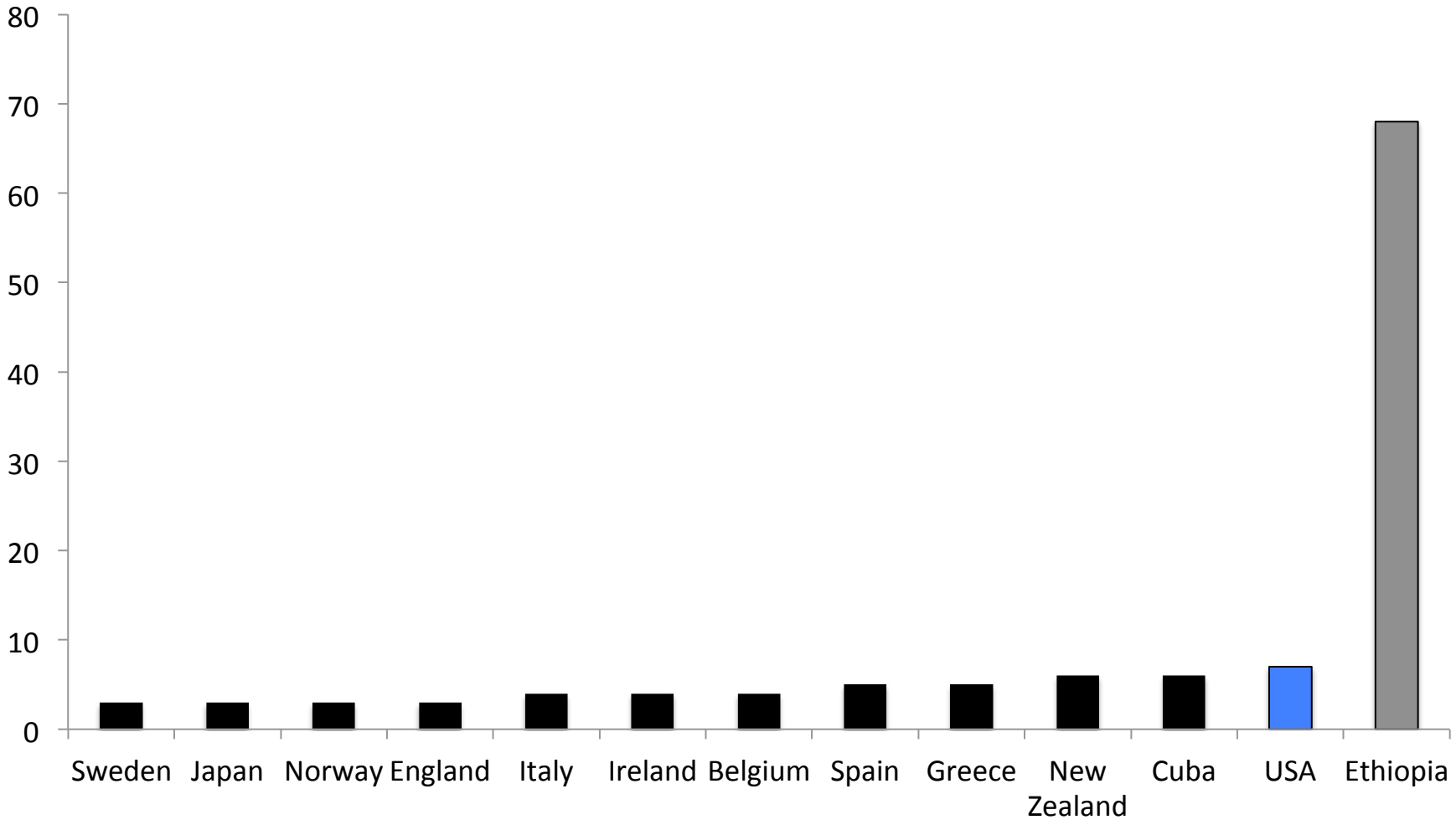

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b. The discipline may not be doing that well

Components of Participating Organizations

Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD), (http://www.nichd.nih.gov)

Title: Predoctoral and Postdoctoral Training Program in Reproductive, Obstetric, Perinatal and Pediatric Epidemiology and Pharmacoepidemiology (T32)

Announcement Type

This is a reissue of PAR-08-271.

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Components of Participating Organizations	*Eunice Kennedy Shriver* National Institute of Child Health and Human Development (NICHD)
Funding Opportunity Title	**Postdoctoral Training Program in Obstetric and Pediatric Pharmacoepidemiology (T32)**
Activity Code	T32 Institutional National Research Service Award (NRSA)
Announcement Type	Reissue of PAR-10-194

Justin Wolfers
@JustinWolfers

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Cold and snow can have strange, often negative effects on people.

Wall Street Journal @WSJ

2:18pm · 16 Feb 14 · web

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> **The investigation of (a) the various external or physical agencies and the different conditions of life which favor their development or influence their character; and (b) the sanitary and hygienic measures best fitted to check, mitigate, or prevent them**

Transactions of the Epidemiological Society of London: Sessions 1866 to 1876: Objects of the Epidemiological Society. London, England: Epidemiological Society of London for the Investigation of Epidemic Diseases, 1876.

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“A consequentialist epidemiology [is] centrally concerned with improving health outcomes. We would be much more concerned with maximizing the good that can be achieved by our studies and by our approaches than we are by our approaches themselves.”

Galea S. An argument for a consequentialist epidemiology. American Journal of Epidemiology. 2013 Oct 15;178(8):1185-91

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An epidemiology of consequence should

a. Focus on what matters most

b. Critically interrogate what we think we know

c. Engage in translating the science

d. Teach epidemiology differently

An epidemiology of consequence should

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a. Focus on what matters most

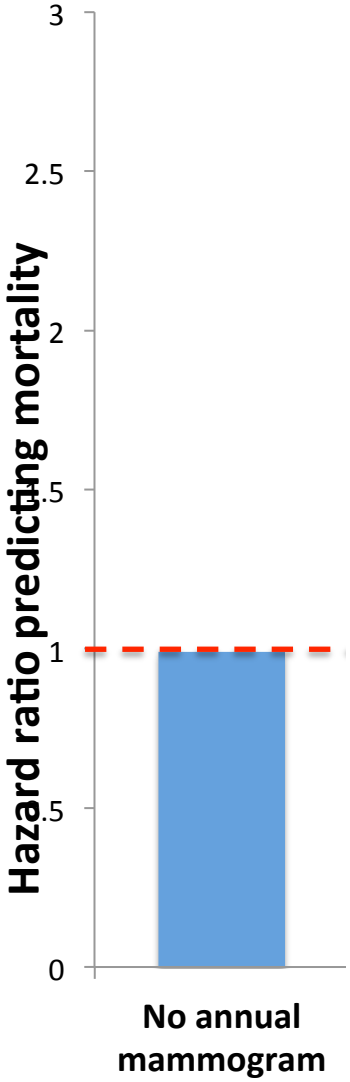
Populations **and improving their health is what matters most**

a. Focus on what matters most

Populations **and improving their health is what matters most. This suggests that we need to**

a1. Identify, and study, the factors that may have greatest impact on population health

‘Accepted’ risk factors and mortality



van Oort F, van Lenthe F, Mackenbach J. Material, psychosocial, and behavioural factors in the explanation of educational inequalities in mortality in the Netherlands. *J Epidemiol Community Health* 2005;59:214-220// Clarke R, Emberson J, Fletcher A, Breeze E, Marmot M, Shipley MJ. Life expectancy in relation to cardiovascular risk factors: 38 year follow-up of 19 000 men in the Whitehall study. *BMJ* 2009;339:b3513// Miller AB, Wall C, Baines CJ, Sun P, Teresa T, Narod SA. Twenty five year follow-up for breast cancer incidence and mortality of the Canadian National Breast Screening Study: randomised screening trial. *BMJ* 2014;348:g366//Chen Y, et al Association between body mass index and cardiovascular disease mortality in east Asians and south Asians: pooled analysis of prospective data from the Asia Cohort Consortium. *BMJ.* 2013 Oct 1;347:f5446.

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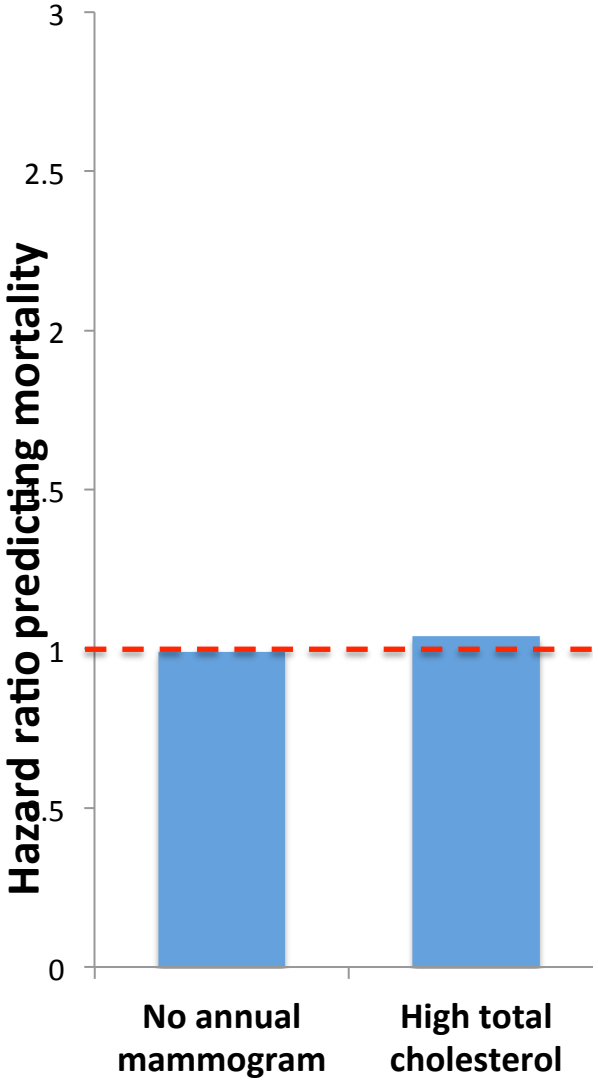

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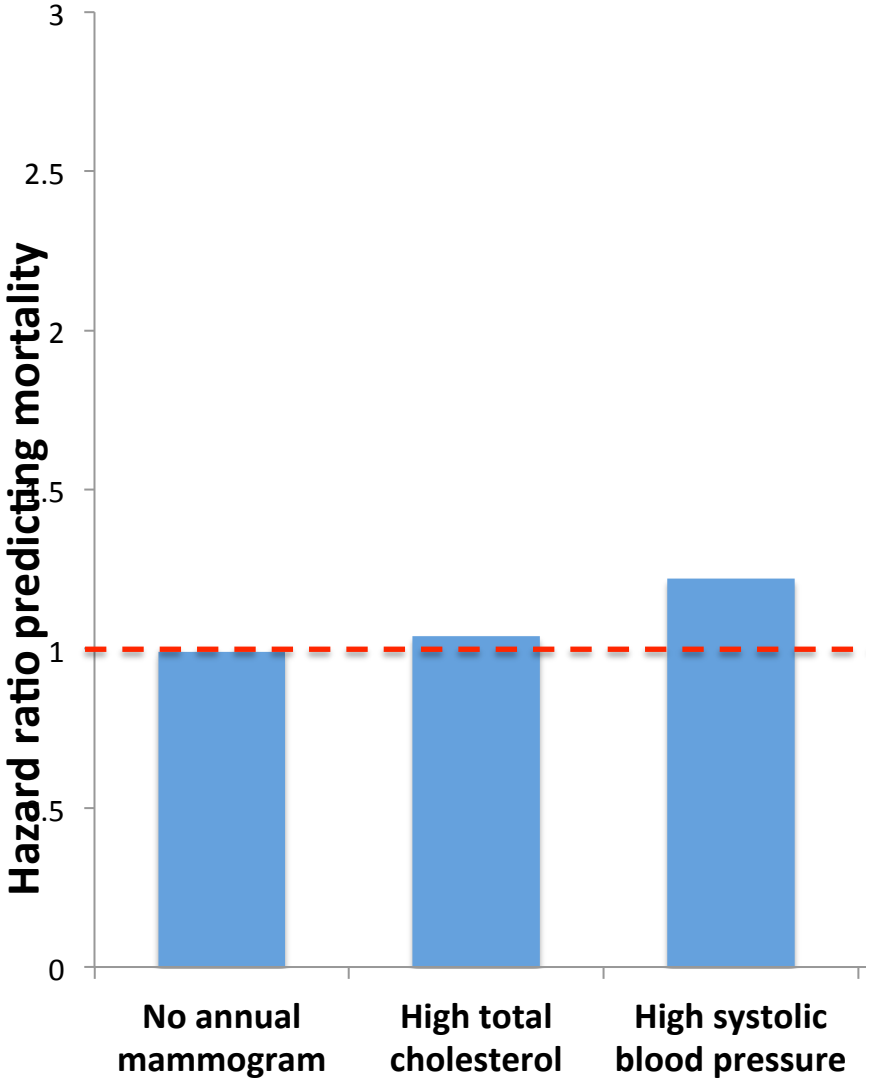

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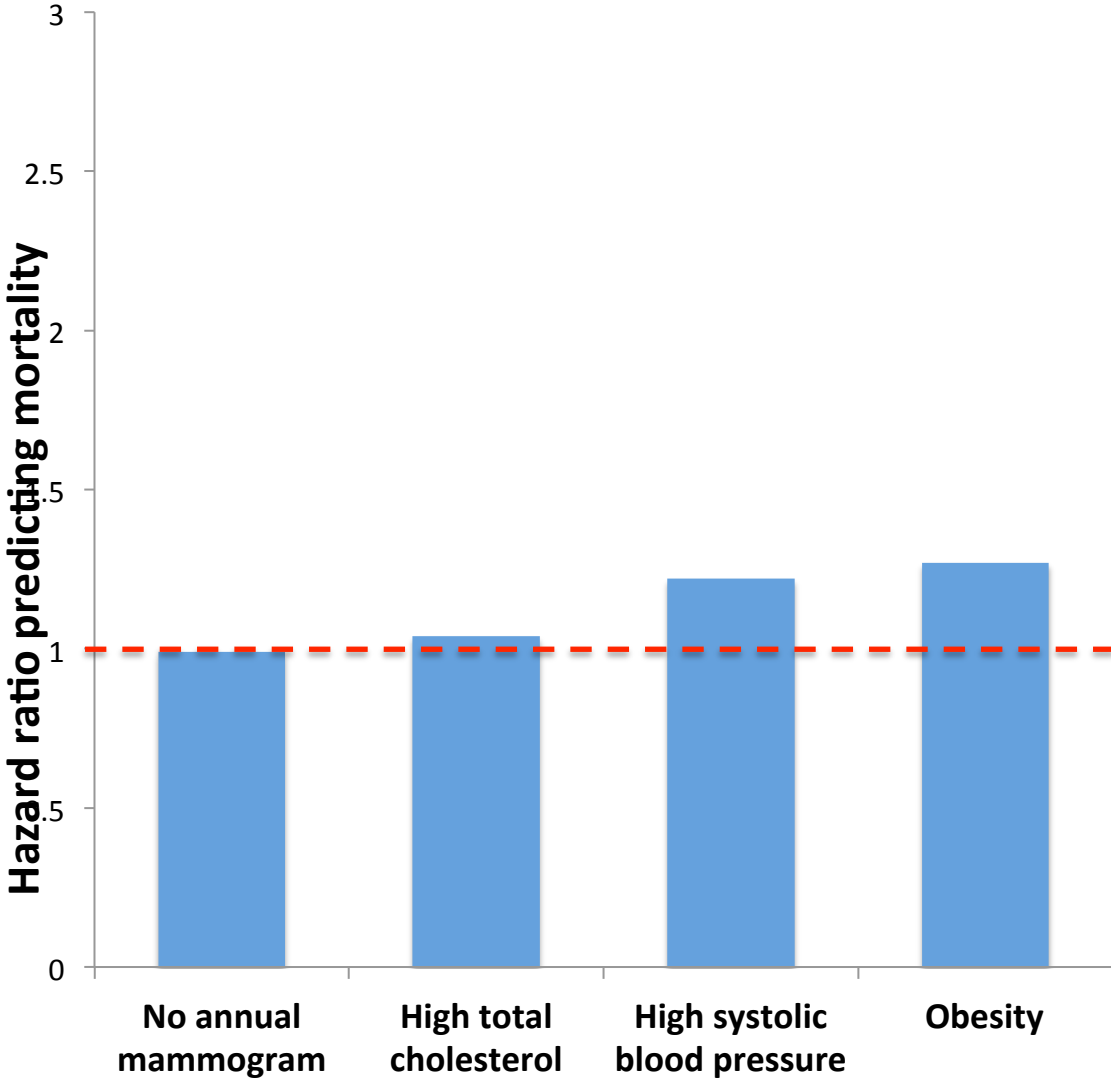

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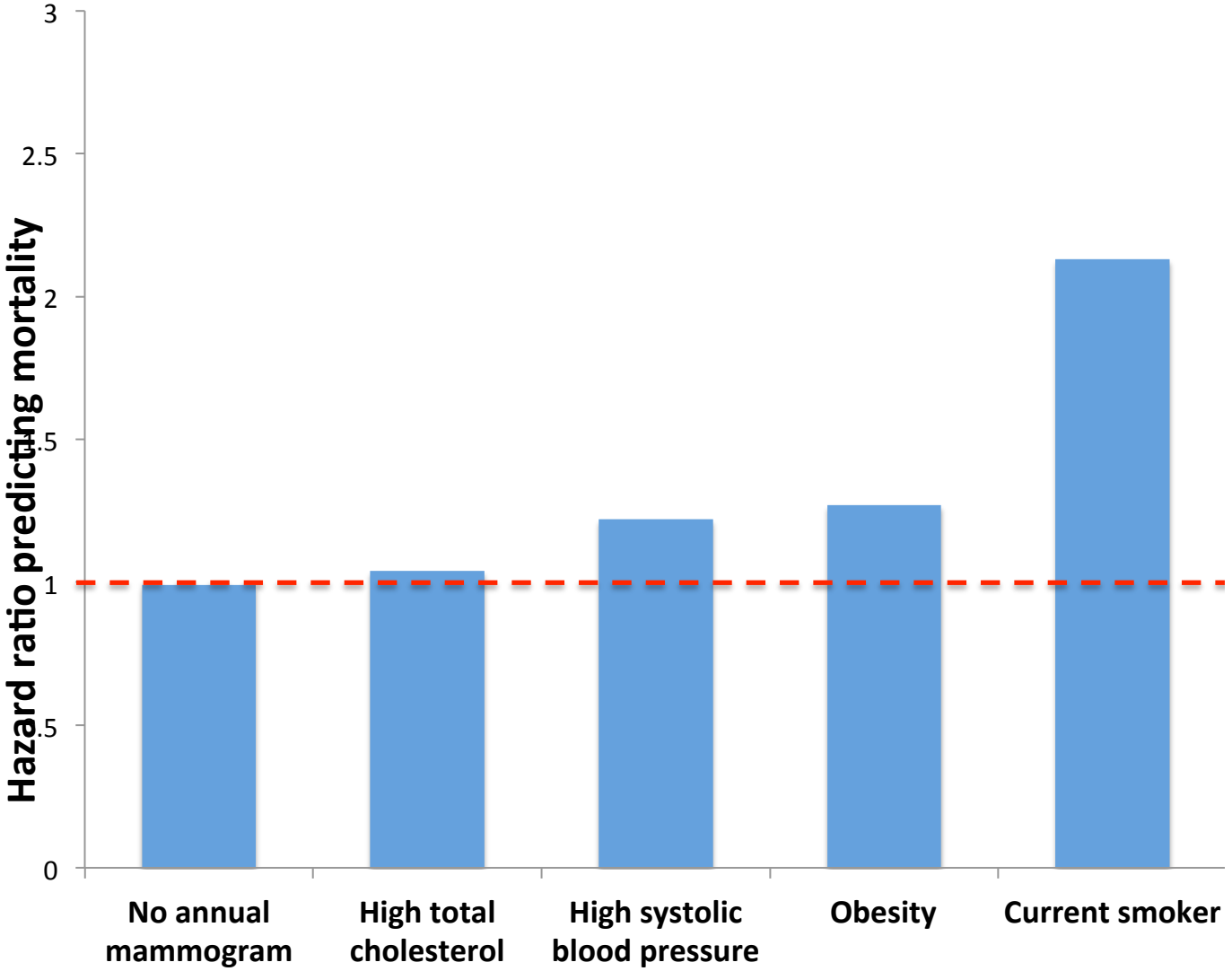

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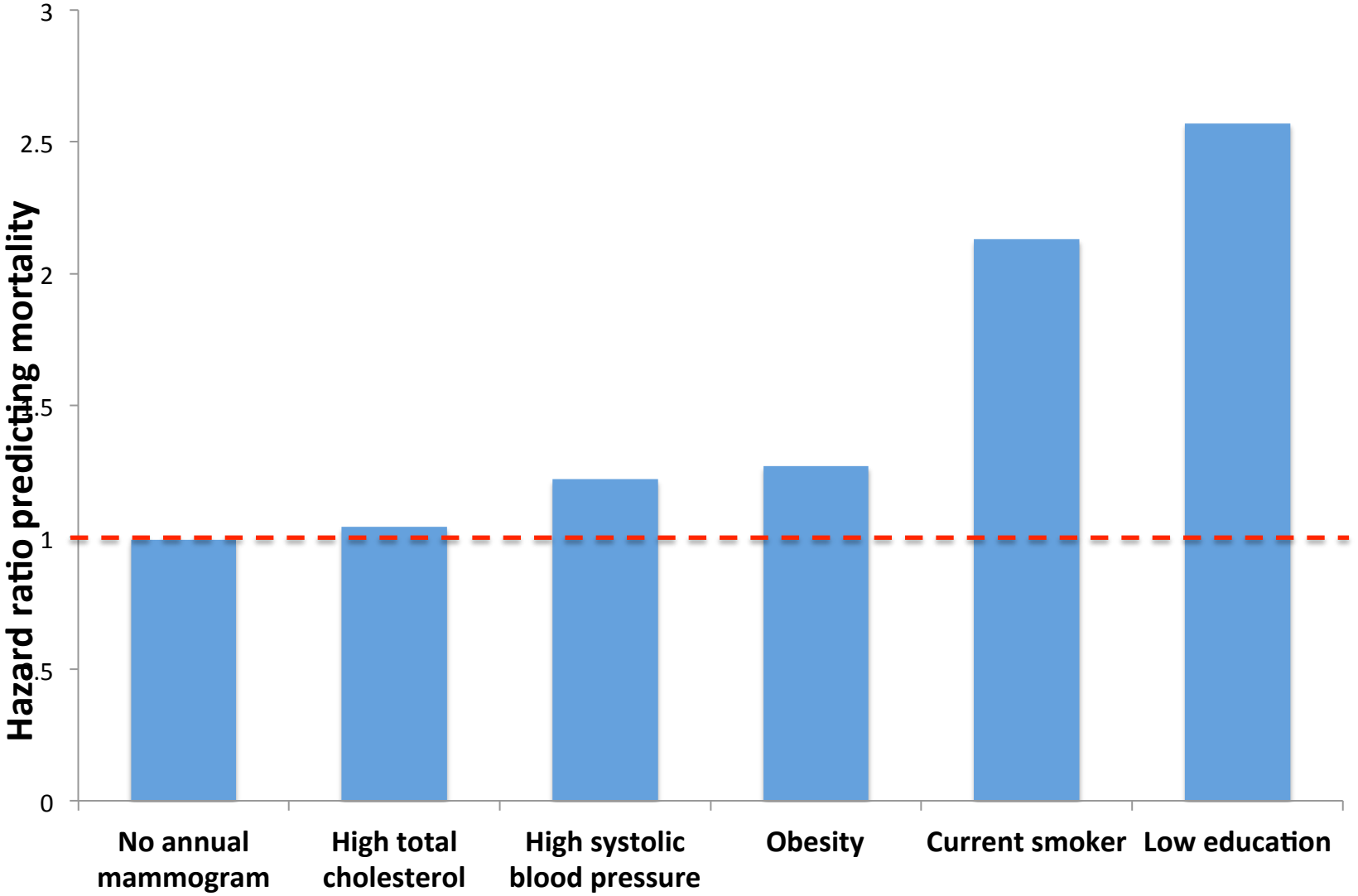

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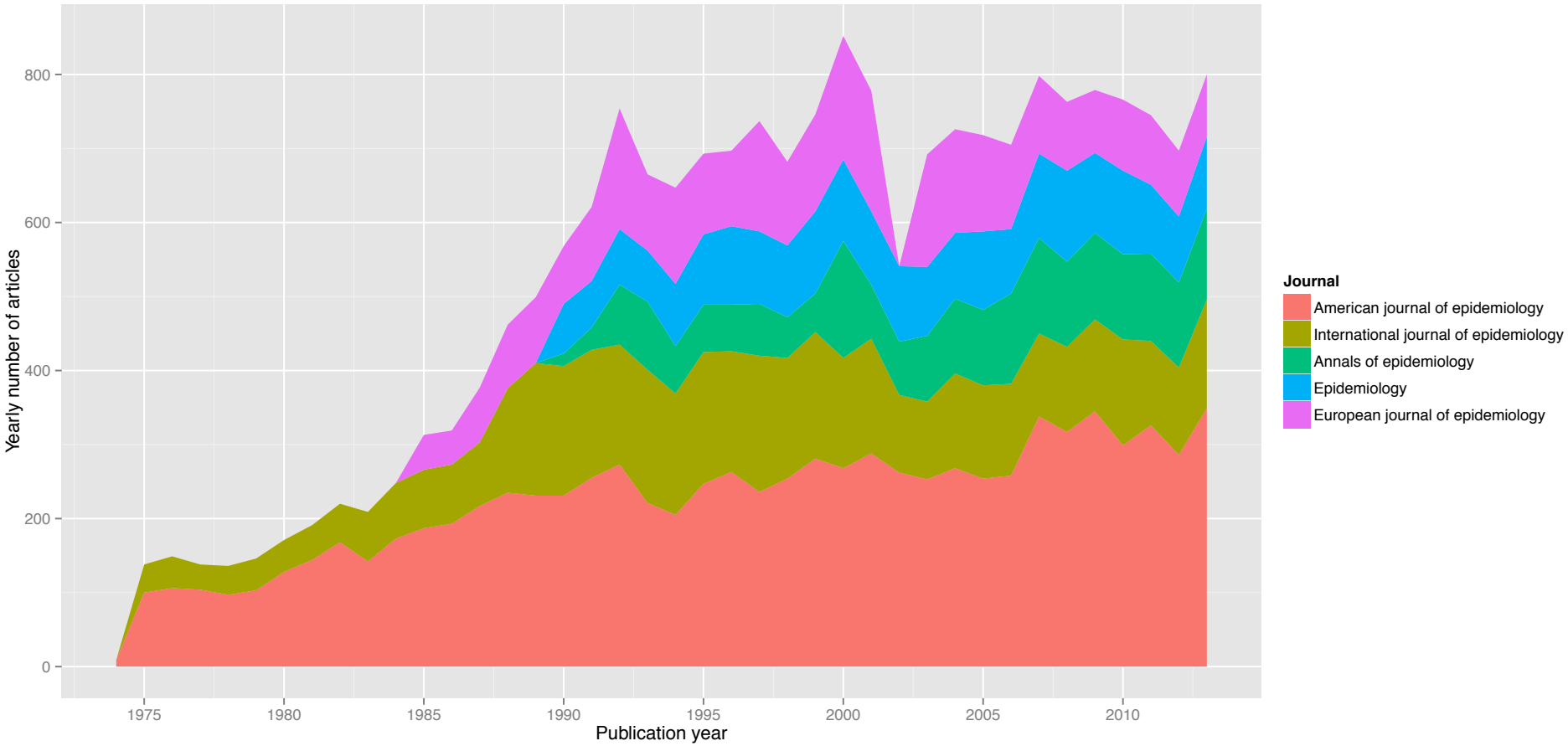
a. Focus on what matters most



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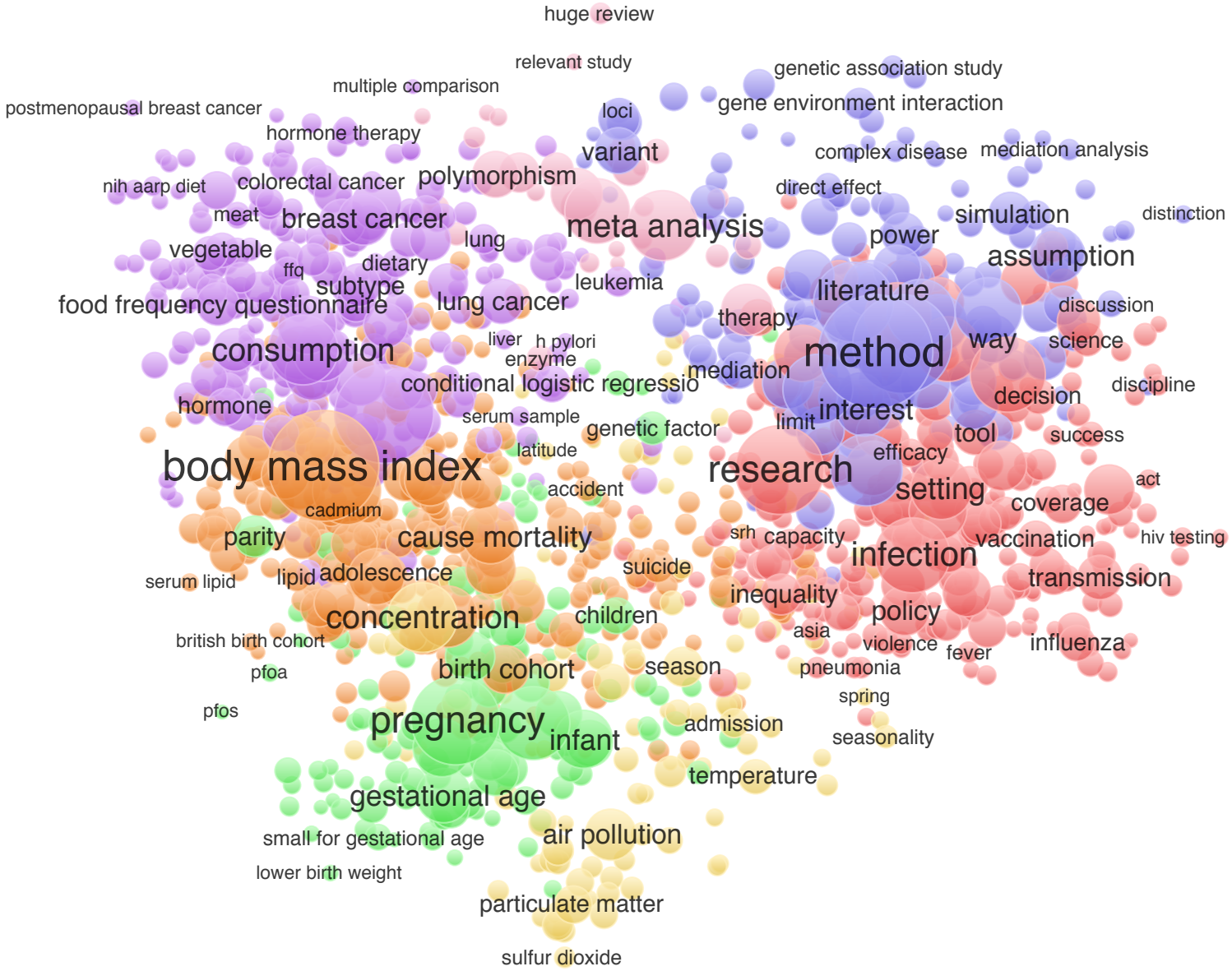

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Global burden of disease

Figure 27: Ten leading causes of burden of disease, world, 2004 and 2030

2004 Disease or injury	As % of total DALYs	Rank	Rank	As % of total DALYs	2030 Disease or injury
Lower respiratory infections	6.2	1	1	6.2	Unipolar depressive disorders
Diarrhoeal diseases	4.8	2	2	5.5	Ischaemic heart disease
Unipolar depressive disorders	4.3	3	3	4.9	Road traffic accidents
Ischaemic heart disease	4.1	4	4	4.3	Cerebrovascular disease
HIV/AIDS	3.8	5	5	3.8	COPD
Cerebrovascular disease	3.1	6	6	3.2	Lower respiratory infections
Prematurity and low birth weight	2.9	7	7	2.9	Hearing loss, adult onset
Birth asphyxia and birth trauma	2.7	8	8	2.7	Refractive errors
Road traffic accidents	2.7	9	9	2.5	HIV/AIDS
Neonatal infections and other[a]	2.7	10	10	2.3	Diabetes mellitus
COPD	2.0	13	11	1.9	Neonatal infections and other[a]
Refractive errors	1.8	14	12	1.9	Prematurity and low birth weight
Hearing loss, adult onset	1.8	15	15	1.9	Birth asphyxia and birth trauma
Diabetes mellitus	1.3	19	18	1.6	Diarrhoeal diseases

World Health Organization. *Global Burden of Disease, 2004 update: Burden of disease, DALYs*. 2008

a. Focus on what matters most

Populations **and improving their health is what matters most. This suggests that we need to**

a1. Identify, and study, the factors that may have greatest impact on population health

a2. Clarify the relative contribution of factors that influence health conditions in populations

a. Focus on what matters most

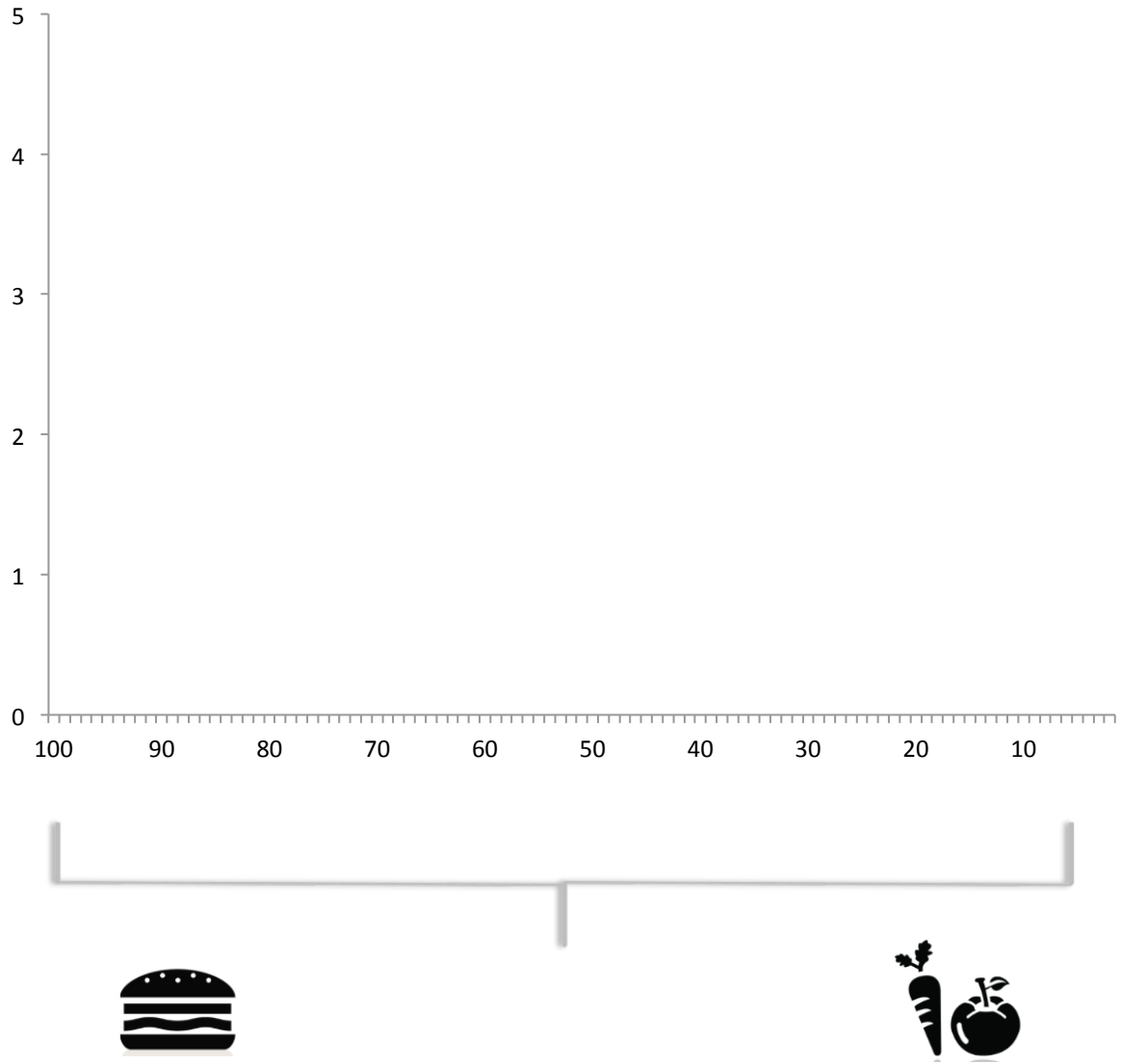
Will reducing the prevalence of junk food eating in the population reduce obesity in the population?

Baseline heritability

Food environment

Junk food eating

Keyes KM, Davey Smith J, Koenen KC, Galea S. The mathematical limits of genetic prediction: implications for personalized medicine. Under review.
Keyes KM, Galea S. Why a focus on changing health behaviors to reduce obesity prevalence will not work: the mathematical limits of individual risk behaviors. Under review.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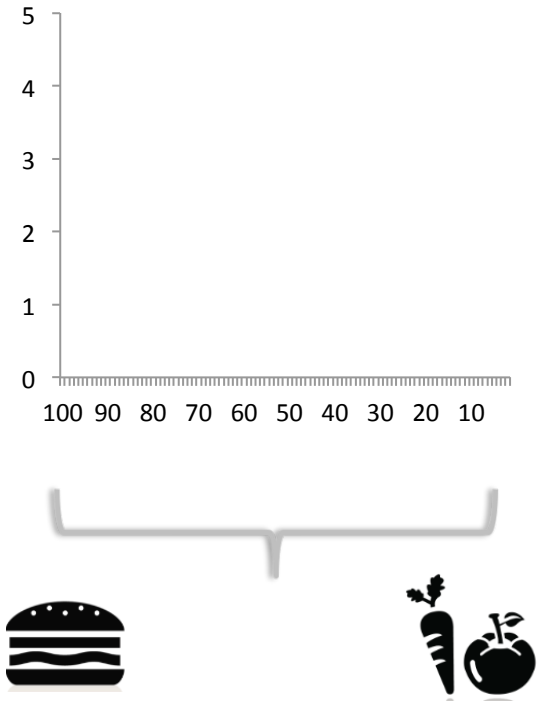
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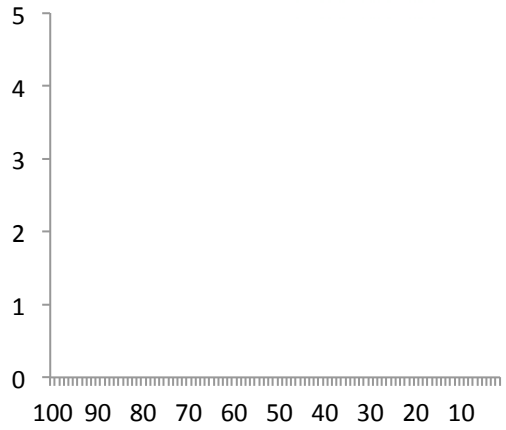
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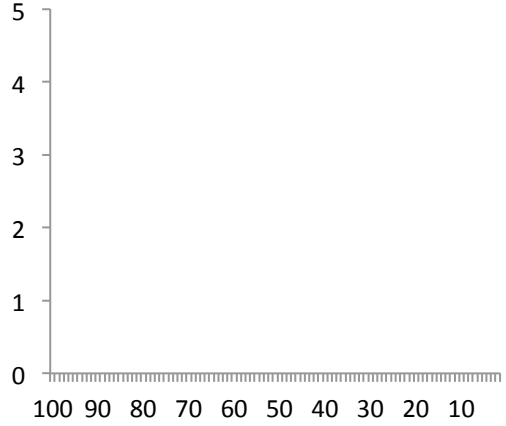
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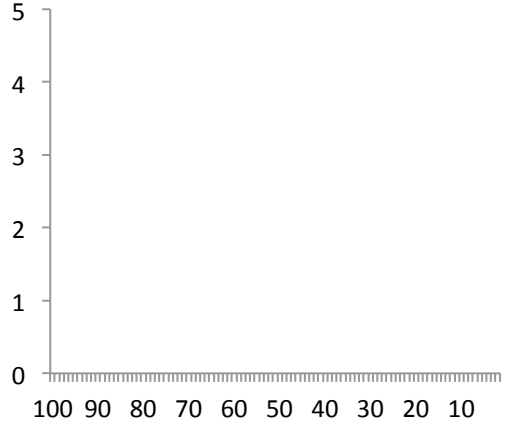
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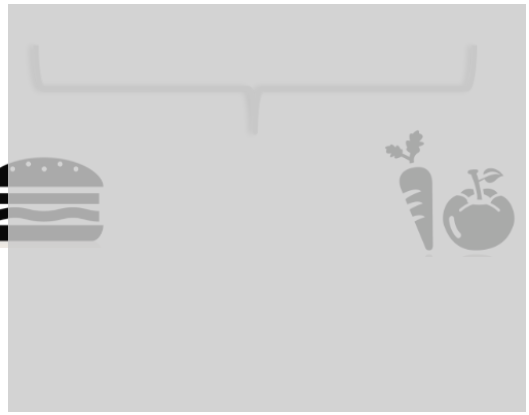

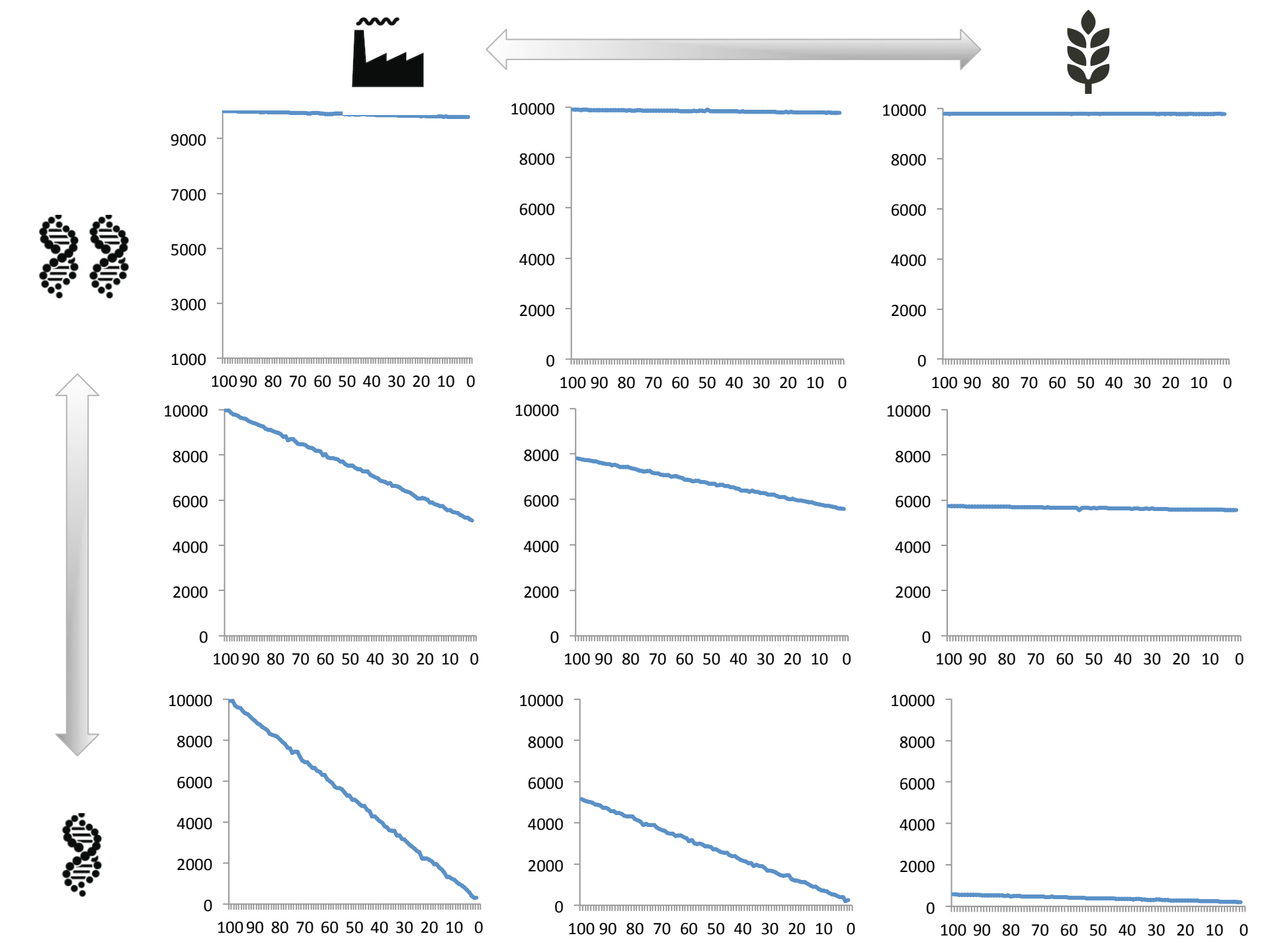
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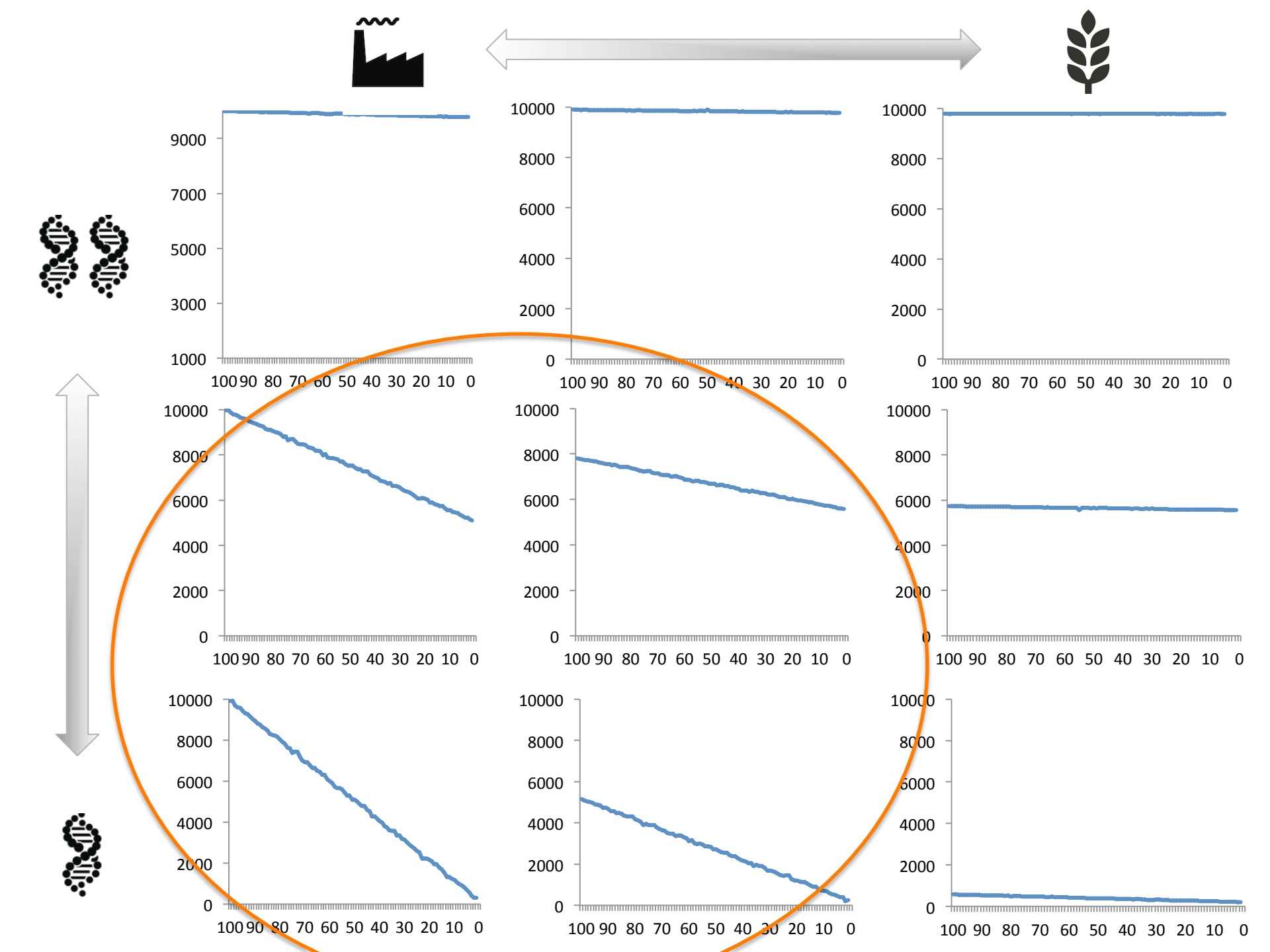
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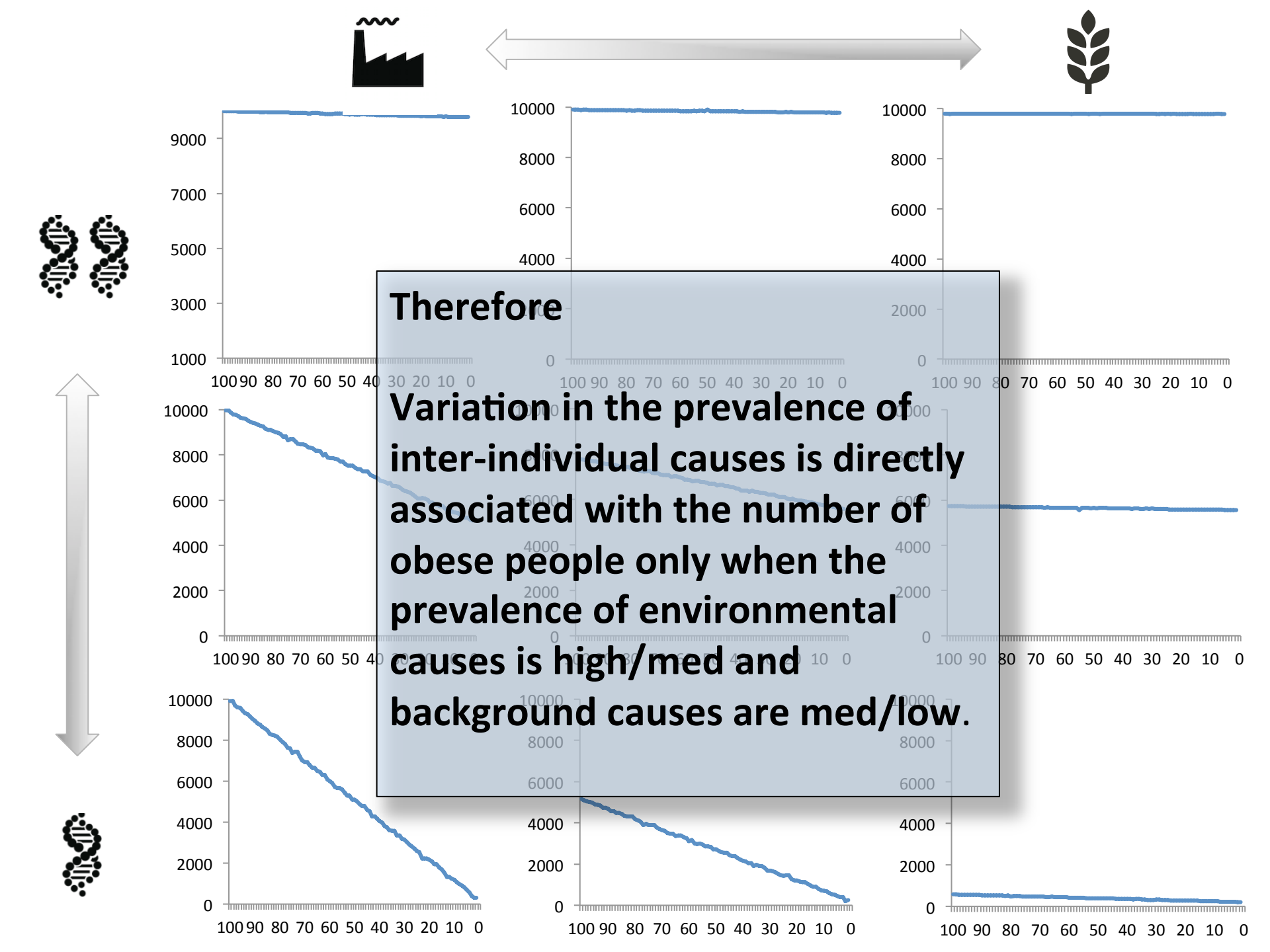
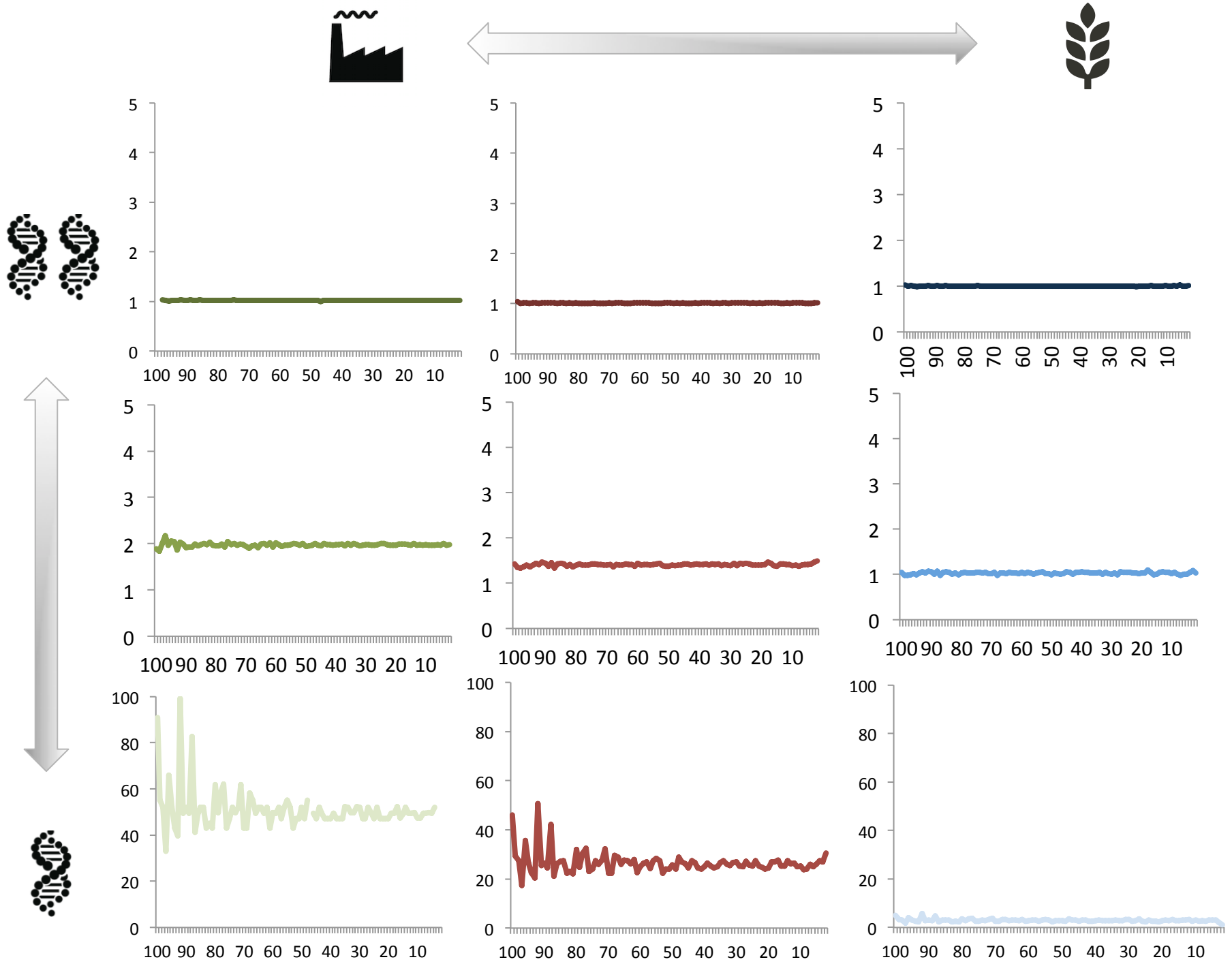

Therefore
Variation in the prevalence of inter-individual causes is directly associated with the number of obese people only when the prevalence of environmental causes is high/med and background causes are med/low.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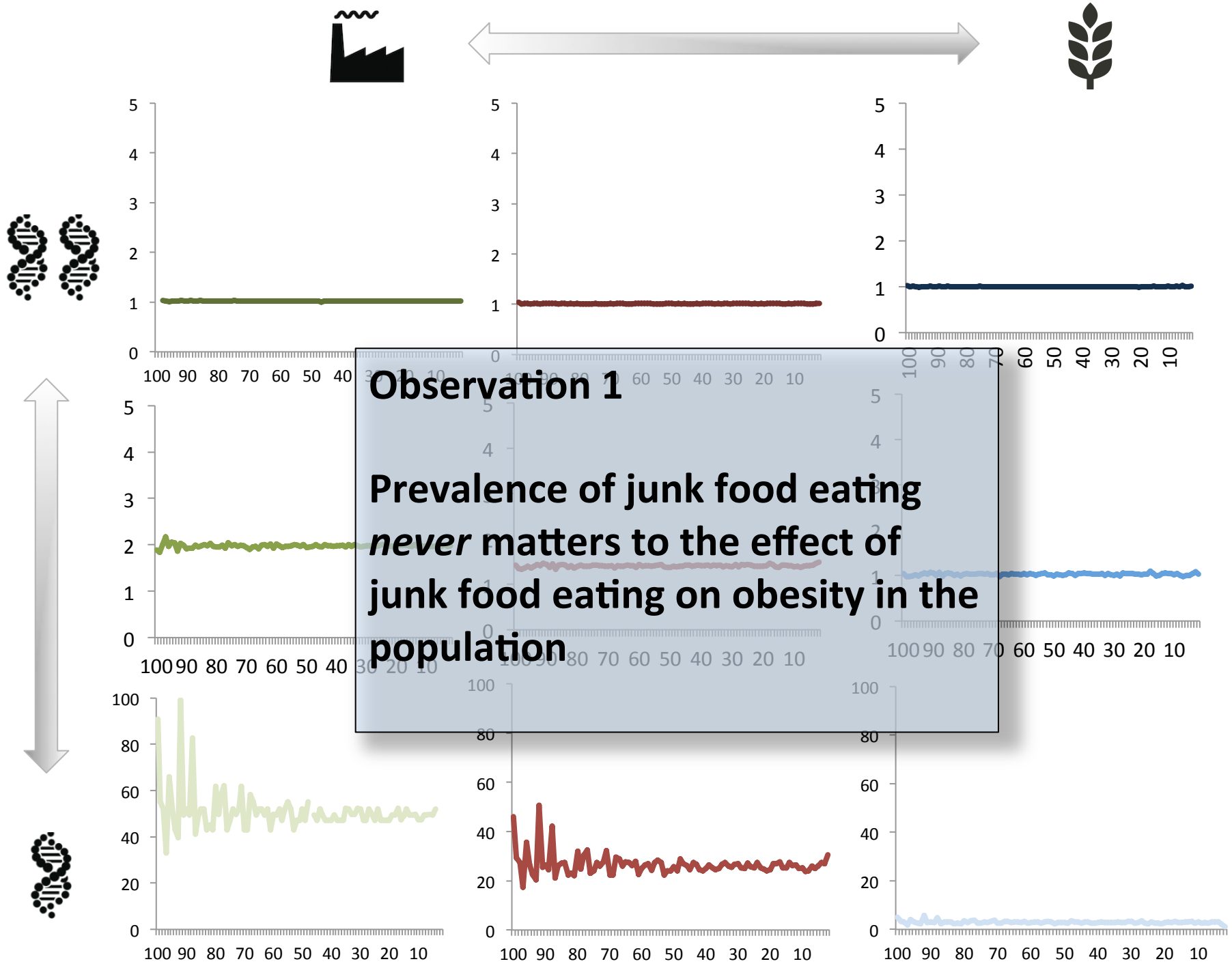
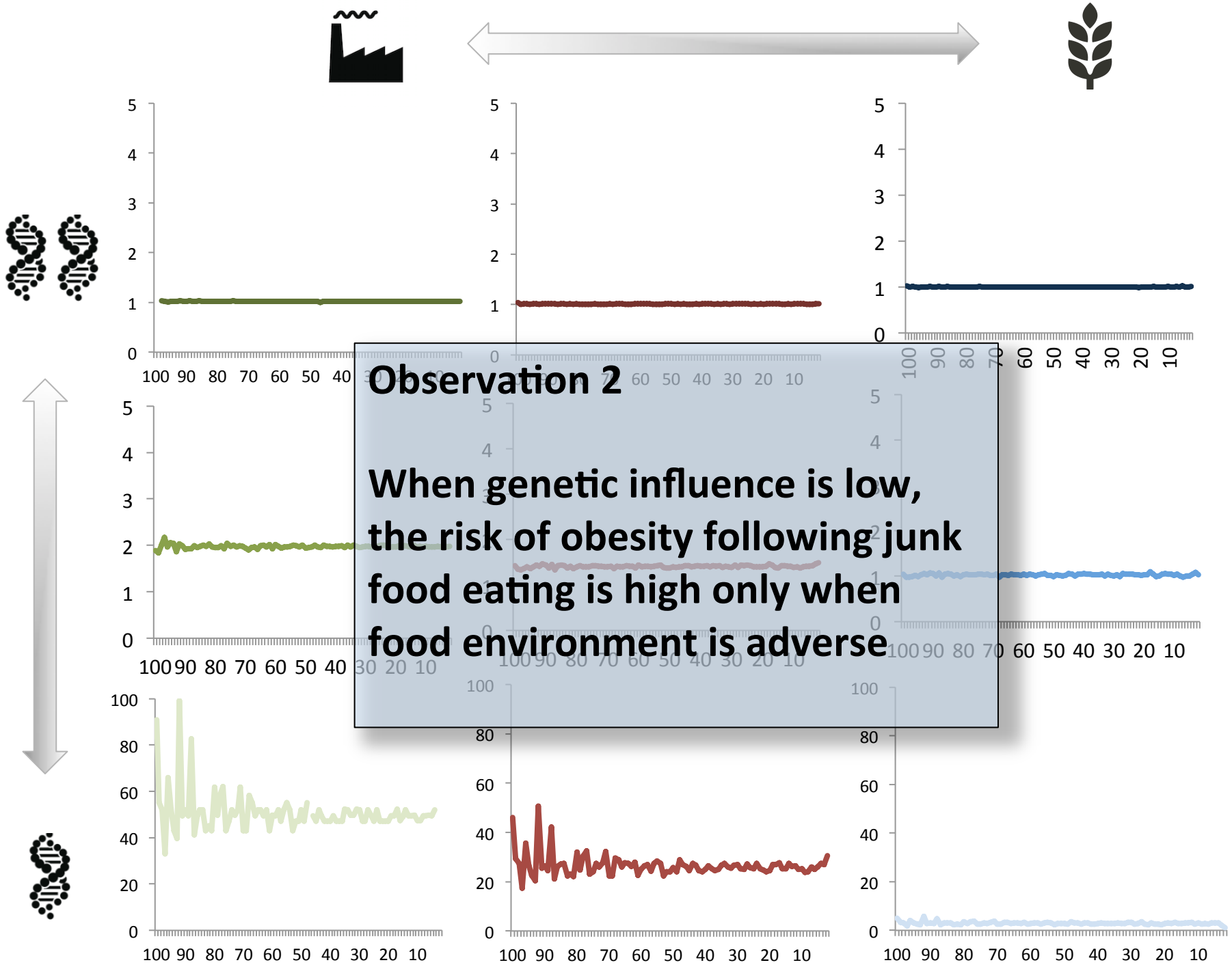
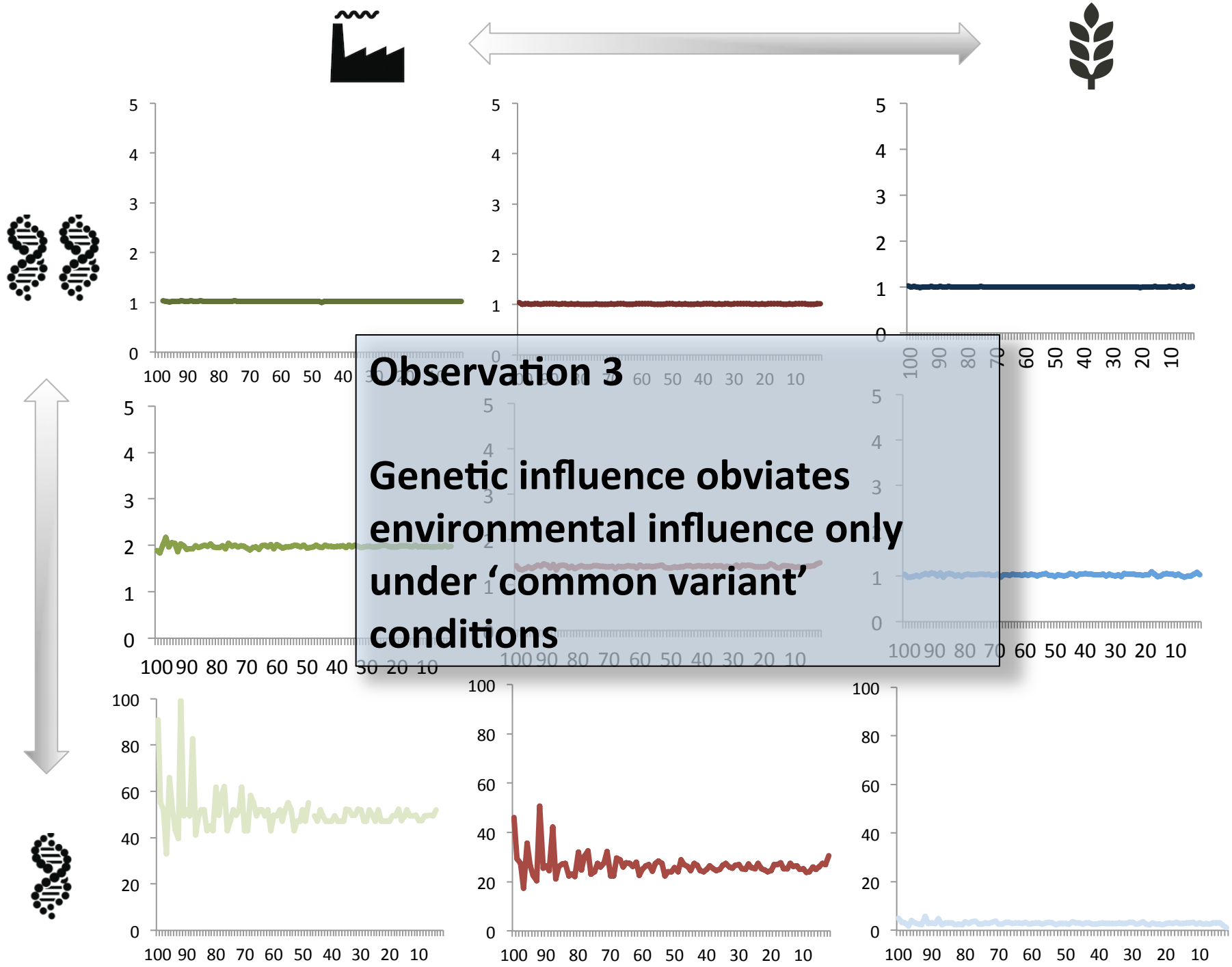

Observation 1
Prevalence of junk food eating never matters to the effect of junk food eating on obesity in the population


Observation 2
When genetic influence is low, the risk of obesity following junk food eating is high only when food environment is adverse


Observation 3
Genetic influence obviates environmental influence only under ‘common variant’ conditions

Therefore, population-wide obesity reduction effectively requires manipulation of the food environment.

Keyes KM, Davey Smith J, Koenen KC, Galea S. The mathematical limits of genetic prediction: implications for personalized medicine. Under review.
Keyes KM, Galea S. Why a focus on changing health behaviors to reduce obesity prevalence will not work: the mathematical limits of individual risk behaviors. Under review.

This is *necessarily* the case in any situation when both individual factors and features of context determine health outcomes, it is a real *mathematical limit* to improvement in health that focuses only on the individual

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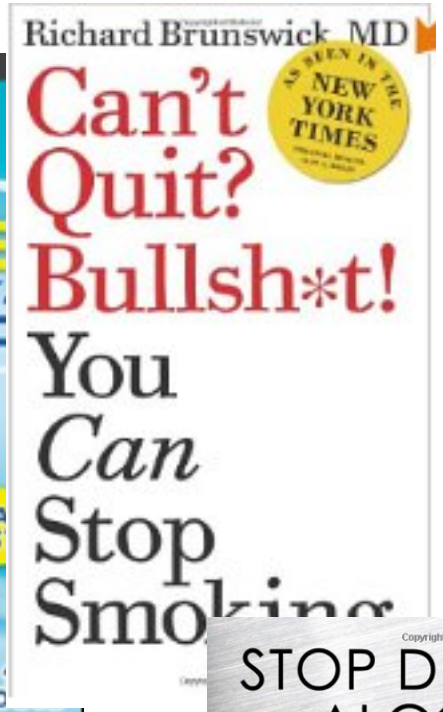
Richard Brunswick, MD
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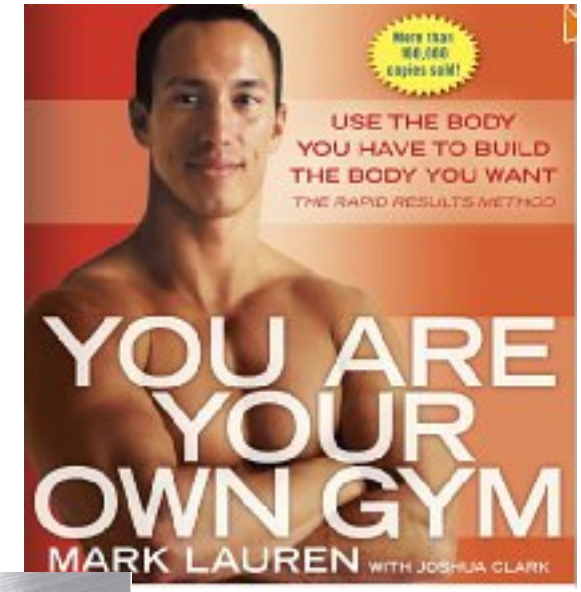
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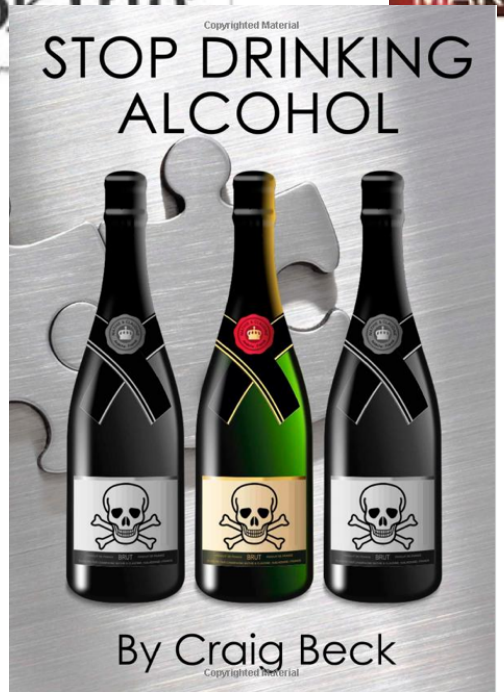
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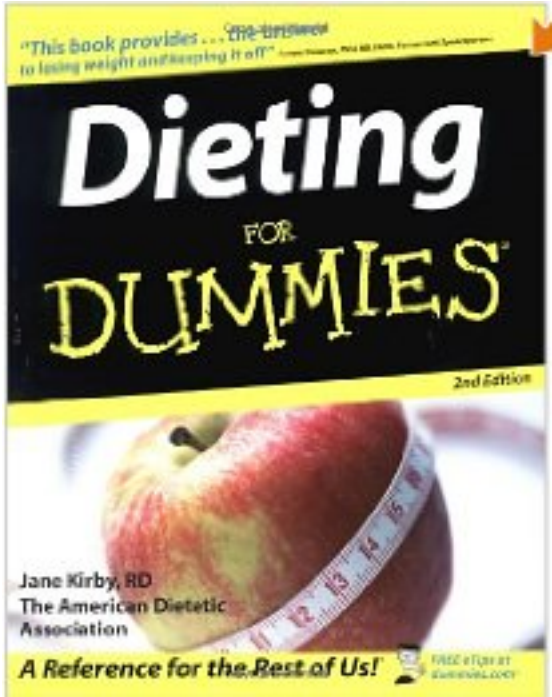
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a. Focus on what matters most

Populations **and improving their health is what matters most. This suggests that we need to**

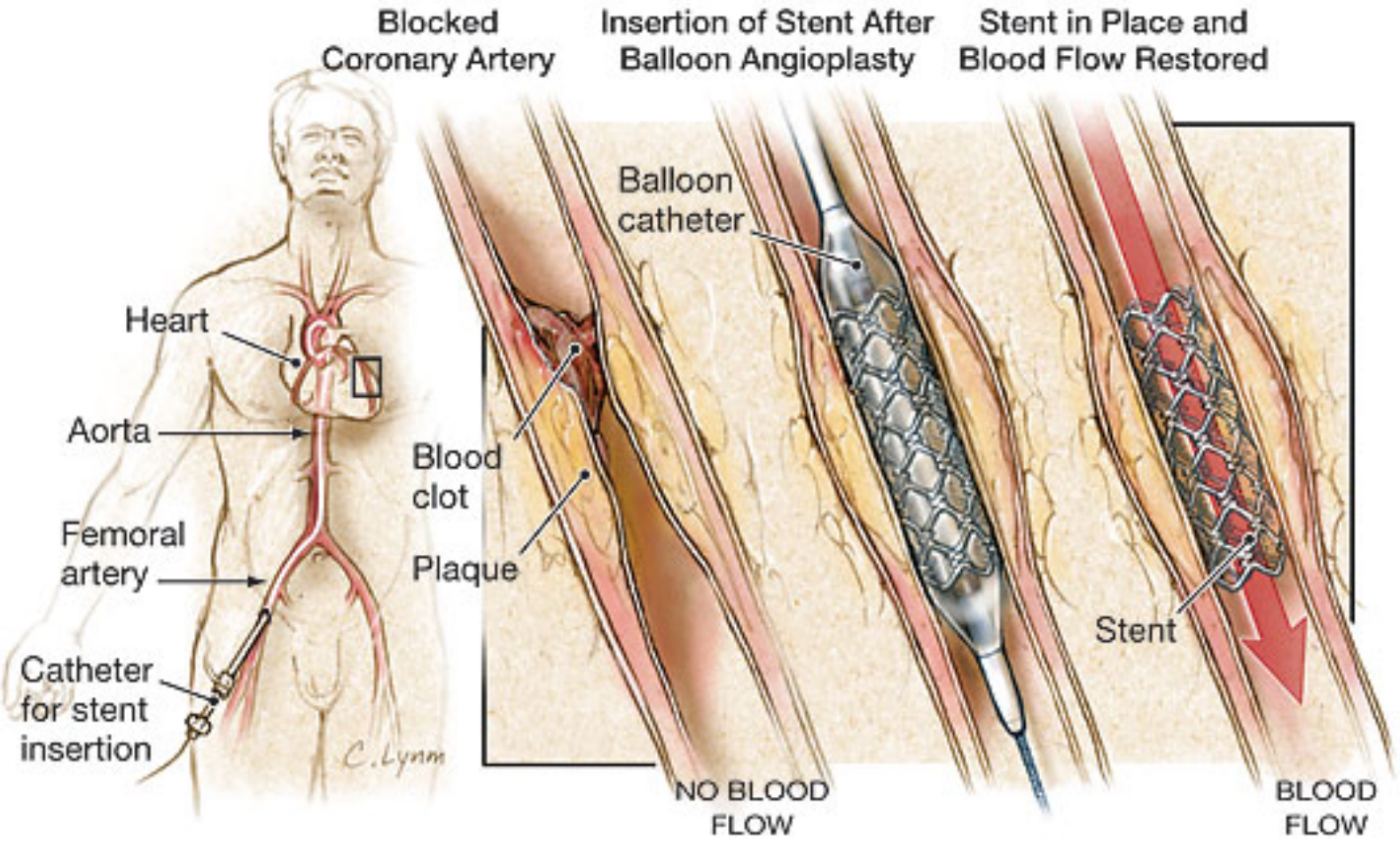
a1. Identify, and study, the factors that may have greatest impact on population health

a2. Clarify the relative contribution of factors that influence health conditions in populations

a3. Focus on understanding how intervening affects populations

a. Focus on what matters most

We know how myocardial infarctions arise



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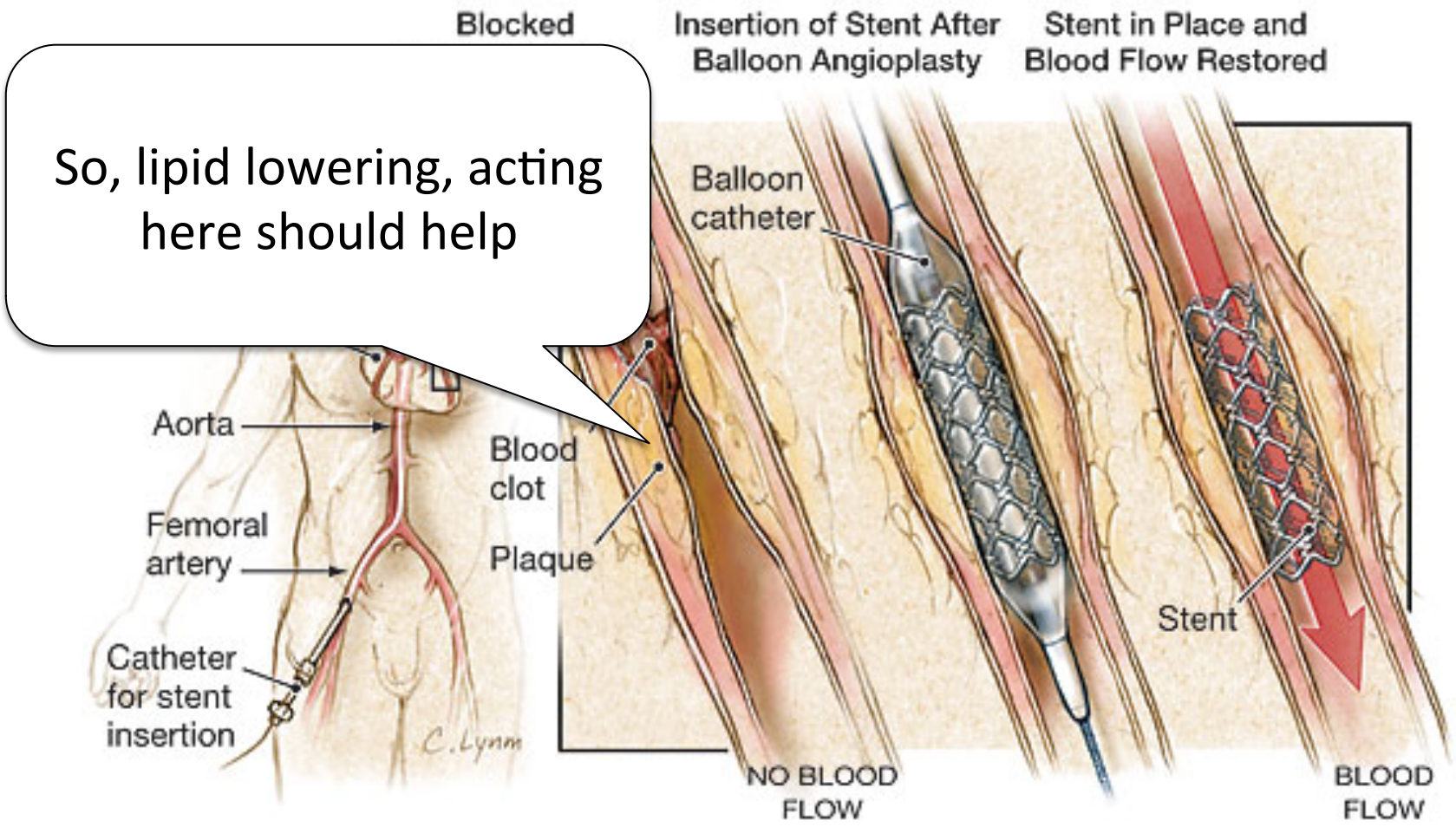
a. Focus on what matters most

We know how myocardial infarctions arise



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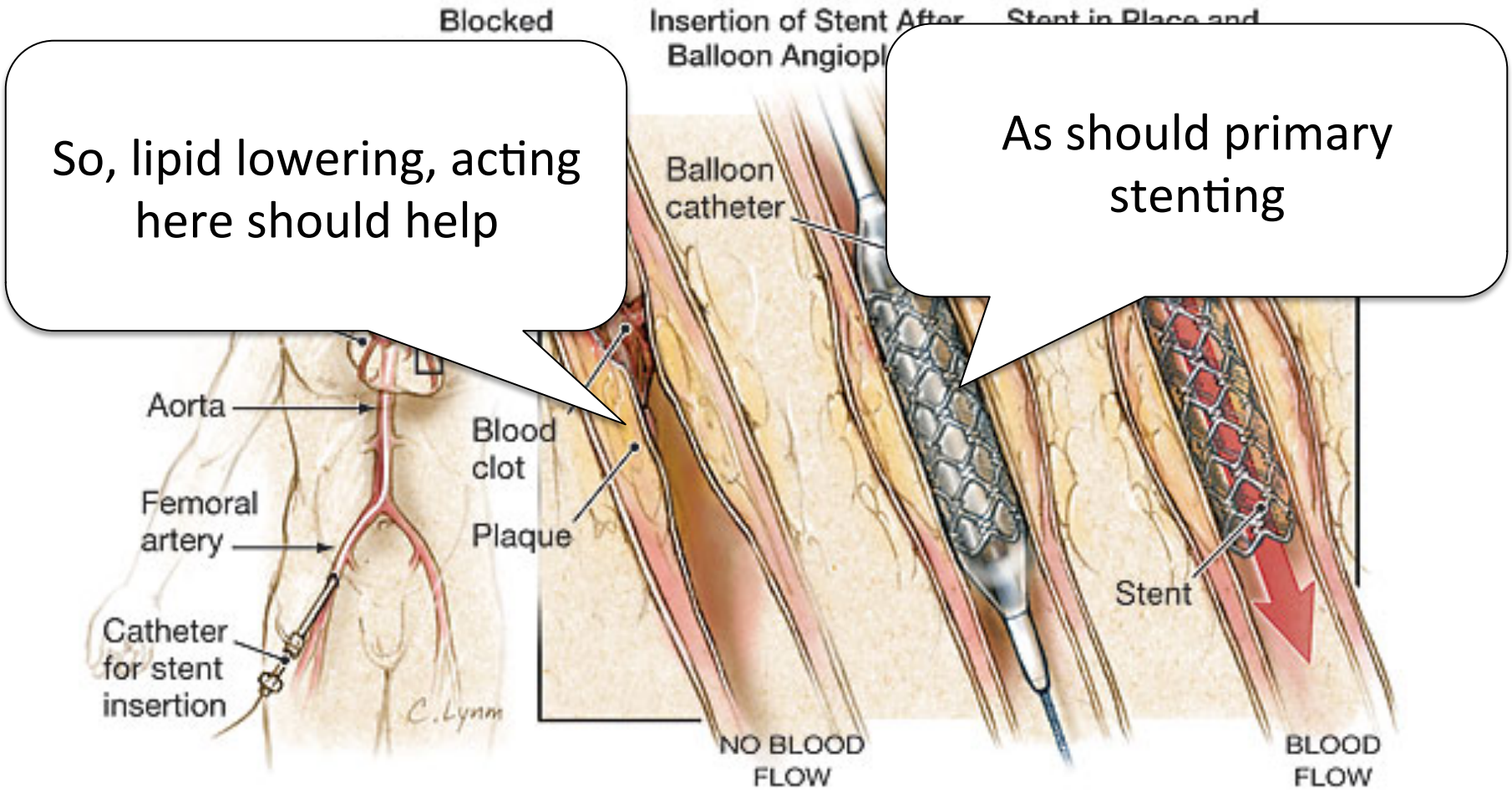

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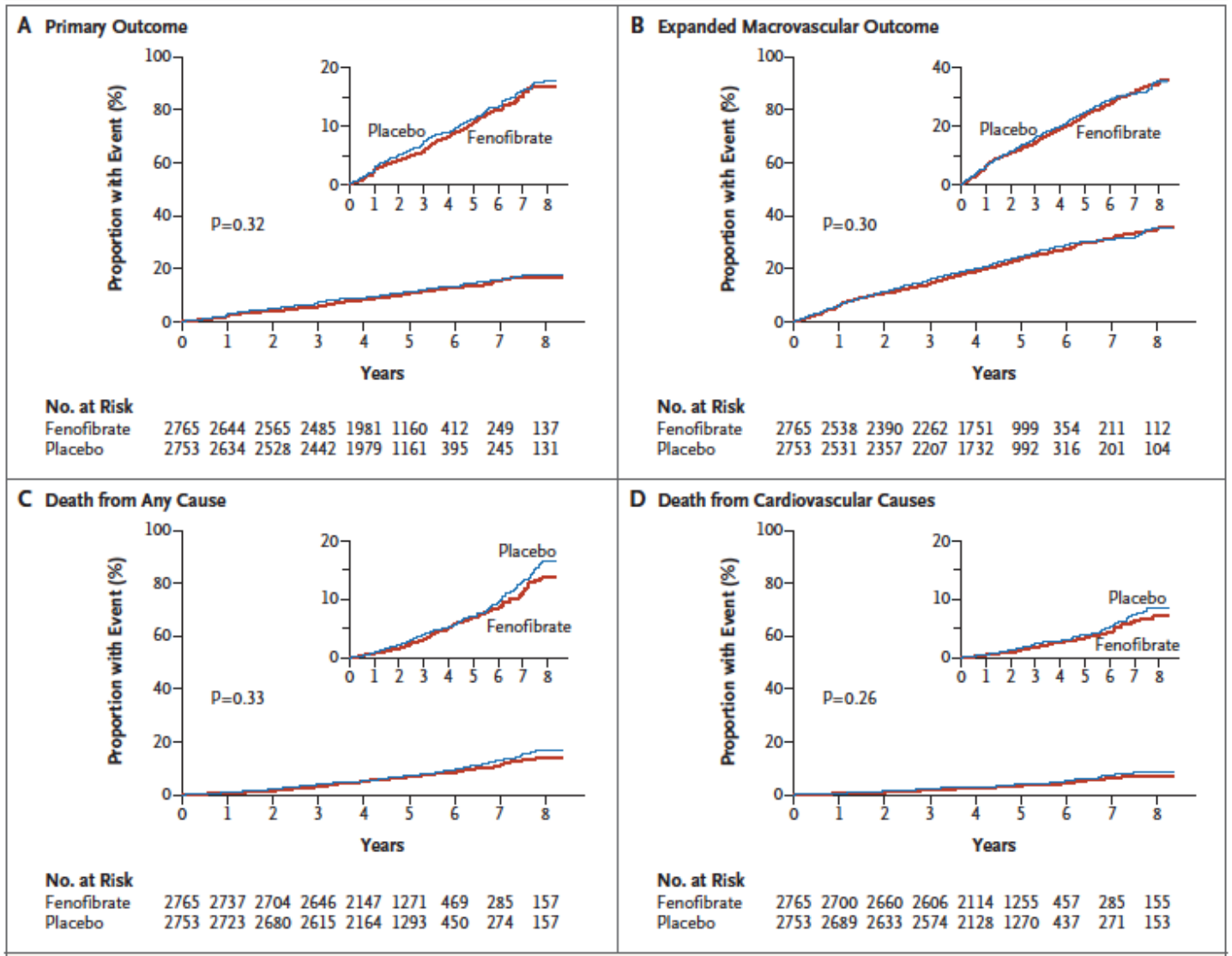


Figure 2. Kaplan–Meier Analyses of the Primary Outcome, Expanded Macrovascular Outcome, and Death.

Shown are the cumulative incidence of the primary outcome (nonfatal myocardial infarction, nonfatal stroke, or death from cardiovascular causes) (Panel A), the expanded macrovascular outcome (a combination of the primary outcome plus revascularization or hospitalization for congestive heart failure) (Panel B), and death from any cause (Panel C) or from cardiovascular causes (Panel D) during follow-up. The insets show close-up versions of the graphs in each panel.

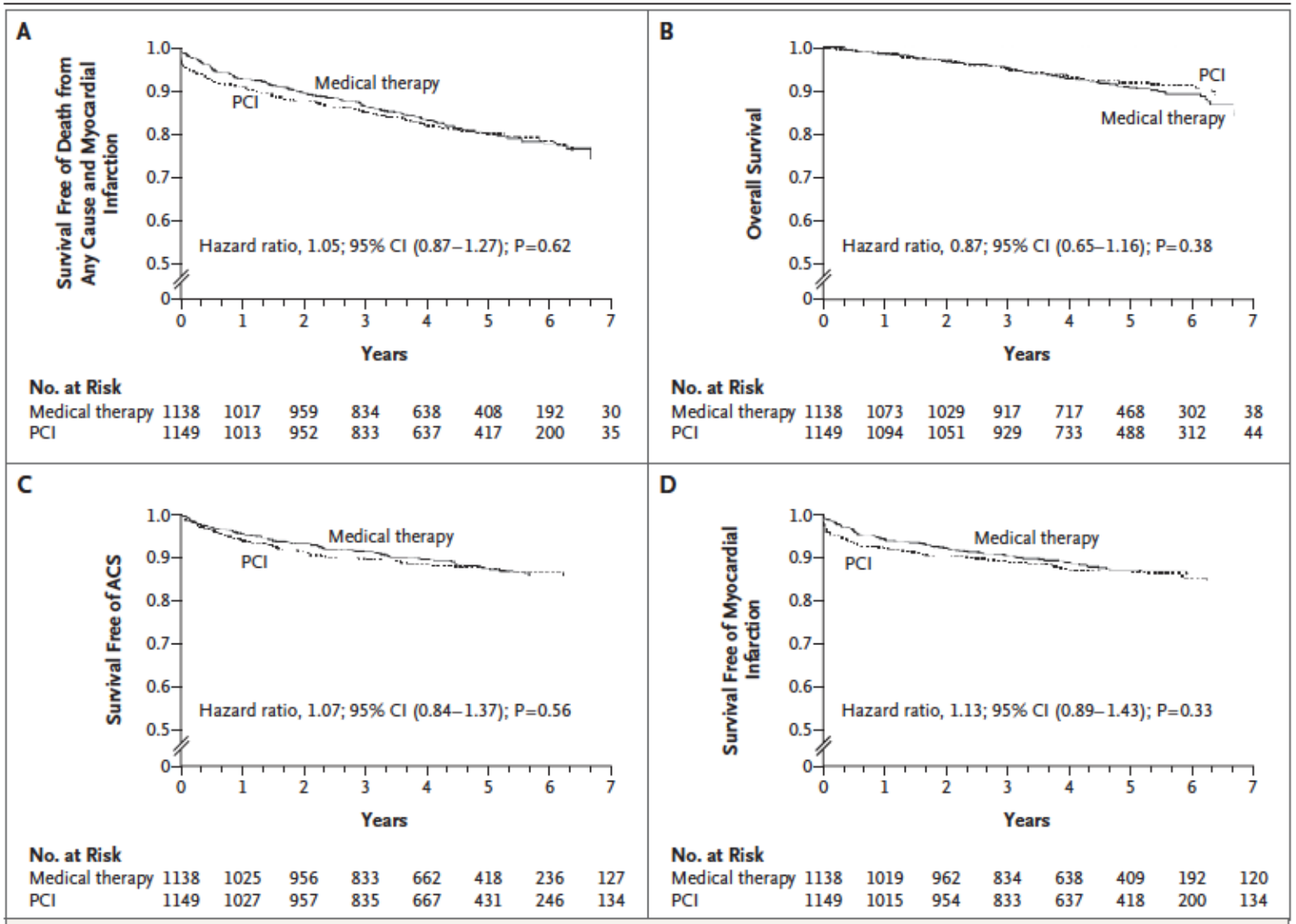


Figure 2. Kaplan–Meier Survival Curves.

In Panel A, the estimated 4.6-year rate of the composite primary outcome of death from any cause and nonfatal myocardial infarction was 19.0% in the PCI group and 18.5% in the medical-therapy group. In Panel B, the estimated 4.6-year rate of death from any cause was 7.6% in the PCI group and 8.3% in the medical-therapy group. In Panel C, the estimated 4.6-year rate of hospitalization for acute coronary syndrome (ACS) was 12.4% in the PCI group and 11.8% in the medical-therapy group. In Panel D, the estimated 4.6-year rate of acute myocardial infarction was 13.2% in the PCI group and 12.3% in the medical-therapy group.

a. Focus on what matters most

Why?

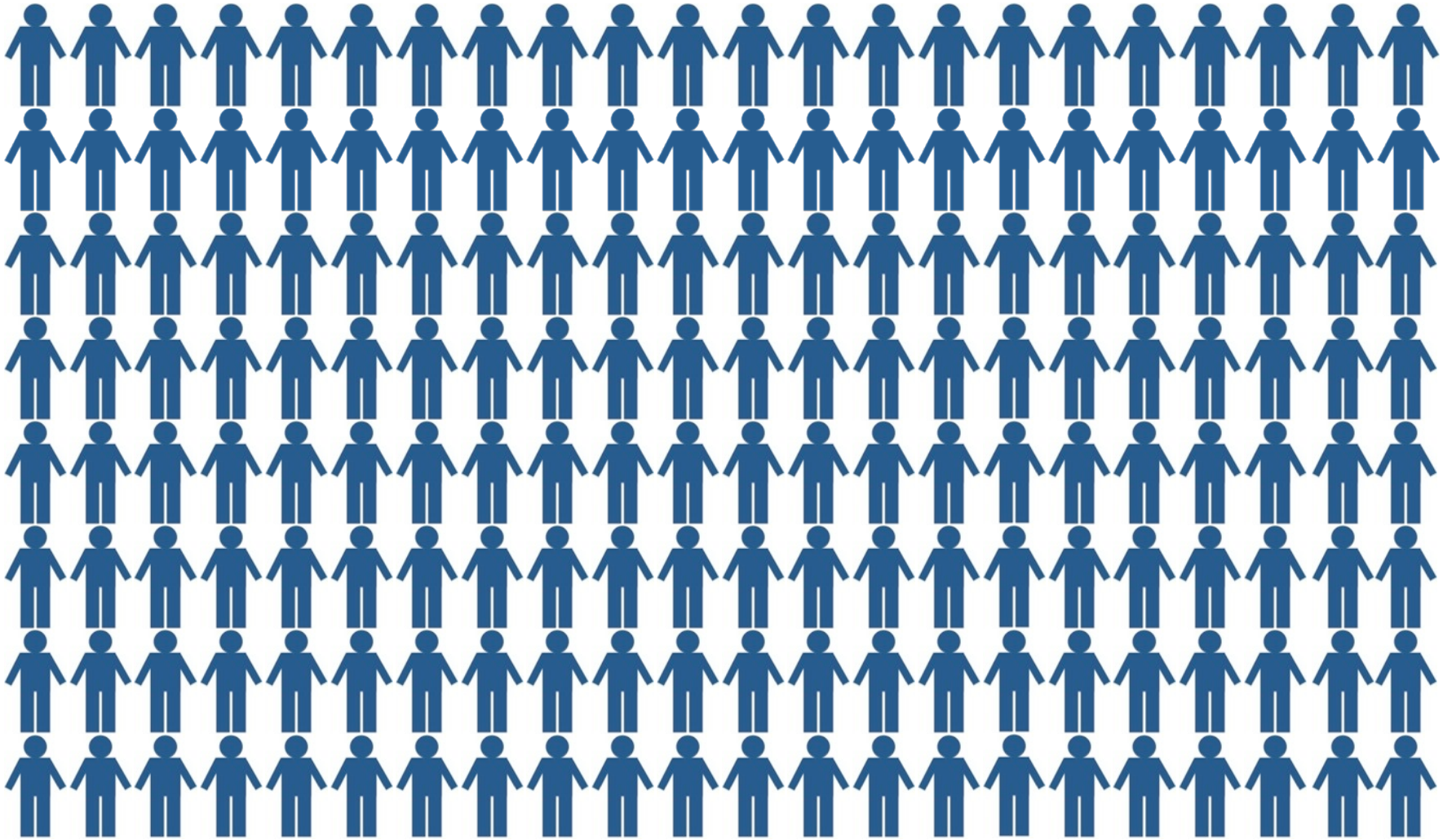
a. Focus on what matters most

> **When a study does not support the central hypothesis, it is critical to examine potential reasons for this outcome.**

The ACCORD study group. Effects of Combination Lipid Therapy in Type 2 Diabetes Mellitus. NEJM. 2010; N Engl J Med 2010;362:1563-74.

a. Focus on what matters most

Perhaps the answer lies in the challenge of taking our inference from pathophysiology and expecting generalizability of answers to populations



http://i.telegraph.co.uk/multimedia/archive/02277/population-crowd-1_2277864b.jpg

http://graphics8.nytimes.com/images/2009/03/11/opinion/13population.480.jpg

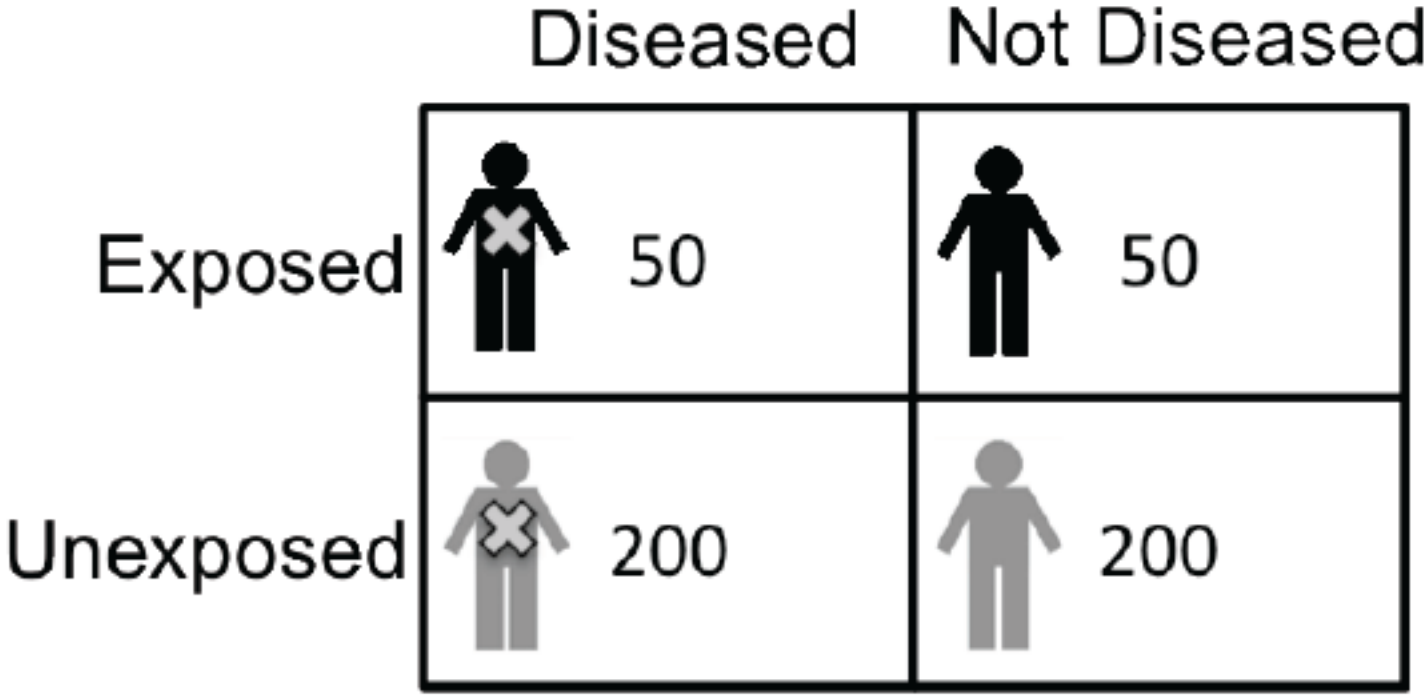
a. Focus on what matters most

Introductory epidemiology, an illustration

Let us test an exposure that may be associated with myocardial infarction

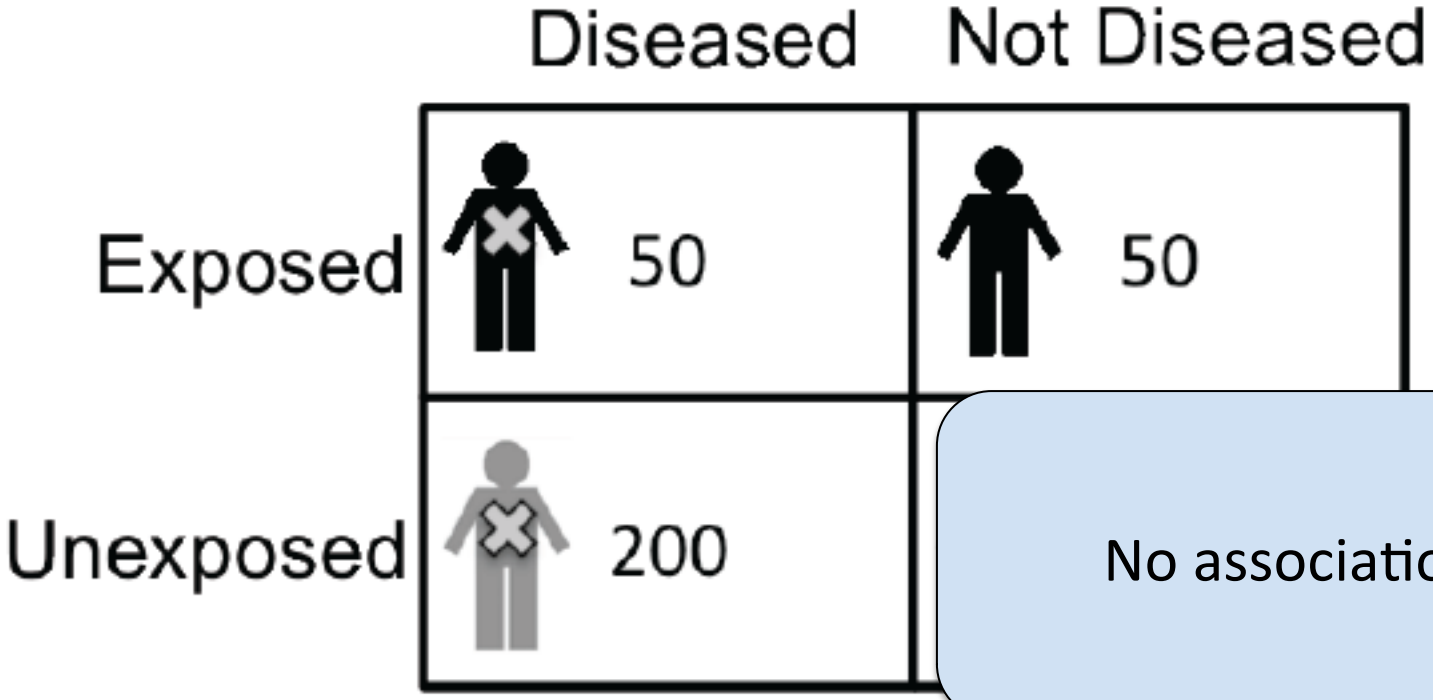
We take three samples from three populations and follow them forward in time

Sample 1 from Population 1:



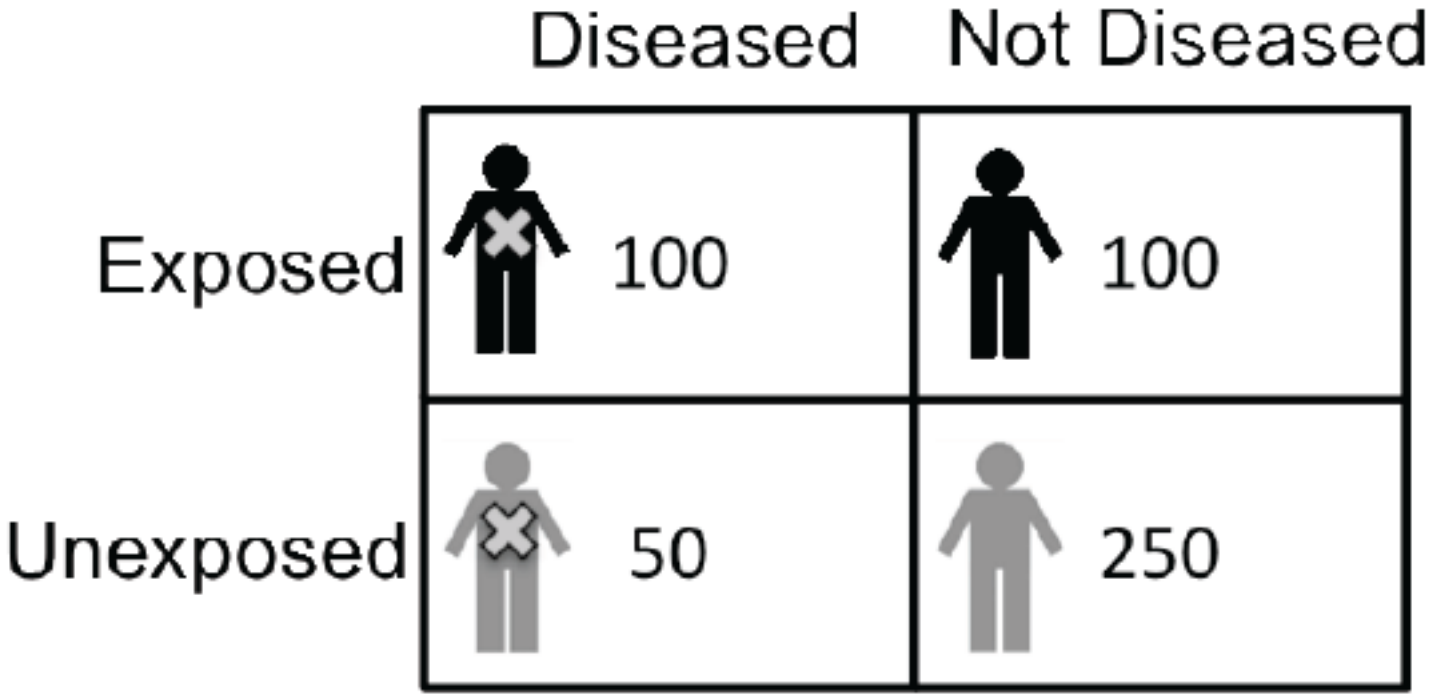
$$\text{Risk difference} = \frac{50}{100} - \frac{200}{400} = 0.5 - 0.5 = 0\ (95\%\ \text{CI}\ -0.109, 0.109)$$

Sample 1 from Population 1:



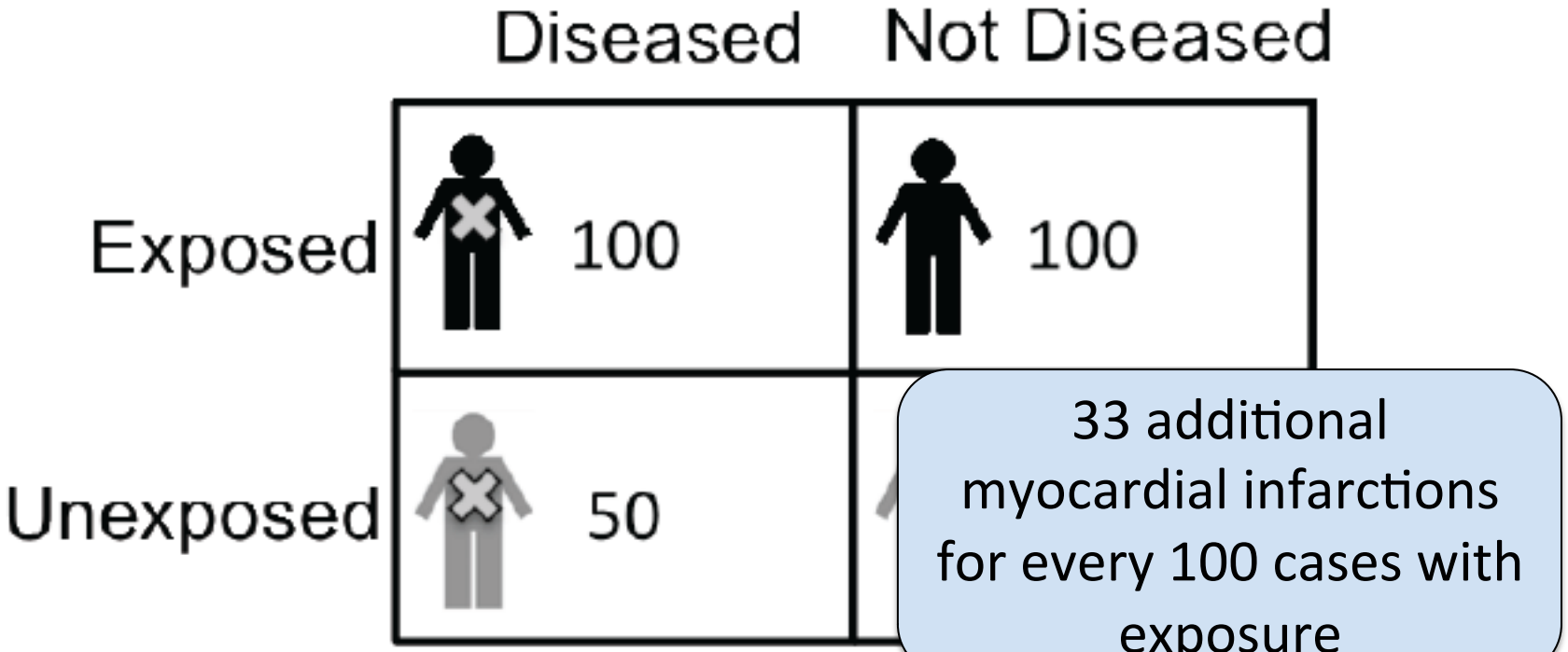
$$\text{Risk difference} = \frac{50}{100} - \frac{200}{400} = 0.5 - 0.5 = 0\ (95\%\ \text{CI}\ -0.109, 0.109)$$

Sample 2 from Population 2:



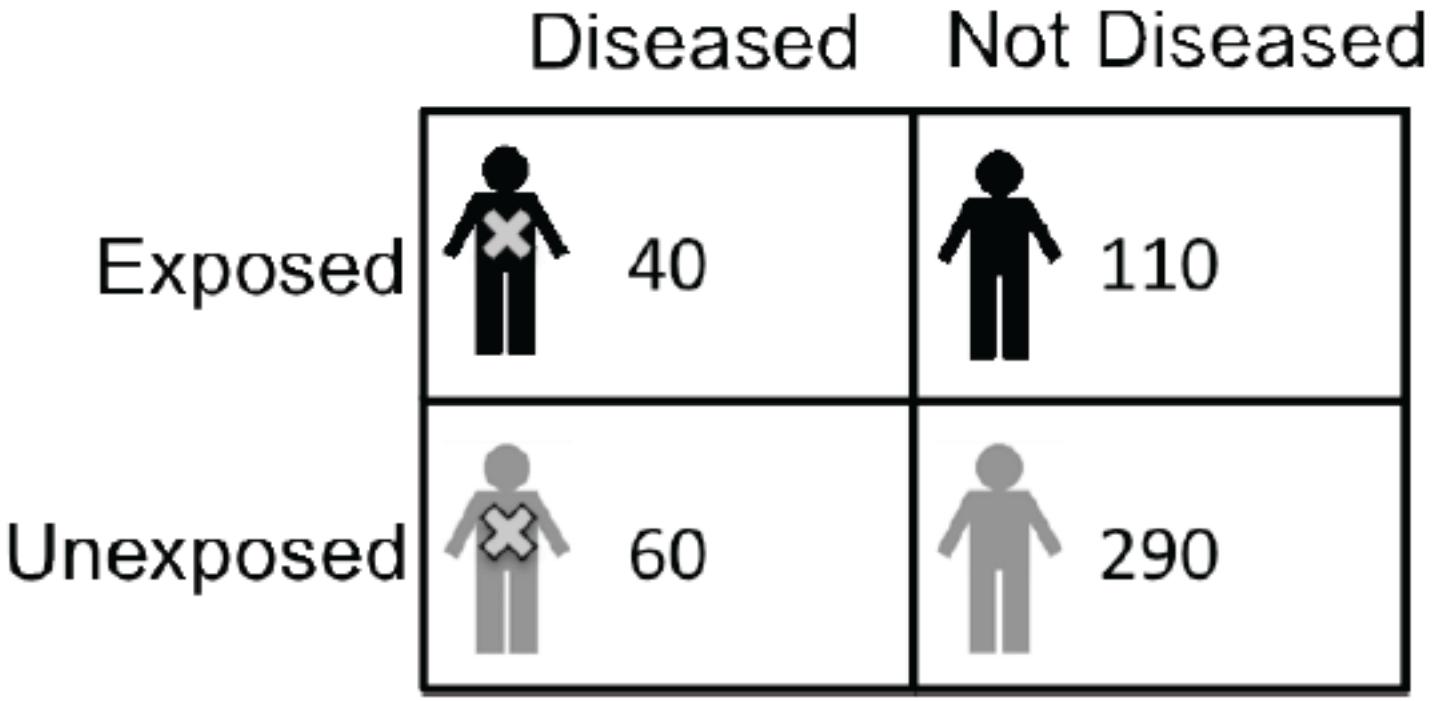
$$\text{Risk difference} = \frac{100}{200} - \frac{50}{300} = 0.5 - 0.166 = 0.333\ (95\%\ \text{CI}\ 0.252, 0.414)$$

Sample 2 from Population 2:



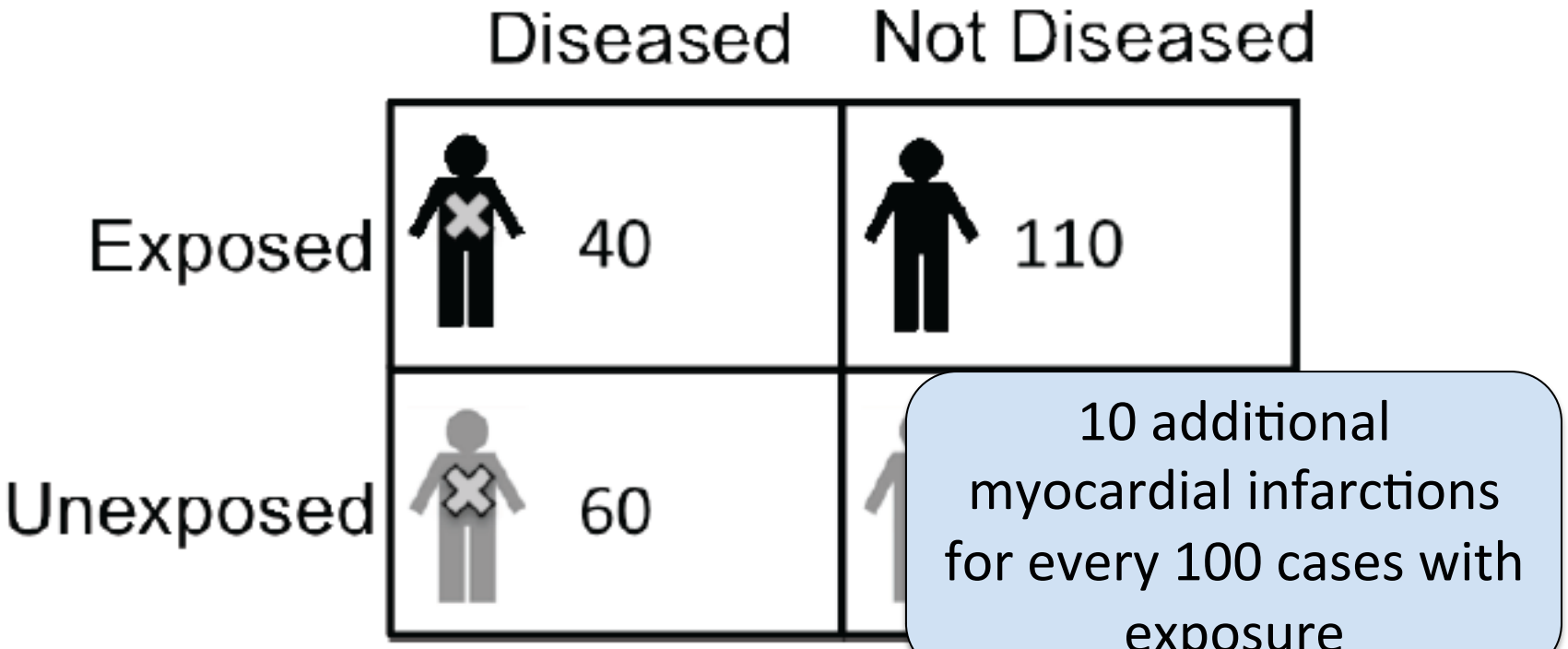
$$\text{Risk difference} = \frac{100}{200} - \frac{50}{300} = 0.5 - 0.166 = 0.333\ (95\%\ \text{CI}\ 0.252, 0.414)$$

Sample 3 from Population 3:



$$\text{Risk difference} = \frac{40}{150} - \frac{60}{350} = 0.266 - 0.171 = 0.095\ (95\%\ \text{CI}\ 0.014, 0.176)$$

Sample 3 from Population 3:



$$\text{Risk difference} = \frac{40}{150} - \frac{60}{350} = 0.266 - 0.171 = 0.095\ (95\%\ \text{CI}\ 0.014, 0.176)$$

Why?

The exposure alone does not cause myocardial infarction; the exposure needs to happen together with socioeconomic adversity

What the causal structure may look like if X, the gene, and Y, socioeconomic adversity are both necessary and insufficient causes of disease

Component Causes	
X	Y
✔	✔
✔	
	✔

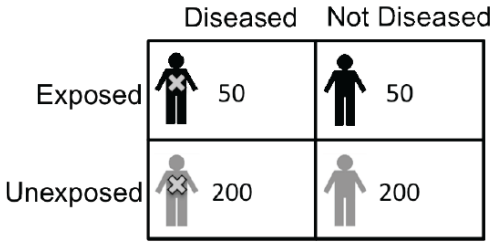
Keyes KM, Galea S. Epidemiology matters: A new introduction to methodological foundations. Oxford University Press; 2014.

What the causal structure may look like if X, the gene, and Y, socioeconomic adversity are both necessary and insufficient causes of disease

Component Causes		Probability of disease
X	Y	
✔	✔	1
✔		0
	✔	0
		0

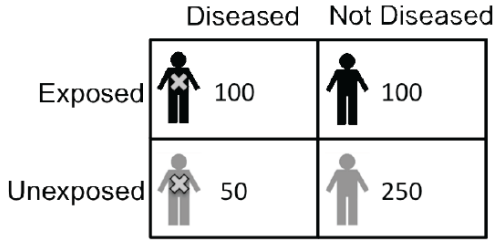
Keyes KM, Galea S. Epidemiology matters: A new introduction to methodological foundations. Oxford University Press; 2014.

Sample 1 from Population 1:



Risk difference $= \frac{50}{100} - \frac{200}{400} = 0.5 - 0.5 = 0$ (95% CI $-0.109, 0.109$)

Sample 2 from Population 2:



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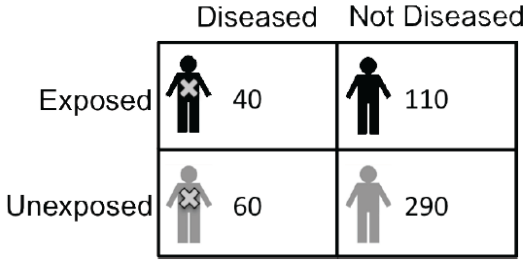

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0% low SES

Sample 1 from Population 1:

	Diseased	Not Diseased
Exposed	50	50
Unexposed	200	200

Risk difference $= \frac{50}{100} - \frac{200}{400} = 0.5 - 0.5 = 0$ (95% CI $-0.109, 0.109$)

50% low SES

Sample 2 from Population 2:

	Diseased	Not Diseased
Exposed	100	100
Unexposed	50	250

Risk difference $= \frac{100}{200} - \frac{50}{300} = 0.5 - 0.166 = 0.333$ (95% CI 0.252, 0.414)

40% low SES

Sample 3 from Population 3:

	Diseased	Not Diseased
Exposed	40	110
Unexposed	60	290

Risk difference $= \frac{40}{150} - \frac{60}{350} = 0.266 - 0.171 = 0.095$ (95% CI 0.014, 0.176)

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0% low SES

50% low SES

40% low sES

Sample 1 from Population 1:

	Diseased	Not Diseased
Exposed	50	50
Unexposed	200	200

Risk difference $= \frac{50}{100} - \frac{200}{400} = 0.5 - 0.5 = 0$ (95% CI $-0.109, 0.109$)

Sample 2 from Population 2:

	Diseased	Not Diseased
Exposed	100	100
Unexposed	50	250

Risk difference $= \frac{100}{200} - \frac{50}{300} = 0.5 - 0.166 = 0.333$ (95% CI 0.252, 0.414)

Sample 3 from Population 3:

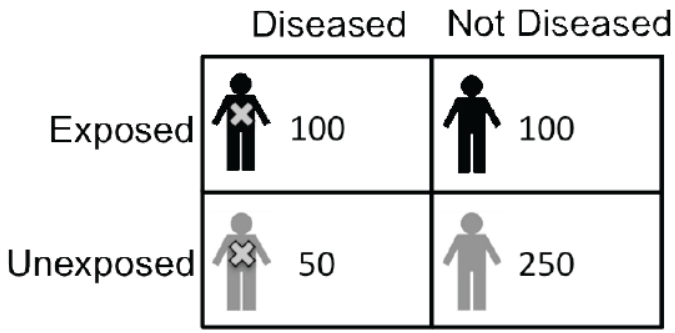
	Diseased	Not Diseased
Exposed		
Unexposed		

Risk difference

Component Causes		Probability of disease
X	Y	
✔	✔	1
✔		0
	✔	0
		0

No myocardial infarctions due to the exposure because the causal partner is not present; hence equal likelihood of myocardial infarction among those exposed/unexposed

Sample 2 from Population 2:



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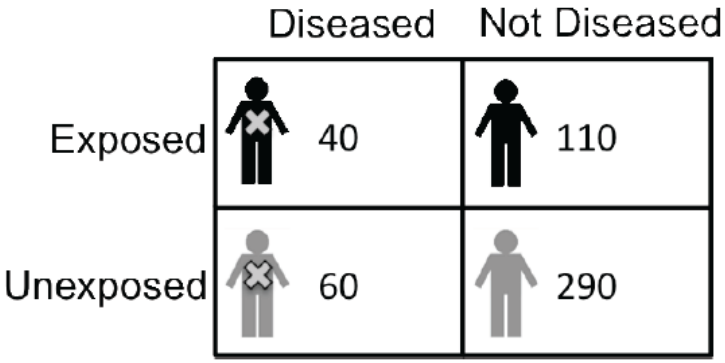
Sample 3 from Population 3:



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Keyes KM, Galea S. Epidemiology matters: A new introduction to methodological foundations. Oxford University Press; 2014.

50% of those who are exposed also have low SES, hence die prematurely

40% of those who are exposed also have low SES, hence die prematurely

Sample 2 from Population 2:

	Diseased	Not Diseased
Exposed	100	100
Unexposed	50	250

Risk difference $= \frac{100}{200} - \frac{50}{300} = 0.5 - 0.166 = 0.333$ (95% CI 0.252, 0.414)

Sample 3 from Population 3:

	Diseased	Not Diseased
Exposed	40	110
Unexposed	60	290

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17% of those who are unexposed have a myocardial infarction

17% of those who are unexposed have a myocardial infarction

Sample 2 from Population 2:

	Diseased	Not Diseased
Exposed	100	100
Unexposed	50	250

$$\text{Risk difference} = \frac{100}{200} - \frac{50}{300} = 0.5 - 0.166 = 0.333 \ (95\% \text{ CI } 0.252, 0.414)$$

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Under a very plausible assumption of co-occurring causes, any given sample will give us different estimates for exposure-death association dependent entirely on *other* factors that distinguish between samples

“But you see, Meg, just because we don't understand doesn't mean that the explanation doesn't exist.”

Madeleine L’Engle, A wrinkle in time

An epidemiology of consequence should

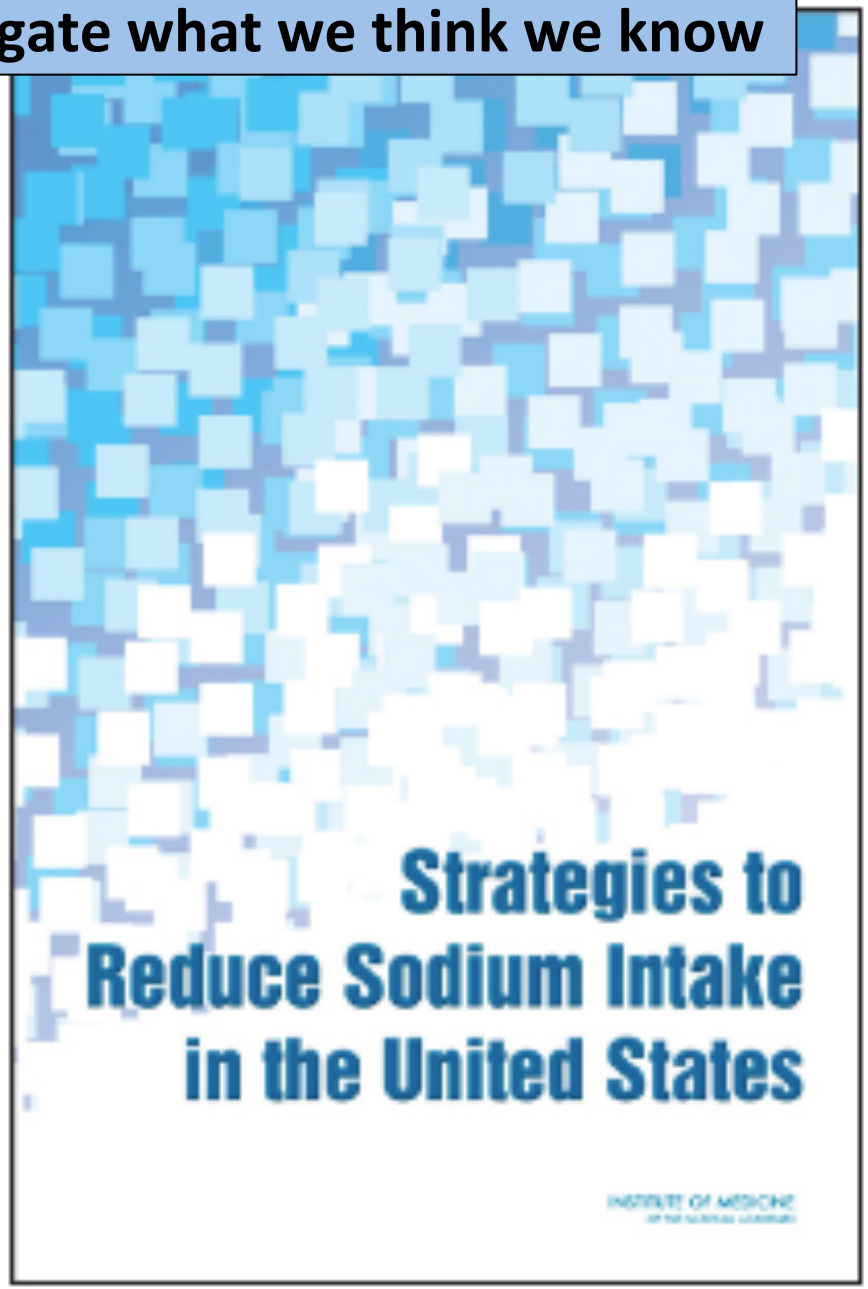
a. Focus on what matters most

b. Critically interrogate what we think we know

c. Engage in translating the science

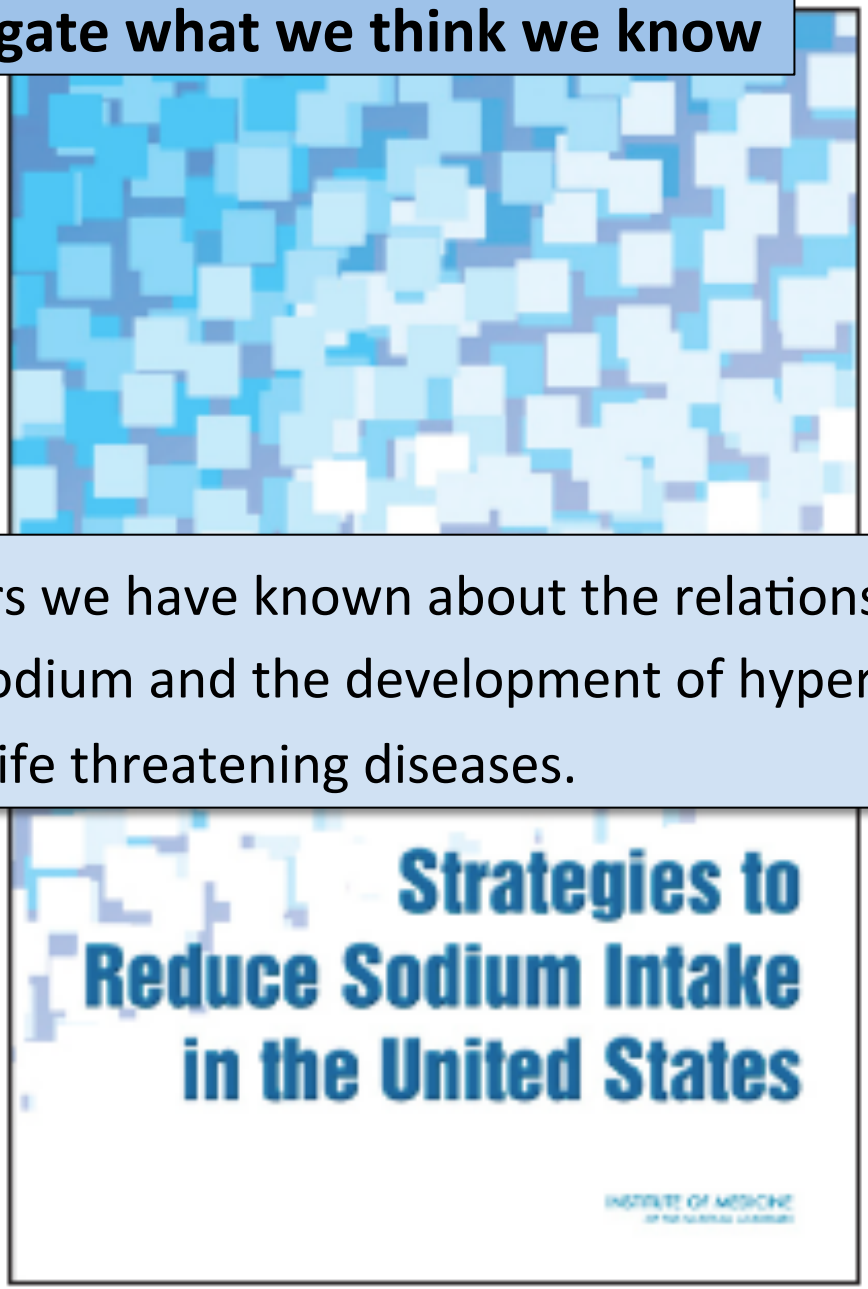
d. Teach epidemiology differently

b. Critically interrogate what we think we know



Bayer R, Johns D, Galea S. Salt, Science, And Public Health: The Challenge Of Evidence-Based Decision Making. Health Affairs. 2012; 31(12): 2738-2746. PMID: 23213158.

b. Critically interrogate what we think we know



For 40 years we have known about the relationship between sodium and the development of hypertension and other life threatening diseases.

Bayer R, Johns D, Galea S. Salt, Science, And Public Health: The Challenge Of Evidence-Based Decision Making. Health Affairs. 2012; 31(12): 2738-2746. PMID: 23213158.

b. Critically interrogate what we think we know

EDITORIAL | Annals of Internal Medicine

We Can Reduce Dietary Sodium, Save Money, and Save Lives

Most Americans consume far more salt than is healthy; the average sodium intake has increased over the past 30 years from already high levels to more than double the recommended amount (1, 2). Excess sodium consumption increases blood pressure (3); each 20–mm Hg increase in systolic blood pressure above 115 mm Hg doubles the risk for heart attack and stroke (4), which are the first and third leading causes of death in the United States, respectively (5). These effects of increased blood pressure on heart attack and stroke begin to occur at blood pressures that are well below levels at which drug treatment of hypertension is recommended currently (6).

Worldwide, cardiovascular disease is the leading cause of death among people aged 60 years or older and second among those aged 15 to 59 years; half or more of all strokes and heart attacks are attributable to high blood pressure (7). In the United States, approximately 100 000 deaths each year have been attributed to excess sodium intake (8). Because about one third of U.S. adults have hypertension and another 28% have levels above the desirable range (9), and because sodium consumption contributes to the increase in blood pressure observed with increasing age (9), reductions in salt intake will lead to substantial population-wide improvements in health.

Clinical care and health education require considerable individual attention and effort to help one person at a time through medical treatment or to adopt healthy behaviors. Policy interventions that change the environment to make savings of $18 billion in direct health care costs (15). Still-larger decreases in sodium intake than were examined in this study would probably result in even larger health improvements and cost savings and would be more cost-effective than using medications to lower blood pressure in people with hypertension (13).

After tobacco control, the most cost-effective intervention to control chronic diseases might be reduction of sodium intake. But because more than three fourths of Americans' sodium intake comes from processed foods and restaurant meals (16), it is very difficult for individuals to limit their consumption to healthy levels. As a result, sodium reduction will rely on action by the food industry (14, 17).

Sodium reduction initiatives involving the food industry in other countries have been successful. In 2003, the United Kingdom introduced a voluntary strategy to decrease the sodium content of processed and packaged food, which has resulted in reductions of 20% to 30% in most processed food sold in stores (14). New sodium reduction targets in the United Kingdom are being established and are expected to lead to a total 40% reduction in population sodium intake by 2012 (14). Japan and Finland have also implemented effective salt reduction programs; Ireland, Australia, and Canada have recently begun similar initiatives; and many other countries have committed to reducing sodium intake at the population level (14).

Although substantial changes in food production will

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b. Critically interrogate what we think we know

REDUCING POPULATION SALT INTAKE

The sodium phantom

Niels Graudal *senior consultant*[1], Gesche Jürgens *MD*[2]

[1]Department of Rheumatology, Copenhagen University Hospital, Rigshospitalet, DK-2100 Copenhagen, Denmark; [2]Department of Clinical Pharmacology, Bispebjerg University Hospital, Copenhagen

BMJ

Niels Graudal and G. Jurgens. "The sodium phantom," BMJ 2011;343:d6119

b. Critically interrogate what we think we know

REDUCING POPULATION SALT INTAKE

The sodium phantom

Niels Graudal *senior consultant*[1], Gesche Jürgens *MD*[2]

[1]Department ... of Clinical Pharmacolog...

It is surprising that many countries have uncritically adopted sodium reduction, which probably is the largest delusion in the history of preventive medicine.

Niels Graudal and G. Jurgens. "The sodium phantom," BMJ 2011;343:d6119

b. Critically interrogate what we think we know

Effects of low sodium diet versus high sodium diet on blood pressure, renin, aldosterone, catecholamines, cholesterol, and triglyceride (Review)

Graudal NA, Hubeck-Graudal T, Jurgens G

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Effects of low sodium diet versus high sodium diet on blood pressure, renin, aldosterone, catecholamines, cholesterol, and triglyceride (Review)

Graudal NA, Hubeck-Graudal T, Jurgens G

We are commonly advised to cut down on salt…[but] we do not know if low salt diets improve or worsen health outcomes.

This is a reprint of a Cochrane review, prepared and maintained by The Cochrane Collaboration and published in *The Cochrane Library* 2011, Issue 11

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Reduced Dietary Salt for the Prevention of Cardiovascular Disease: A Meta-Analysis of Randomized Controlled Trials (Cochrane Review)

Rod S. Taylor[1], Kate E. Ashton[2], Tiffany Moxham[3], Lee Hooper[4] and Shah Ebrahim[5]

BACKGROUND

Although meta-analyses of randomized controlled trials (RCTs) of salt reduction report a reduction in the level of blood pressure (BP), the effect of reduced dietary salt on cardiovascular disease (CVD) events remains unclear.

METHODS

We searched for RCTs with follow-up of at least 6 months that compared dietary salt reduction (restricted salt dietary intervention or advice to reduce salt intake) to control/no intervention in adults, and reported mortality or CVD morbidity data. Outcomes were pooled at end of trial or longest follow-up point.

RESULTS

Seven studies were identified, three in normotensives, two in hypertensives, one in a mixed population of normo- and hypertensives and one in heart failure. Salt reduction was associated with reductions in urinary salt excretion of between 27 and 39 mmol/24 h and reductions in systolic BP between 1 and 4 mm Hg. Relative risks (RRs) for all-cause mortality in normotensives (longest follow-up—RR: 0.90, 95% confidence interval (CI): 0.58–1.40, 79 deaths) and hypertensives (longest follow-up RR 0.96, 0.83–1.11, 565 deaths) showed no strong evidence of any effect of salt reduction CVD morbidity in people with normal BP (longest follow-up: RR 0.71, 0.42–1.20, 200 events) and raised BP at baseline (end of trial: RR 0.84, 0.57–1.23, 93 events) also showed no strong evidence of benefit. Salt restriction increased the risk of all-cause mortality in those with heart failure (end of trial RR 2.59, 1.04–6.44, 21 deaths).We found no information on participant's health-related quality of life.

CONCLUSIONS

Despite collating more event data than previous systematic reviews of RCTs (665 deaths in some 6,250 participants) there is still insufficient power to exclude clinically important effects of reduced dietary salt on mortality or CVD morbidity. Our estimates of benefits from dietary salt restriction are consistent with the predicted small effects on clinical events attributable to the small BP reduction achieved.

Keywords: *blood pressure; cardiovascular disease; diet; hypertension; meta-analysis; salt; sodium; systematic review*

This article is based on a Cochrane Review published in the *Cochrane Database of Systematic Reviews* (*CDSR*) YYYY, Issue X, DOI: 10.1002/14651858.CD00xxxx (see www.thecochranelibrary.com for information). Cochrane Reviews are regularly updated as new evidence emerges and in response to feedback, and the *CDSR* should be consulted for the most recent version of the review.

A more detailed review has been published and will be updated in the Cochrane Database of Systematic Reviews [Taylor RS, Ashton KE, Moxham T, Hooper L, Ebrahim S. Reduced dietary salt for the prevention Of cardiovascular disease. Cochrane Database of Systematic Reviews (CDSR) 2011, Issue X, DOI: 10.1002/14651858.CD00xxxx (see www.thecochranelibrary.com for information). This is a version of a Cochrane review, which is available in The Cochrane Library. Cochrane systematic reviews are regularly updated to include new research, and in response to feedback from readers. The results of a Cochrane review can be interpreted differently, depending on people's perspectives and circumstances. Please consider the conclusions presented carefully. They are the opinions of review authors, and are not necessarily shared by The Cochrane Collaboration.

American Journal of Hypertension, advance online publication 6 July 2011; doi:10.1038/ajh.2011.115

High dietary intake of salt has been identified as an important risk factor for cardiovascular disease (CVD). The current public health recommendations in most developed countries are to reduce salt intake by about half, i.e., from ~10 to 5 g/day.[1–4] However, the evidence for the reduction of CVD morbidity and mortality as the result of reduced salt intake remains controversial.[5]

A number of observational studies support the link between salt intake and CVD. A meta-analysis of 13 prospective studies including 177,000 participants reported a high salt intake was associated with a greater risk of stroke (RR, 1.23, 95% confidence interval (CI): 1.06–1.43).[6] However, there was no association between salt intake and all CVD events, and total mortality was not reported. Furthermore, the interpretation of this observational evidence base is complicated by the heterogeneity in estimating sodium intake (diet or urinary salt excretion), types of participants (healthy, hypertensive, obese, and nonobese), different end points, and definition of outcomes across studies.[5]

[1]Peninsula College of Medicine and Dentistry, University of Exeter, Exeter, UK; [2]Clinical Trials and Evaluation Unit, University of Bristol, Bristol, UK; [3]Wimberly Library, Florida Atlantic University, Boca Raton, Florida, USA; [4]Norwich Medical School, University of East Anglia, Norwich, UK; [5]Faculty of Epidemiology and Population Health, London School of Hygiene and Tropical Medicine, London, UK. Correspondence: Rod S. Taylor (rod.taylor@pms.ac.uk)

Received 1 May 2011; first decision 9 May 2011; accepted 9 May 2011.

Reduced Dietary Salt for the Prevention of Cardiovascular Disease: A Meta-Analysis of Randomized Controlled Trials (Cochrane Review)

Rod S. Taylor[1], Kate E. Ashton[2], Tiffany Moxham[3], Lee Hooper[4] and Shah Ebrahim[5]

BACKGROUND
Although meta-analyses of randomized controlled trials (RCTs) of salt reduction report a reduction in the level of blood pressure (BP), the effect of reduced dietary salt on cardiovascular disease (CVD) events remains unclear.

METHODS
We searched for RCTs with follow-up of at least 6 months that compared dietary salt reduction (restricted salt dietary intervention or advice to reduce salt intake) to control/no intervention in adults, and reported mortality or CVD morbidity data. Outcomes were pooled at end of trial or longest follow-up point.

CONCLUSIONS
Despite collating more event data than previous systematic reviews of RCTs (665 deaths in some 6,250 participants) there is still insufficient power to exclude clinically important effects of reduced dietary salt on mortality or CVD morbidity. Our estimates of benefits from dietary salt restriction are consistent with the predicted small effects on clinical events attributable to the small BP reduction achieved.

Keywords: blood pressure; cardiovascular disease; diet; hypertension; meta-analysis; salt; sodium; systematic review

This article is based on a Cochrane Review published in the *Cochrane Database*

We found no strong evidence that salt reduction reduced all-cause mortality or CVD morbidity in normotensives or hypertensives.

0.57–1.23, 93 events) also showed no strong evidence of benefit. Salt restriction increased the risk of all-cause mortality in those with heart failure (end of trial RR 2.59, 1.04–6.44, 21 deaths).We found no information on participant's health-related quality of life.

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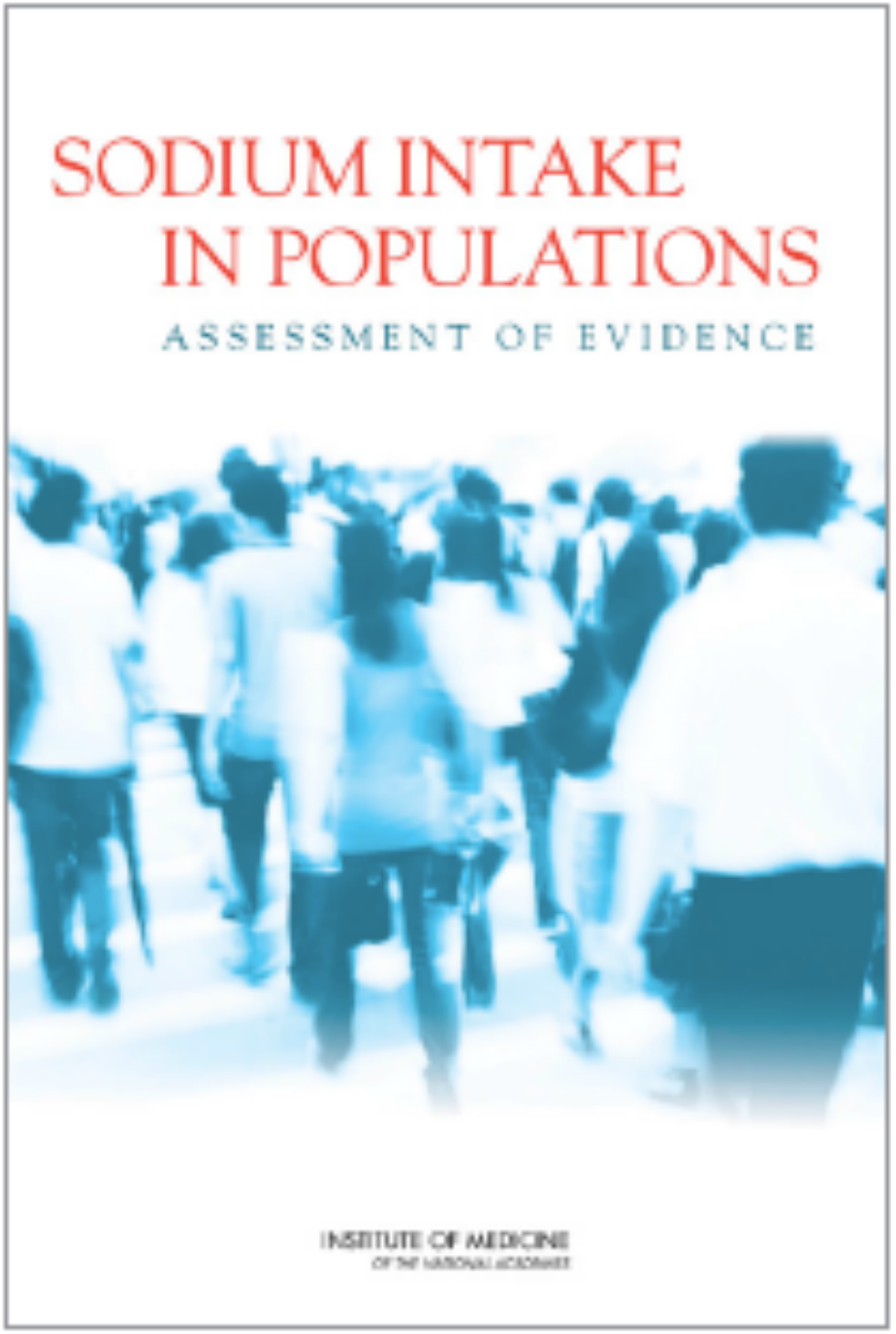
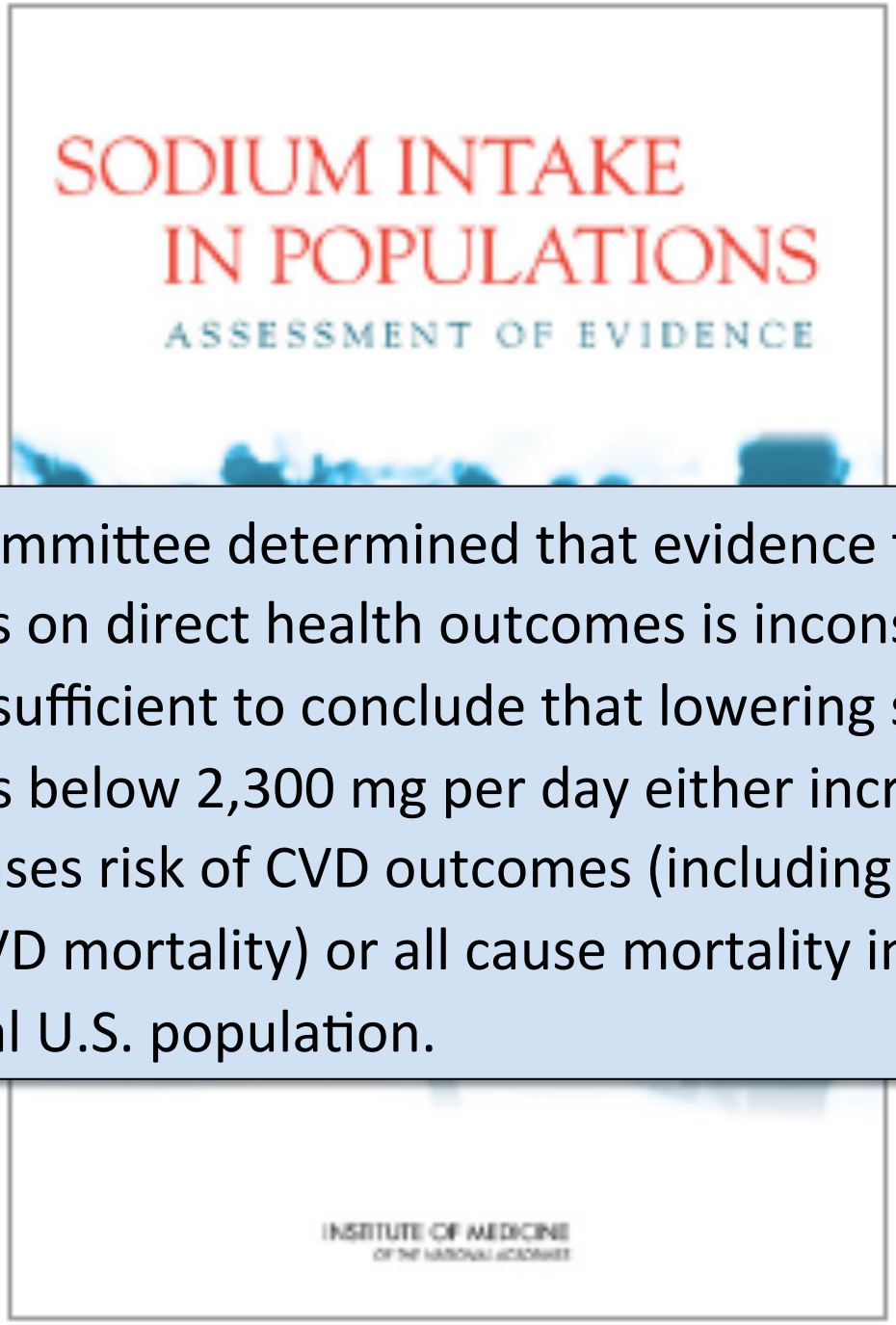
Received 1 May 2011; first decision 9 May 2011; accepted 9 May 2011.

Bayer R, Johns D, Galea S. Salt, Science, And Public Health: The Challenge Of Evidence-Based Decision Making. Health Affairs. 2012; 31(12): 2738-2746. PMID: 23213158.



Bayer R, Johns D, Galea S. Salt, Science, And Public Health: The Challenge Of Evidence-Based Decision Making. Health Affairs. 2012; 31(12): 2738-2746. PMID: 23213158.



The committee determined that evidence from studies on direct health outcomes is inconsistent and insufficient to conclude that lowering sodium intakes below 2,300 mg per day either increases or decreases risk of CVD outcomes (including stroke and CVD mortality) or all cause mortality in the general U.S. population.

Bayer R, Johns D, Galea S. Salt, Science, And Public Health: The Challenge Of Evidence-Based Decision Making. Health Affairs. 2012; 31(12): 2738-2746. PMID: 23213158.

Controversial Salt Report Peppered with Uncertainty

A RECENT INSTITUTE OF MEDICINE (IOM) assessment (*1*) provoked controversy by concluding that there is a lack of evidence for health benefits of reducing sodium intake to the very low levels recommended by some authoritative groups ("Report reignites battle over low-salt diets," K. Kupferschmidt, News & Analysis, 24 May, p. 908). The IOM

mentary by three members of the IOM salt committee likewise stressed that the Dietary Guidelines for Americans (DGA), IOM, AHA, and the World Health Organization (WHO) were "congruent" in the belief that excess sodium intake should be reduced (*6*).

Other key players essentially ignored the IOM study. The New York City Health Department, which leads a coalition of health organizations in a partnership with industry to reduce sodium in restaurant

Johns DM, Bayer R, Galea S. Controversial salt report peppered with uncertainty. [letter]. Science. 2013;341: 1063. PMID: 24009376. doi: 10.1126/science.341.6150.1063.

Why?

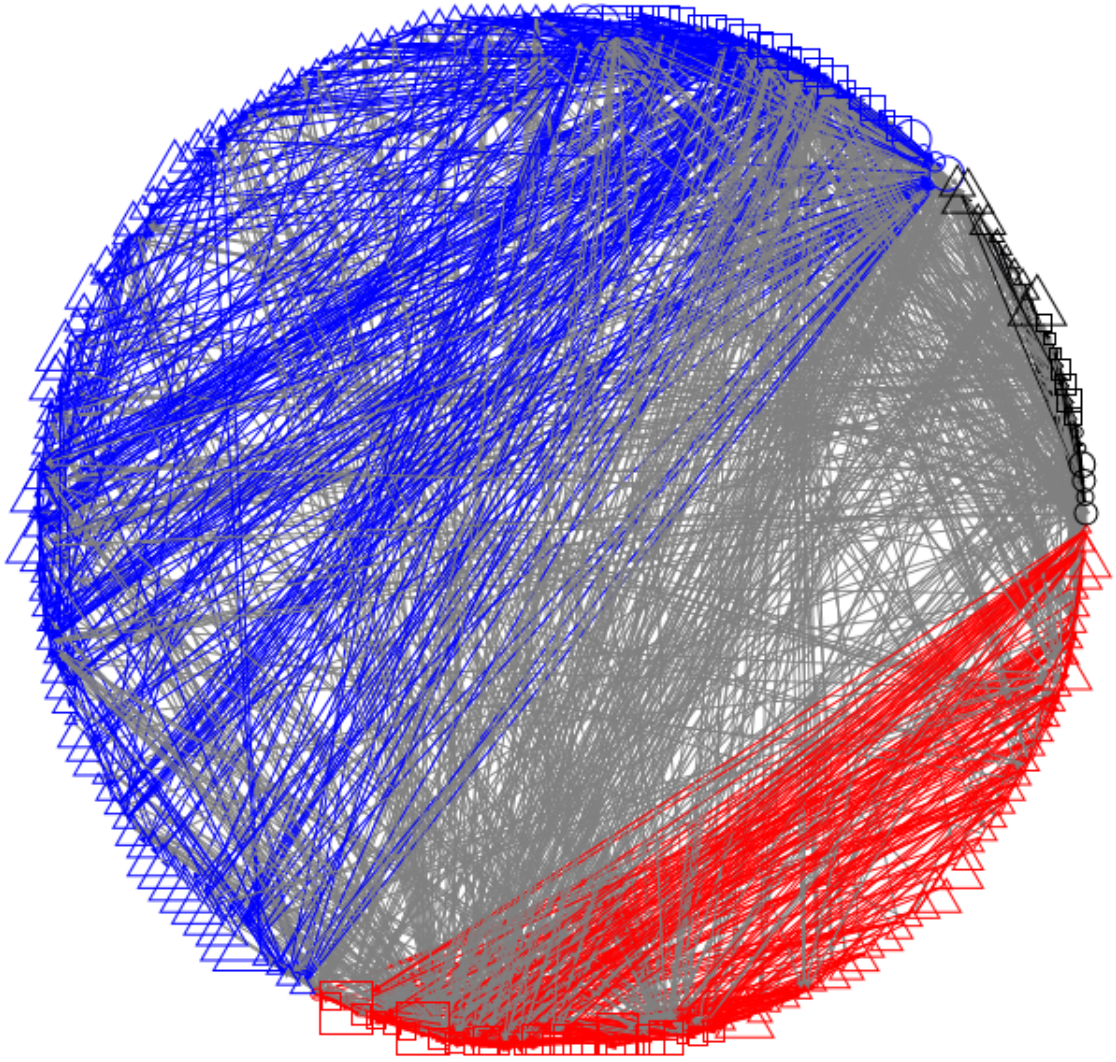
Patterns of clustering of citations between publications

		Citing articles			
		Pro	Con	NC	Total
Cited articles	Pro	381	146	65	592
	Con	275	235	51	561
	No conclusion	105	39	48	192
	Total	761	420	164	1345

Supportive citation likelihood ratio (381/761)/(146/420) = 1.44

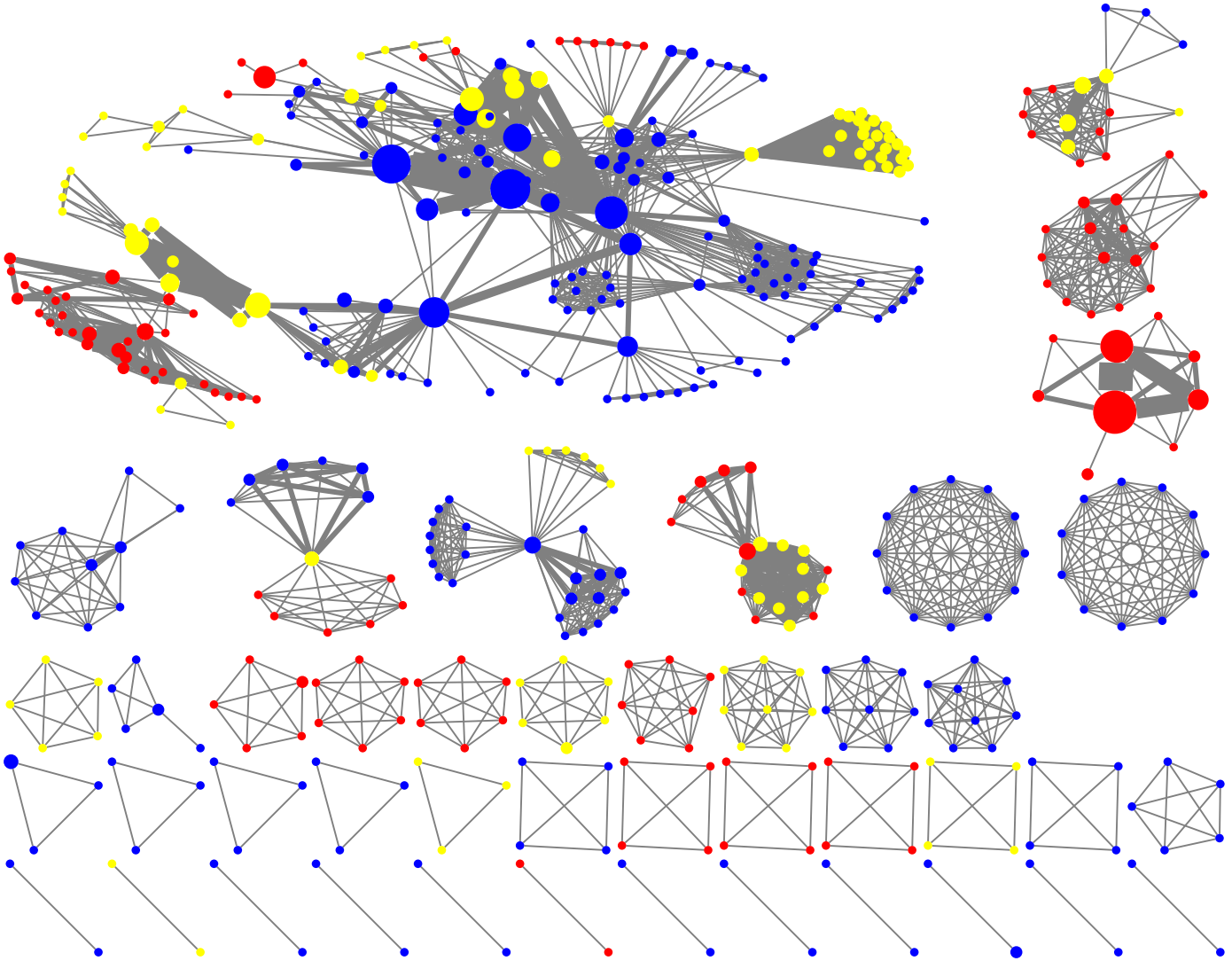
Contradictory citation likelihood ratio (275/761)/(235/420) = 0.65

Patterns of clustering of citations between publications



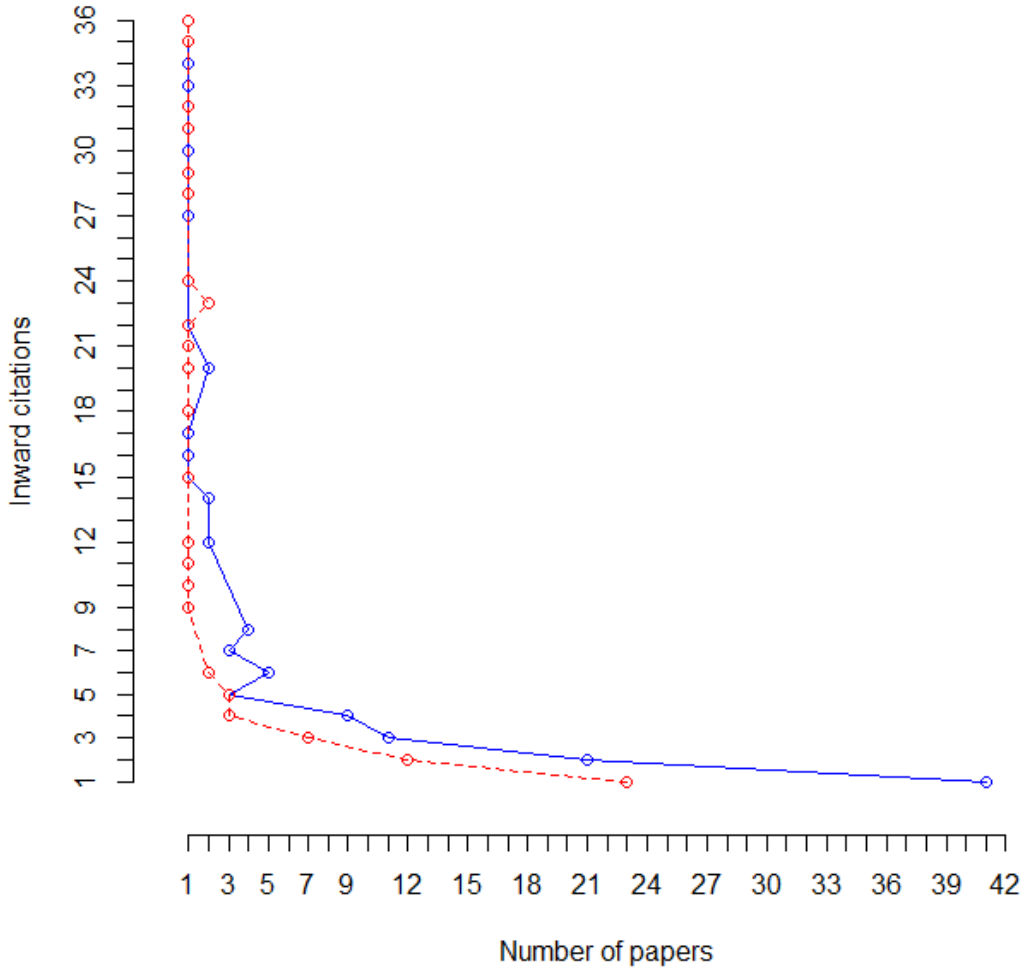
Blue is supportive of hypothesis, red is against, black is indeterminate; gray is cross-category citation

Patterns of clustering of co-authors between publications

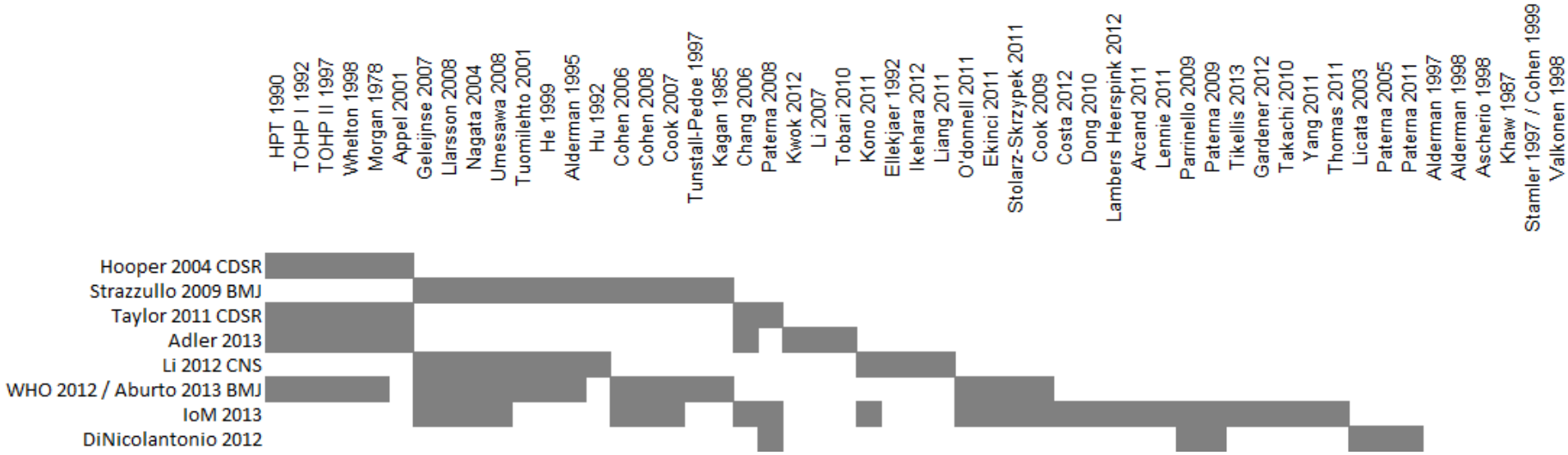


Authors with at least two articles in the field, and articles with at least two citations; density of line thickness refers to number of articles. Blue is supportive of hypothesis, red is against, black is indeterminate

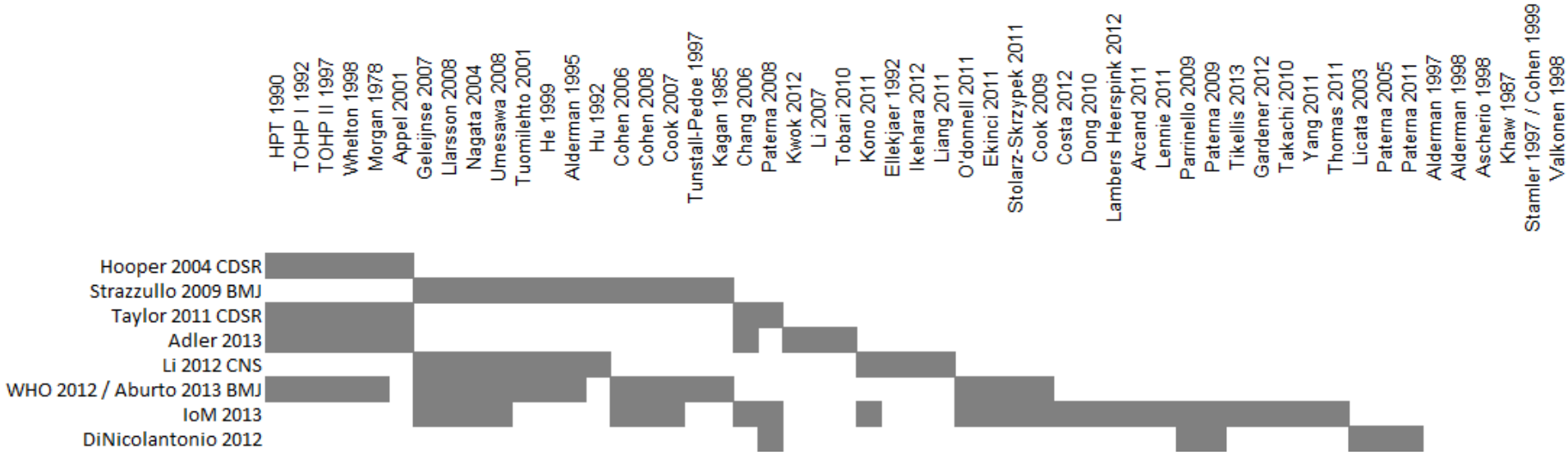
A few papers are cited quite a bit; most papers are not cited much at all



Patterns of uncertainty in systematic reviews



Patterns of uncertainty in systematic reviews



On average, there is only 27% chance that if a primary study is selected by a systematic review, a further review would also select it

An epidemiology of consequence should

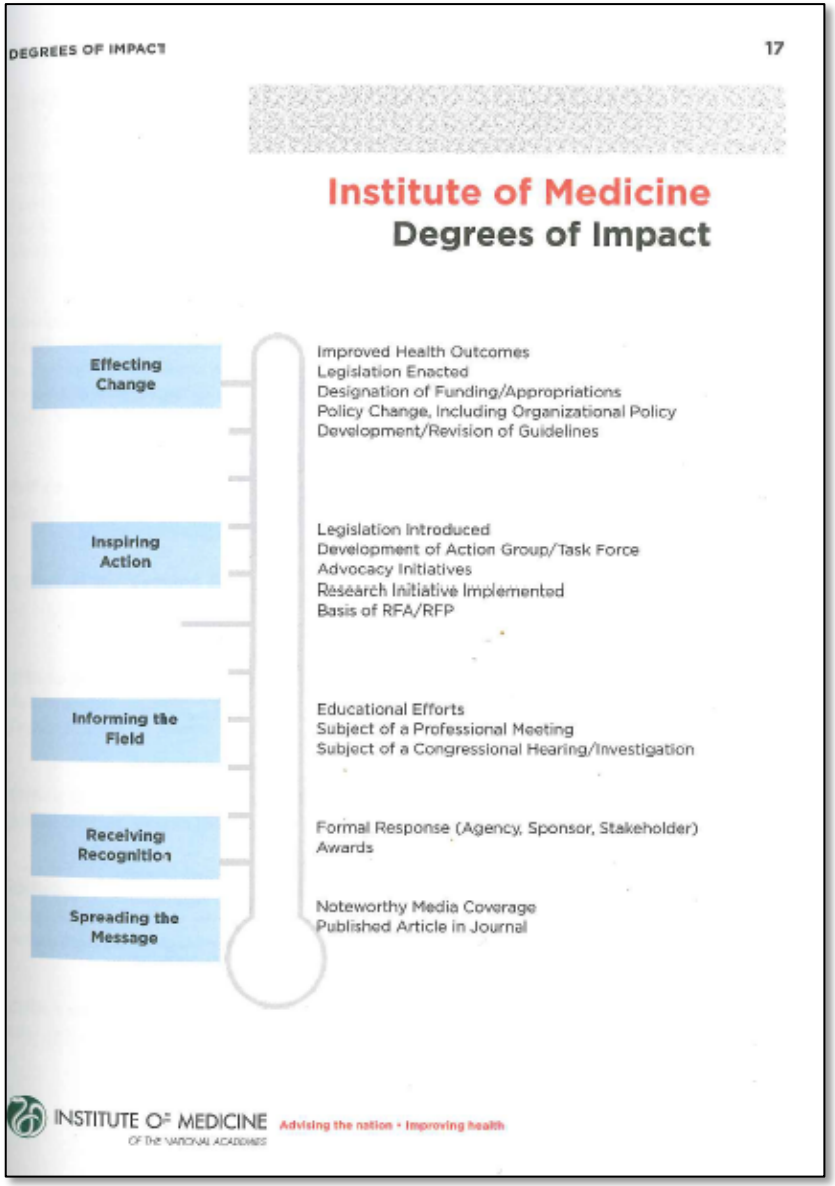
a. Focus on what matters most

b. Critically interrogate what we think we know

c. Engage in translating the science

d. Teach epidemiology differently

c. Engage in translating the science



Institute of Medicine
Degrees of Impact

Effecting Change
- Improved Health Outcomes
- Legislation Enacted
- Designation of Funding/Appropriations
- Policy Change, Including Organizational Policy Development/Revision of Guidelines

Inspiring Action
- Legislation Introduced
- Development of Action Group/Task Force
- Advocacy Initiatives
- Research Initiative Implemented
- Basis of RFA/RFP

Informing the Field
- Educational Efforts
- Subject of a Professional Meeting
- Subject of a Congressional Hearing/Investigation

Receiving Recognition
- Formal Response (Agency, Sponsor, Stakeholder)
- Awards

Spreading the Message
- Noteworthy Media Coverage
- Published Article in Journal

INSTITUTE OF MEDICINE
OF THE NATIONAL ACADEMIES
Advising the nation • Improving health

c. Engage in translating the science

Median number of citations for papers indexed in ISI: 0

http://physics.bu.edu/~redner/projects/citation/isi.html
http://www.dti.gov.uk/files/file27330.pdf

c. Engage in translating the science

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Crime and Punishment and Obama

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A Second Front in the Privacy Wars

PAUL KRUGMAN
Health Care Horror Hooey

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SundayReview | OP-ED COLUMNIST

Professors, We Need You!

FEB. 15, 2014

Nicholas Kristof

SOME of the smartest thinkers on problems at home and around the world are university professors, but most of them just don't matter in today's great debates.

The most stinging dismissal of a point is to say: "That's academic." In other words, to be a scholar is, often, to be irrelevant.

One reason is the anti-intellectualism in American life, the kind that led Rick Santorum to scold President Obama as "a snob" for wanting more kids to go to college, or that led congressional Republicans to denounce spending on social science research. Yet it's not just that America has marginalized some of its sharpest minds. They have also marginalized themselves.

"All the disciplines have become more and more specialized and more and more quantitative, making them less and less accessible to the general public," notes Anne-Marie Slaughter, a former dean of the Woodrow Wilson School at Princeton and now the president of the New America Foundation.

c. Engage in translating the science

"[Our task] is not to tell people what they should do. That is a matter for societies and their individual members to decide. [Rather, our task is] to analyze the options, so that such important choices can be based on a clearer understanding of the issues"

Rose G. The strategy of preventive medicine. Oxford University Press, 1991. p. 6.

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Orange
N. Arlington
Kearny
Harrison
Newark
Irvington
Elizabeth
Linden
Rahway
Cateret
Teterboro Airport (TEB)
Ft Lee
W. New York
Union City
Hoboken
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Bayonne
St George
Port Wadsworth
Great Kills
Staten Island
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New York
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Yankee Stadium
NY Botanical Garden
Hunts Point
Rikers Island
East River
LaGuardia Airport (LGA)
Astoria
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Great Neck
Port Washinton
Long Island Sound
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City Is.
Hart Is.
Eastchester Bay
Manchester Bay
Little Neck Bay
Northern Blvd
Elmont
Valley Stream
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Lawrence
Long Beach
Rockaway
Breezy Point
Jamaica Bay
Ozone Park
Bushwick
East New York
Brooklyn
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Red Hook
Governor's Island
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Times Sq
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Brighton Beach
Astroland
Aquarium
Newark Int'l Airport (EWR)
Cathedral of the Sacred Heart
Upper New York Bay
Lower New York Bay
Gowanus Bay
Raritian Bay
Gr. Kills Harbour
Newark Bay
Hudson River
New Jersey
New York
Verrazano Narrows Br (Toll)
Holland Tunnel
Lincoln Tunnel
Queensboro Br
Williamsburg Br
Pulaski Skyway
Newark Bay Br (Toll)
Bayonne Br
Kill Van Kull
Arthur Kill
Fresh Kills
Garden State Pkwy
New Jersey Turnpike
W. Jersey Turnpike
Brooklyn Queens Expressway
Interborough Central Pkwy
Utopia Expressway
Springfield Blvd
Southern St. Pkwy
Belt Parkway
Shore Pkwy
Bay Pkwy
Ocean Pkwy
Kings Hwy
Eastern Pkwy
Atlantic Av
Myrtle Av
Broadway
Jamaica Ave
Union Tpk
Jericho Tpk
Greenpoint Av
Houston
14th Av
8th Av
Park Av
3rd Av
Victory Blvd
Forest Av
Hylan


Metropolitan Transportation Authority
MTA New York City Subway
THE BRONX
QUEENS
BROOKLYN
MANHATTAN
Long Island Sound
Hudson River
East River
Jamaica Bay
Key
Part-time line extension
Local service only
All trains stop (local and express service)
Free subway transfer
Free out-of-system subway transfer (excluding single ride ticket)
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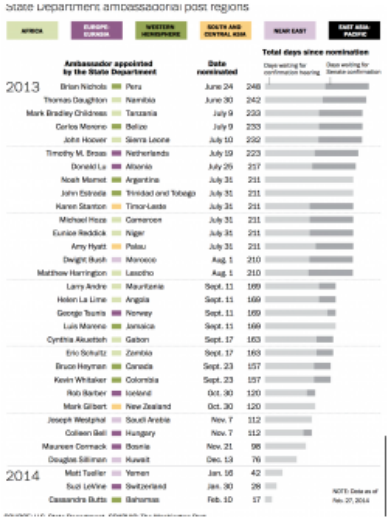

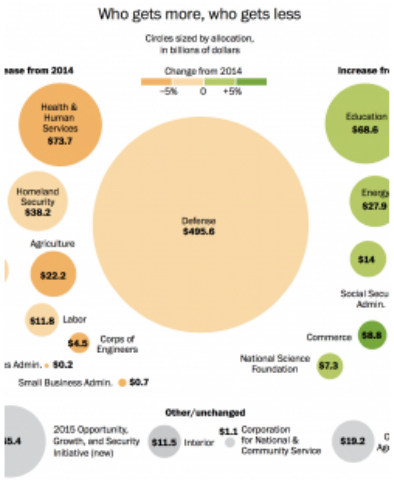

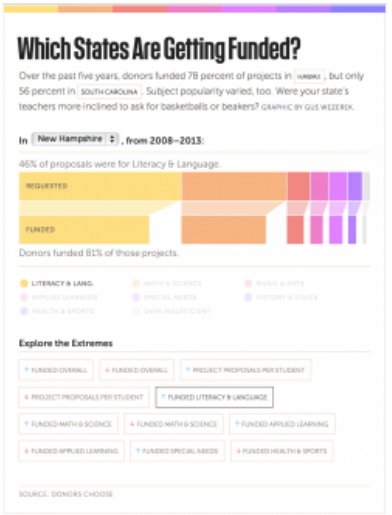

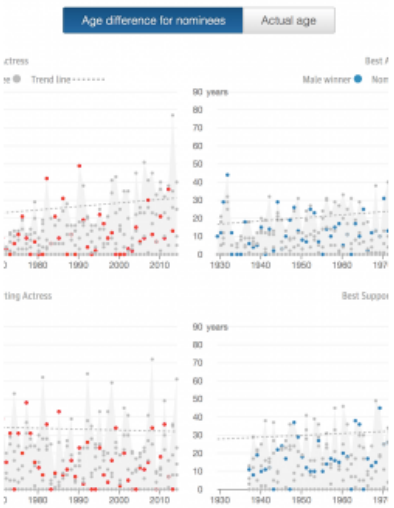

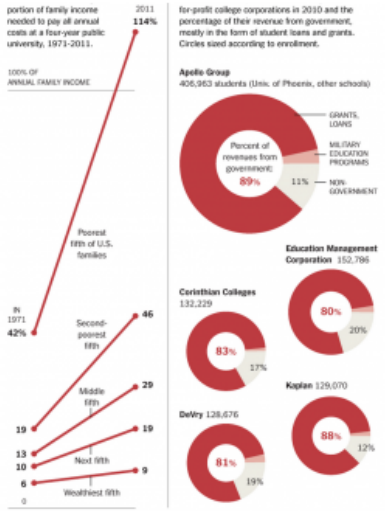

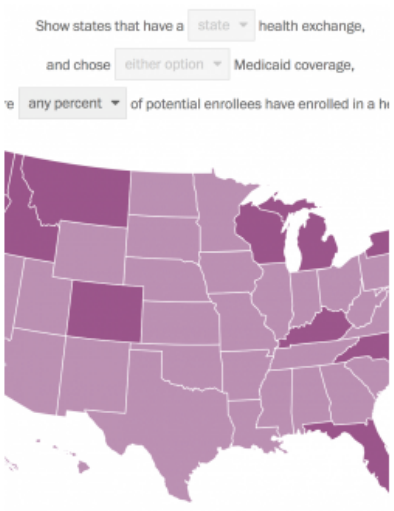

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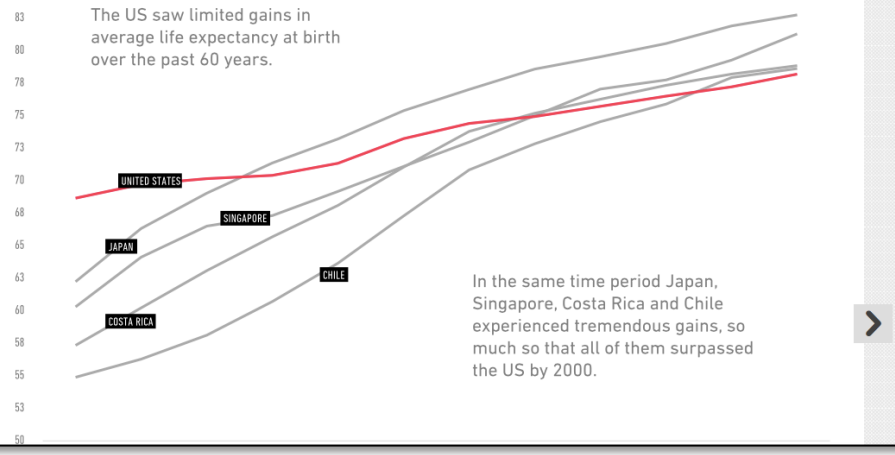

An epidemiology of consequence should

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d. Teach epidemiology differently

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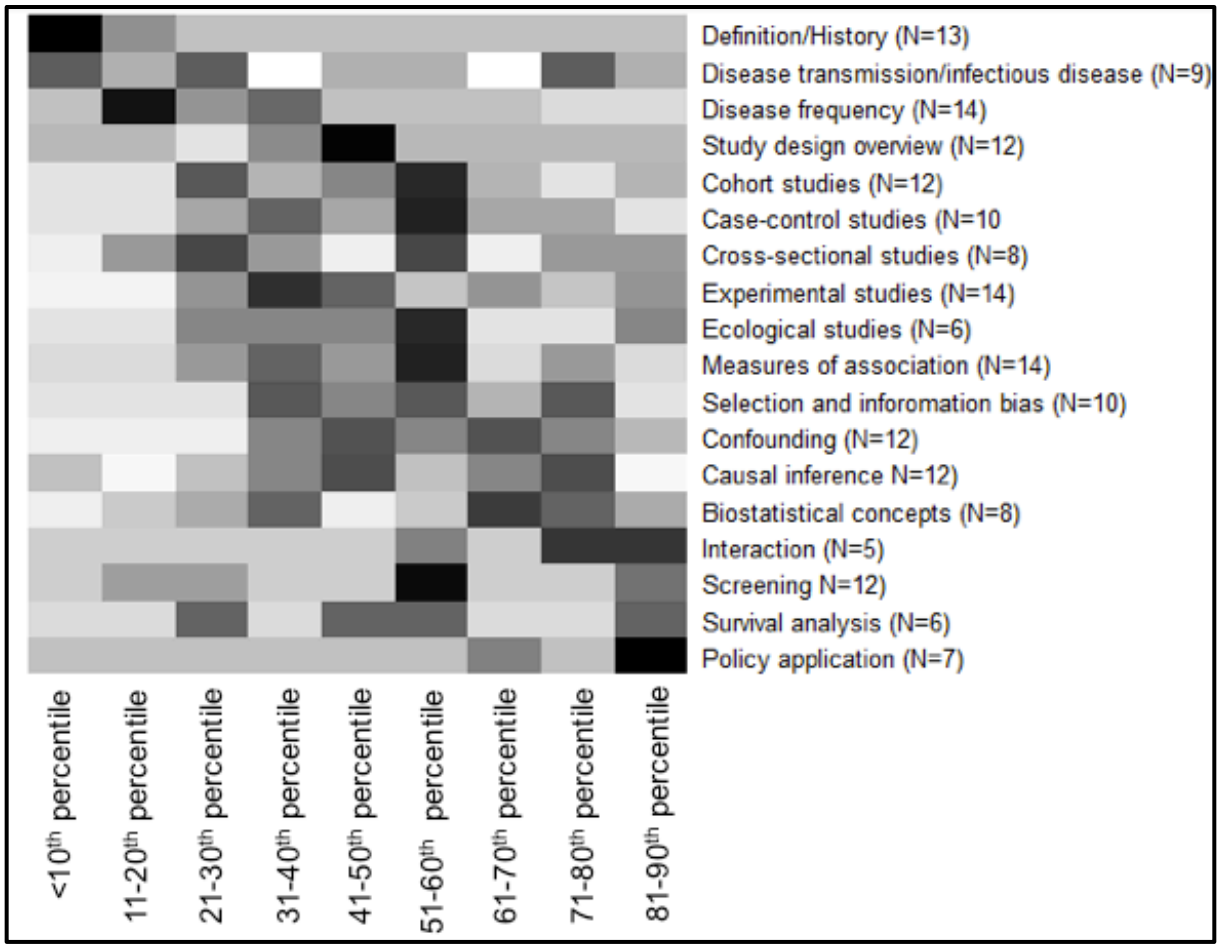
Our current teaching in the field is based on an accepted canon, taught similarly throughout epidemiology books



*Keyes KM, Galea S. Current practices in teaching introductory epidemiology: how we got here, where to go. American Journal of Epidemiology. In press.
Textbooks included: Aschengrau & Seage, Bhopal, Bonita et al., Carneiro & Howard, Carr et al., Friis, Gertman, Gordis, Kestenbaum, Merrill, Rothman, Saracci, Webb, Wassertheil-Smoller.

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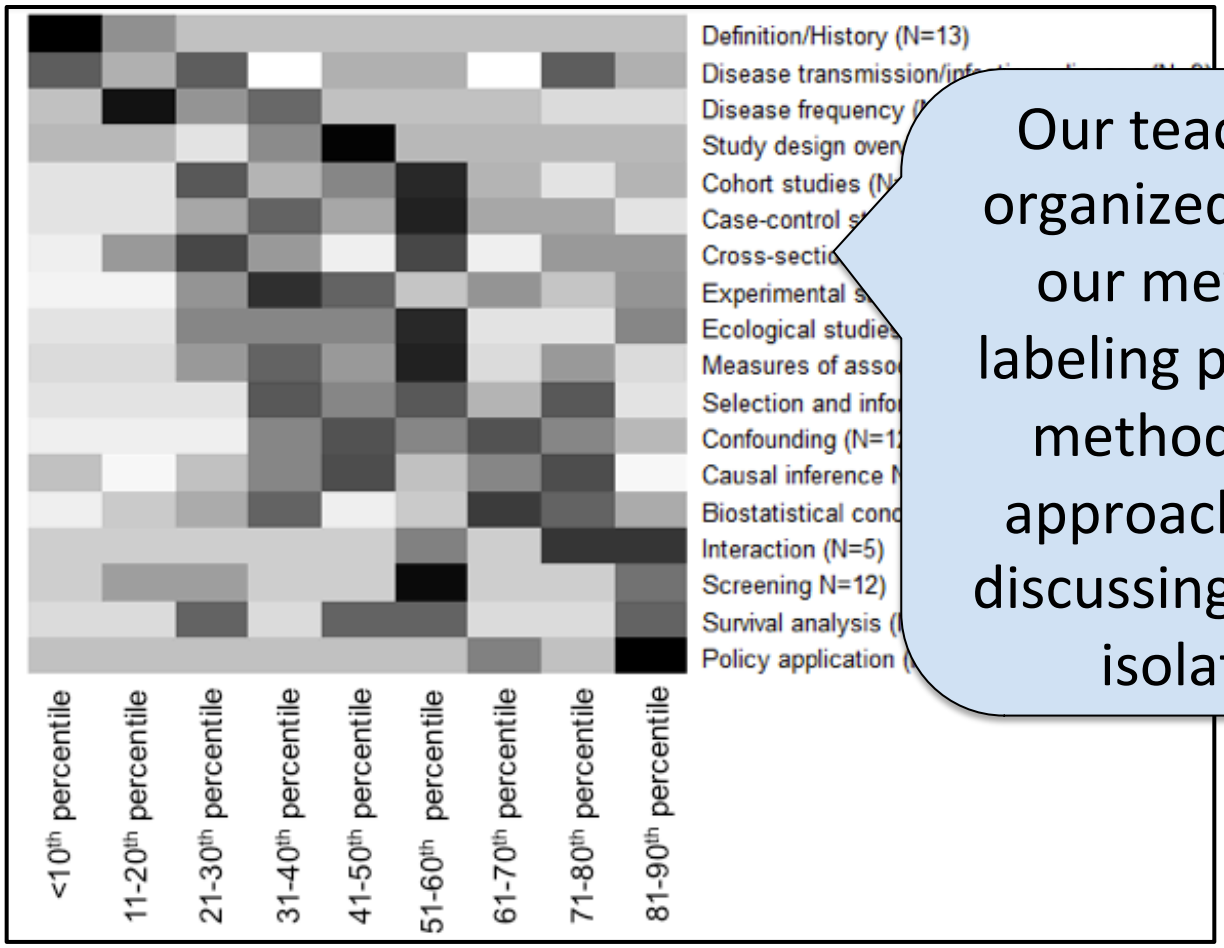

Our teaching is organized around our methods, labeling particular methodologic approaches and discussing them in isolation

*Keyes KM, Galea S. Current practices in teaching introductory epidemiology: how we got here, where to go. Under review.
Textbooks included: Aschengrau & Seage, Bhopal, Bonita et al., Carneiro & Howard, Carr et al., Friis, Gertman, Gordis, Kestenbaum, Merrill, Rothman, Saracci, Webb, Wassertheil-Smoller.

d. Teach epidemiology differently

What if we taught instead from first principles, informed by an epidemiology of consequence?

First principles of epidemiology?

1. Define the population of interest
2. Conceptualize and create measures of exposures and health indicators
3. Take a sample of the population
4. Estimate measures of association between exposures and health indicators of interest
5. Rigorously evaluate whether the association observed suggests a causal association
6. Assess the evidence for causes working together
7. Assess the extent to which the result matters—is externally valid—to other populations

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It is all about populations and potential ways to improve them; this is the first underlying principle

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The science matters if we are able to influence populations

*Keyes KM, Galea S. Current practices in teaching introductory epidemiology: how we got here, where to go. American Journal of Epidemiology. In press.

1. Motivations
2. A disciplinary definition
3. A call for recalibration
4. The consequences of consequentialism
5. **Controversial consequential thoughts**
6. Other consequences, not discussed

“Although biological principles seem to be vastly more varied than physics, and more dependent on locally varying modifying influences, the ultimate aim of biological research on humans or other species, is like that of physics, to be able to make general statements about nature.”

Rothman KJ, Gallacher JEJ, Hatch EE. Why representativeness should be avoided. International Journal of Epidemiology 2013;42:1014–1015

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But *can we* make "general statements about nature" in quantitative population health science?

1. Motivations
2. A disciplinary definition
3. A call for recalibration
4. The consequences of consequentialism
5. Controversial consequential thoughts
6. **Other consequences, not discussed**

An epidemiology of consequence should also

a. Use any method necessary

b. Cast a *global* population conceptual net

c. Engage issues of equity v. efficiency

Thank you

Kerry Keyes

Many collaborators

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