New directions in population health science: from risks to consequences

Sandro Galea

Boston University School of Public Health
1. What we do
### Year 1

**N=40**

![Sample Exposed and Unexposed](image)

<table>
<thead>
<tr>
<th></th>
<th>Diseased</th>
<th>Not Diseased</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exposed</td>
<td>8</td>
<td>2</td>
<td>10</td>
</tr>
<tr>
<td>Unexposed</td>
<td>5</td>
<td>5</td>
<td>10</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>13</td>
<td>7</td>
<td>20</td>
</tr>
</tbody>
</table>
2. What this leads to
a. The state of the world’s health
The world, actual size
The world, by preventable deaths

The world, by unhealthy life expectancy
Average spending on health per capita ($US PPP)

Note: $US PPP = purchasing power parity.
FIGURE 1-1  Mortality from noncommunicable diseases in 17 peer countries, 2008.
SOURCE: Data from World Health Organization (2011a, Table 3).
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).

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).
Inequality in life expectancy widens for women

Wealthier women can expect to live longer than their parents did, while life expectancy for poor women may have declined.

Life expectancy for 50-year-olds in a given year, by quintile of income over the previous 10 years

Source: National Academies of Science, Engineering and Medicine

b. A paradoxical approach to this state of the world
Personalized medicine: inevitable

The Path to Personalized Medicine
Margaret A. Hamburg, M.D., and Francis S. Collins, M.D., Ph.D.

Major investments in basic science have created... the NIH and the FDA will invest
in advancing translational and

Journal of Diabetes Science and Technology
Volume 3, Issue 4, July 2009
© Diabetes Technology Society

The Case for Personalized Medicine
Edward Abrahams, Ph.D.¹ and Mike Silver, Ph.D.²
Proportion of papers in PubMed, 2002 - 2012

Among NIH funding for the current fiscal year, only 0.4% was awarded to projects with the terms "population" or "public" in the title.

NIH RePORTER. Current projects from FY 2013-2014 for which funding data is available. <http://projectreporter.nih.gov/reporter_ChartResults.cfm?icde=22550343>
Proportion of NIH funding awarded to projects with the terms “genetic” or “genetics” in the title, abstract or terms

Proportion of NIH funding awarded to projects with the terms “population” or “public” in the title, abstract, or terms

Expectations that communicating DNA based risk estimates changes behaviour is not supported by existing evidence

**ABSTRACT**

**OBJECTIVE**
To assess the impact of communicating DNA based disease risk estimates on risk-reducing health behaviours and motivation to engage in such behaviours.

**DESIGN**
Systematic review with meta-analysis, using Cochrane methods.

**DATA SOURCES**
Medline, Embase, PsycINFO, CINAHL, and the Cochrane Central Register of Controlled Trials up to 25 February 2015. Backward and forward citation searches were also conducted.

**WHAT IS ALREADY KNOWN ON THIS TOPIC**
Genetic testing is being increasingly used in a growing number of healthcare settings and in direct-to-consumer testing for a range of common complex disorders. There is an expectation that communicating DNA based disease risk estimates will motivate changes in key health behaviours, including smoking, diet, and physical activity.

**WHAT THIS STUDY ADDS**
The results of this updated systematic review with meta-analysis using Cochrane methods suggest that communicating DNA based disease risk estimates has little or no impact on risk-reducing health behaviour.

Existing evidence does not support expectations that such interventions could play a major role in motivating behaviour change to improve population health.
Comprehensive enumeration of sequence variation is necessary to identify functional alleles that provide important clues to disease pathophysiology, but large-scale sequencing does not support the idea that lower-frequency variants have a major role in predisposition to type 2 diabetes.
c. A somewhat shambling scientific approach
Figure 2. Number of PubMed-Indexed Articles Published Each Year Between 2005 and 2014 That Carry the Tag “Meta-analysis” for Type of Publication and Have Author Affiliations From China or From the United States (USA)
Figure 4. A Summary Overview of Currently Produced Meta-analyses

- Decent and clinically useful: 20%
- Flawed beyond repair: 3%
- Misleading, abandoned genetics: 13%
- Redundant and unnecessary: 27%
- Unpublished: 20%
- Decent, but not useful: 17%
Short report

What types of interventions generate inequalities? Evidence from systematic reviews

Theo Lorenc,1 Mark Petticrew,1 Vivian Welch,2 Peter Tugwell2

ABSTRACT

Background Some effective public health interventions may increase inequalities by disproportionately benefiting less disadvantaged groups (‘intervention-generated inequalities’ or IGIs). There is a need to understand which types of interventions are likely to produce IGIs, and which can reduce inequalities.

Methods We conducted a rapid review of systematic reviews to identify evidence on IGIs by socioeconomic status. We included any review of non-healthcare interventions in high-income countries presenting data on differential intervention effects on any health status or health behaviour outcome. Results were synthesised narratively.

Results The following intervention types show some evidence of increasing inequalities (IGIs) between socioeconomic status groups: media campaigns; and workplace smoking bans. However, for many intervention types, data on potential IGIs are lacking. By contrast, the following show some evidence of reducing health inequalities: structural workplace interventions; provision of resources; and fiscal interventions, such as tobacco pricing.

Conclusion Our findings are consistent with the idea that ‘downstream’ preventive interventions are more likely to increase health inequalities than ‘upstream’ interventions. More consistent reporting of differential intervention effectiveness is required to help build the evidence base on IGIs.

INTRODUCTION

A number of researchers have raised concerns about the possibility that public health interventions may increase inequalities in the population. This has been expressed as an ‘inverse prevention law’,1 analogous to the ‘inverse care law’ posited for medical care by Tudor Hart,2 stating that those in most need of benefitting from preventive interventions are least likely to receive them. That is, even where interventions are successful at improving health across the population, they may increase health inequalities. This can happen where an intervention is of greater benefit to advantaged (lower-risk) groups than to disadvantaged (higher-risk) groups. Such ‘intervention-generated inequalities’ (IGIs) may arise at a number of points in the implementation of an intervention, including intervention efficacy, service provision or access, uptake, and compliance.4 Conversely, some interventions may reduce inequalities, if they are of greater benefit to disadvantaged groups.

A number of intervention types have been investigated for the possibility of IGIs, and there is a substantial body of theoretical work and guidance on the kinds of interventions which are likely to reduce or increase inequalities.5–7 However, few studies have sought to bring together what is known about IGIs across the whole field of public health interventions. The aim of this paper is to provide an overview of evidence from systematic reviews in order to provide preliminary indications as to which types of interventions are more likely to produce IGIs, and which have the potential to reduce inequalities.

METHODS

The method used was a systematic review of reviews, with limited searching but systematic screening. Analogous methods have been widely used to produce ‘rapid reviews’: they aim to minimise selection bias, as in a full systematic review, but not to be fully comprehensive, and so cannot rule out publication bias.8,9 We searched MEDLINE using the string ‘(inequalities$ or inequalit$ or disparit$ or disadvantage$ or disadvantage$).tw.’ in conjunction with SIGN’s filter for systematic reviews (http://www.sign.ac.uk/methodology/filters.html#systematic), and searched the bibliography from a recent review of reviews on inequalities.10 We included systematic reviews which evaluated the effectiveness of any non-healthcare intervention in a high-income country on any health outcome, and which reported differences in intervention effectiveness between population groups, defined in terms of the PROGRESS-Plus framework.11,12 Full methods and the flow of literature are presented in the web-only appendix to this paper.

It should be noted that a differential intervention effect does not necessarily imply an IGI. Strictly speaking, we can identify an IGI only where we know that an intervention has created a health inequality where none existed at baseline, or widened an existing inequality. To confirm this, we would need detailed information on the study sample at baseline, showing that more disadvantaged groups on the relevant demographic (PROGRESS-Plus) dimension are worse off, or at least no better off, with respect to the relevant health variable(s). However, such baseline data are often not recoverable from secondary or tertiary research findings. For this reason, our analysis focuses on differences in intervention effect between groups of lower and higher socioeconomic status (SES), broadly defined to include measures such as income, occupational status, employment status, housing tenure or level of education), rather than on other PROGRESS-Plus factors, such as gender or ethnicity. This is because, while the existence and direction of an inequality gradient by SES for most health behaviour and health status variables is reasonably well established, and can in...
What types of interventions generate inequalities? Evidence from systematic reviews

Theo Lorenc,∗ Mark Petticrew,† Vivian Welch,‡ Peter Tugwell§

ABSTRACT

Background Some effective public health interventions may increase inequalities by disproportionately benefiting less disadvantaged groups (‘intervention-generated inequalities’ or IGIs). There is a need to understand which types of interventions are likely to produce IGIs, and which can reduce inequalities.

Methods We conducted a rapid overview of systematic reviews, with limited searching but systematic screening. Analogous methods have been widely used to produce rapid reviews. They aim to minimise selection bias, as in a full systematic review, but not to be fully comprehensive, and so cannot rule out publication bias.

Results The following intervention types show some evidence of reducing inequalities: structural workplace interventions; health interventions. The aim of this paper is to provide an overview of evidence from systematic reviews in order to provide preliminary indications as to which types of interventions are most likely to reduce or increase inequalities.5–7 However, few studies have sought to bring together what is known about IGIs across the whole field of public health interventions. The aim of this paper is to provide an overview of evidence from systematic reviews in order to provide preliminary indications as to which types of interventions are most likely to reduce or increase inequalities.5–7 However, few studies have sought to bring together what is known about IGIs across the whole field of public health interventions. The aim of this paper is to provide an overview of evidence from systematic reviews in order to provide preliminary indications as to which types of interventions are most likely to reduce or increase inequalities.

Conclusions A number of researchers have raised concerns about the possibility of IGIs, and there is evidence of increasing inequalities (IGIs) between groups of lower and higher socioeconomic status (SES) for most health behaviour and health status variables. However, such baseline data are often not recoverable from secondary or tertiary research findings. For this reason, our analysis focuses on differences in intervention effect between groups of lower and higher socioeconomic status (SES), broadly defined to include measures such as income, occupational status, employment status, housing tenure or level of education), rather than on other PROGRESS-Plus factors, such as gender or ethnicity. This is because, while the existence and direction of an inequality gradient by SES for most health behaviour and health status variables is reasonably well established, and can in
d. Loss of confidence in what we find/promote
Figure 3: New England Journal of Panic-Inducing Gobbledygook.
WITH A PROPER DIET, MODERATE BUT CONSISTENT EXERCISE, AND THE DEVELOPMENT OF A HEALTHY MIND-SET, I SHOULD BE ABLE TO FIT IN FINE.
3. We know there are problems with this approach
“We need to foster...research, not only by improving our methodology and sharing our scientific experience, but by helping to convince the...public and its legislators that prevention is far more important than treatment...and that the application of our findings to improve the health of the public must become the highest priority for health policy...”
If mouse models are like looking for your keys under the street lamp, big data is like looking all over the world for your keys because you can— even if you don’t know what they look like or where you might have dropped them or whether they actually fit your lock.
The dangerous threat to science comes from areas of research where the stakes are high but the validity of the science cannot be determined...In these cases, science delivers partial truths, any one of which can advance the career of a researcher and attract a constituency of believers among scientists, political interest groups, and members of the public alike.
4. An alternative approach, a focus on consequences
“…academic epidemiology now spends most of its time concerned with identifying the causes and distributions of disease in human populations and far less of its time and imagination asking how we might improve population health, what might happen if a particular approach were taken to try to do so, where and when it may be appropriate to attempt inflections to the course of the health of populations, and whether our efforts to elucidate particular causes is usefully guiding our way to population health improvement.”
Public health, PubMed
Population health science/public health, PubMed
What is population health science?
Population health science is the study of the conditions that shape distributions of health within and across populations, and of the mechanisms through which these conditions manifest as the health of individuals.
1. Population health manifests as a continuum.
2. The causes of differences in health across populations are not necessarily an aggregate of the causes of differences in health within populations.
3. Large benefits to population health may not improve the lives of all individuals.
4. The causes of population health are multilevel, accumulate throughout the life course, and are embedded in dynamic interpersonal relationships.
5. Small changes in ubiquitous causes may result in more substantial change in the health of populations than larger changes in rarer causes.
6. The magnitude of an effect of exposure on disease is dependent on the prevalence of the factors that interact with that exposure.
7. Prevention of disease often yields a greater return on investment than curing disease after it has started.
8. Efforts to improve overall population health may be a disadvantage to some groups; whether equity or efficiency is preferable is a matter of values.
9. We can predict health in populations with much more certainty than we can predict health in individuals.
Principle 1. Population health manifests as a continuum
Divertente campagna per la prevenzione dell'obesità.
Figure 1. Distribution of BMI in two populations illustrating health as a continuum in the population.

Panel A
Figure 3 Percentage distribution of serum cholesterol levels (mg/dl) in men aged 50–62 who did or did not subsequently develop coronary heart disease (Framingham Study).
Figure 3 Percentage distribution of serum cholesterol levels (mg/dl) in men aged 50–62 who did or did not subsequently develop coronary heart disease (Framingham Study).
Principle 2. The causes of differences in health across populations are not necessarily an aggregate of the causes of differences in health within populations.
Figure 1. Conceptualizing causation at the individual level: 20 students in a classroom

Amy  Stephanie  Jane  Joe  Stephen
James  Matthew  Manuel  Abdul  Angelina
Sarah  Ming  Leila  Peter  Kim
John  Lucy  Naomi  Alex  Jorge
Why does Joe have asthma but not Leila?
Figure 2. Conceptualizing causation at the population level: comparing two classrooms
Why there are twice as many cases of asthma in Classroom 2 compared with Classroom 1?
Principle 3. Large benefits to population health may not improve the lives of all individuals
What are the causes of high blood pressure among civil servants in London?

Figure 1. Hypothetical data on the systolic blood pressure of London civil servants
Figure 2: Hypothetical data on the systolic blood pressure of Kenyan nomads
How do we best lower LDL cholesterol in the towns of Chadwick and Farr?
27.5% of population has high LDL
Table 1. The relation between potato chip eating and cholesterol in Chadwick

<table>
<thead>
<tr>
<th></th>
<th>High LDL cholesterol</th>
<th>Low LDL cholesterol</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chip eater</td>
<td>150</td>
<td>350</td>
<td>500</td>
</tr>
<tr>
<td>Non-chip eater</td>
<td>125</td>
<td>375</td>
<td>500</td>
</tr>
<tr>
<td>Total</td>
<td>275</td>
<td>725</td>
<td>1000</td>
</tr>
</tbody>
</table>

$$\text{Risk difference} = \left( \frac{150}{500} \right) - \left( \frac{125}{500} \right) = 0.05$$

i.e., 5 additional cases of high LDL cholesterol for every 100 additional chip eaters
7.5% of population has high LDL
Table 2. The relation between potato chip eating and cholesterol in Farr

<table>
<thead>
<tr>
<th></th>
<th>High LDL cholesterol</th>
<th>Low LDL cholesterol</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chip eater</td>
<td>50</td>
<td>450</td>
<td>500</td>
</tr>
<tr>
<td>Non-chip eater</td>
<td>25</td>
<td>475</td>
<td>500</td>
</tr>
<tr>
<td>Total</td>
<td>75</td>
<td>925</td>
<td>1000</td>
</tr>
</tbody>
</table>

Risk difference = \( \frac{50}{500} - \frac{25}{500} = 0.05 \)

i.e., also 5 additional cases of high LDL cholesterol for every 100 additional chip eaters
In both populations the risk of high LDL linked to potato chip eating is the same. But, in Population Chadwick, 27.5% of the population has high LDL cholesterol, compared with 7.5% of the population in Population Farr. What is driving the difference?
Table 3. The relation between childhood poverty and cholesterol in both Chadwick (all exposed to childhood poverty) and Farr (all unexposed to childhood poverty)

<table>
<thead>
<tr>
<th></th>
<th>High LDL cholesterol</th>
<th>Low LDL cholesterol</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Childhood poverty</td>
<td>275</td>
<td>725</td>
<td>1000</td>
</tr>
<tr>
<td>No poverty</td>
<td>75</td>
<td>925</td>
<td>1000</td>
</tr>
<tr>
<td>Total</td>
<td>350</td>
<td>1650</td>
<td>2000</td>
</tr>
</tbody>
</table>

Risk difference = \( \frac{275}{1000} - \frac{75}{1000} = 0.20 \)

i.e., there is an excess of 20 cases of high LDL cholesterol for every 100 adults exposed to childhood poverty across these populations.
Figure 7. High-risk and population approaches to disease prevention

High-Risk Approach

Percentage of population affected

Disease risk

Intervention

Population Approach

Percentage of population affected

Disease risk

= Pre-intervention

= Post-intervention
Principle 5. Small changes in ubiquitous causes may result in more substantial change in the health of populations than larger changes in rarer causes
Figure 1. A metaphor for ubiquity

The goldfish are surrounded by water and everything they do is influenced by the quality of the water in which they live; therefore, water is a ubiquitous factor influencing the fish and needs to be taken into consideration every time we may want to improve the lives of the fish.
For Decades, the Richest Pulled Away...

Share of income for the:

- **Top 1%**
  - In 2007, 24% of income went to the 1.5 million families who earned more than $400,000.

- **Top 1/10,000th**

Share of income for the **bottom 90%**

- In 2007, 50% of income went to the 135 million families who earned less than $110,000.
The prevalence of IV drug use is 1.96% and IV drug users have 6.25 times the risk of mortality compared with non-IV drug users. For every 100 IV drug users, we would expect 4.2 additional deaths.

<table>
<thead>
<tr>
<th></th>
<th># died</th>
<th># alive</th>
<th>total</th>
</tr>
</thead>
<tbody>
<tr>
<td>IV drug user</td>
<td>50</td>
<td>950</td>
<td>1000</td>
</tr>
<tr>
<td>Non-IV drug user</td>
<td>400</td>
<td>49600</td>
<td>50000</td>
</tr>
<tr>
<td>Total</td>
<td>450</td>
<td>50550</td>
<td>51000</td>
</tr>
</tbody>
</table>

\[
RR = \frac{\frac{50}{1000}}{\frac{400}{50,000}} = 6.25
\]

\[
RD = \left(\frac{50}{1000}\right) - \left(\frac{400}{50,000}\right) = 0.042
\]
Suppose we reduce income inequality by 25%, keeping prevalence of IV drug use the same, but reducing excess mortality in all groups.

<table>
<thead>
<tr>
<th></th>
<th># died</th>
<th># alive</th>
<th>total</th>
</tr>
</thead>
<tbody>
<tr>
<td>IV drug user</td>
<td>40</td>
<td>960</td>
<td>1000</td>
</tr>
<tr>
<td>Non-IV drug user</td>
<td>320</td>
<td>49680</td>
<td>50000</td>
</tr>
<tr>
<td></td>
<td>360</td>
<td>50640</td>
<td>51000</td>
</tr>
</tbody>
</table>
Therefore, those in the unequal society had 1.25 times the risk of death, and we would expect 1.8 deaths per 1,000 persons exposed to the unequal society.
In the hypothetical income inequality intervention, we have “saved” 90 lives (450-360=90). In our IV drug use example, we found that we would “save” an additional 4.2 times as many lives per 100 users; with 1000 total users in our population, we would estimate that we save a maximum of 42 lives even if all IV drug users stopped using.
FIGURE 1. Motor-vehicle–related deaths per million vehicle miles traveled (VMT) and annual VMT, by year — United States, 1925–1997
Crack Babies: The Worst Threat Is Mom Herself

By Douglas J. Besharov

LAST WEEK in this city, Greater Southeast Community Hospital released a 7-week-old baby to her homeless, drug-addicted mother even though the child was at severe risk of pulmonary arrest. The hospital's explanation: "Because [the mother] demanded that the baby be released."

The hospital provided the mother with an apnea monitor to warn her if the baby stopped breathing while asleep, and trained her in CPR. But on the very first night, the mother went out drinking and left the child at a friend's house—without the monitor. Within seven hours, the baby was dead. Like Dooney Waters, the 6-year-old living in his mother's drug den, whose shocking story was reported in The Washington Post last week, this child was all but abandoned by relatives.

Children of Cocaine
(By Charles Krauthammer)
<table>
<thead>
<tr>
<th>Predictor for Peabody Picture Vocabulary Test score</th>
<th>Coefficient</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gestational cocaine exposure</td>
<td>-2.89</td>
<td>0.26</td>
</tr>
<tr>
<td>Assessment no.</td>
<td>2.72</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Gestational cocaine exposure x assessment no.</td>
<td>0.58</td>
<td>0.51</td>
</tr>
<tr>
<td>Age at 1st assessment</td>
<td>-0.36</td>
<td>0.76</td>
</tr>
<tr>
<td>Female gender</td>
<td>-4.93</td>
<td>0.058</td>
</tr>
<tr>
<td>Parental nurturance</td>
<td>-0.31</td>
<td>0.89</td>
</tr>
<tr>
<td>Environmental stimulation</td>
<td>5.91</td>
<td>0.039</td>
</tr>
<tr>
<td>Caregiver BDI-II depression score</td>
<td>0.03</td>
<td>0.84</td>
</tr>
</tbody>
</table>

Principle 6. The magnitude of an effect of exposure on disease is dependent on the prevalence of the factors that interact with that exposure.
How much of our cognitive ability is determined by our genes?
= GE+
= ENV+
Scenario 1

= GE+  = CA+  = ENV+
Scenario 1

= GE+  = CA+  = ENV+
Scenario 1

RR (CA+|GE+) = 334
PARP (CA+|GE+) = 1

= GE+  = CA+  = ENV+
Scenario 2

= GE+  = CA+  = ENV+
Scenario 2

= GE+  = CA+  = ENV+
Scenario 2

RR (CA+|GE+) = 1.7
PARP (CA+|GE+) = 0.4

= GE+  = CA+  = ENV+
Is this all theoretical?
Proportion of total Full-Scale IQ variance accounted for by A, C, and E plotted as a function of observed socioeconomic status (SES).

Turkheimer E et al. Psychological Science 2003;14:623-628
Principle 9. We can predict health in populations with much more certainty than we can predict health in individuals
Table 1. Childhood poverty as a risk factor for adult heart disease by age 65 in a population of 1,000 individuals, Population 1

<table>
<thead>
<tr>
<th></th>
<th>Heart disease</th>
<th>No heart disease</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Childhood poverty</td>
<td>25</td>
<td>175</td>
<td>200</td>
</tr>
<tr>
<td>No childhood poverty</td>
<td>50</td>
<td>750</td>
<td>800</td>
</tr>
<tr>
<td></td>
<td>100</td>
<td>900</td>
<td>1000</td>
</tr>
</tbody>
</table>

Risk ratio: \[ \frac{\frac{25}{200}}{\frac{50}{800}} = 2.0 \]

Positive predictive value = \( \frac{25}{200} = 0.125 \) or 12.5%

Negative predictive value = \( \frac{750}{800} = 0.9375 \) or 93.8%
Table 2. Childhood poverty as a risk factor for adult heart disease by age 65 in a population of 1,000 individuals, Population 2

<table>
<thead>
<tr>
<th>Childhood poverty</th>
<th>Heart disease</th>
<th>No heart disease</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Childhood poverty</td>
<td>100</td>
<td>100</td>
<td>200</td>
</tr>
<tr>
<td>No childhood poverty</td>
<td>200</td>
<td>600</td>
<td>800</td>
</tr>
<tr>
<td></td>
<td>300</td>
<td>700</td>
<td>1000</td>
</tr>
</tbody>
</table>

Risk ratio = \( \frac{100}{200} = \frac{200}{800} = 2.0 \)

Positive predictive value = \( \frac{100}{200} = 0.50 \) or 50%

Negative predictive value = \( \frac{600}{800} = 0.75 \) or 75%
While strength of association stays the same, predictive value changes as prevalence of heart disease increases from 10% in Population 1 to 30% in Population 2.
Nate Silver: politics ‘geek’ hailed for Barack Obama wins US election forecast

An American political blogger, Nate Silver, has emerged as the other major winner during the US election for predicting all major results and Barack Obama’s victory.

By Andrew Hough
8:30PM GMT 07 Nov 2012

Nate Silver, who writes the FiveThirtyEight blog for the New York Times, basked in worldwide adulation, after his polling correctly forecast the presidential outcome in 49 states.

The statistician, who tracked the president’s statistical odds, will have correctly foreseen the result in every US state if Mr Obama’s lead is maintained in Florida.

On election day, the 34 year-old, who had become a target for conservatives, also offered a 90.9 per cent probability of an Obama win in the US election.

He forecast that Mr Obama would win 332 electoral college votes compared to 208 for Mr Romney, which will be correct if the Democratic candidate wins Florida.
Semifinal: July 8, 2014

Adjusted odds for Neymar and Silva absences

65% BRA vs. 35% GER

Non-adjusted odds

73% BRA vs. 27% GER
Brazil crushed by Germany in the most humiliating defeat in World Cup history

1-7

MARTIN SAMUEL
Chief Sports Writer
reports from Sao Paulo

TREK must have been a moment when even those watching in their white Mannschaft shirts inside Estadio Mineiro would see. Germans are only human, after all.

Admittedly, the 1-7 on Brazil seemed to be农场农场that wasn't there, but there isn't anything - they must

NEGILIGENCE AT HOSPITAL OR DOCTORS?

important information

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*FIGURE 2.* Probability distributions of a marker, \( X \), in cases (solid curves) and controls (dashed curves) consistent with the logistic model \( \logit P(D = 1|X) = \alpha + \beta X \). It has been assumed that \( X \) has a mean of 0 and a standard deviation of 0.5 in controls so that a unit increase represents the difference between the 84th and 16th percentiles of \( X \) in controls. The marker is normally distributed, with the same variance in cases. The odds ratio (OR) per unit increase in \( X \) is shown.
1. Population health manifests as a continuum.
2. The causes of differences in health across populations are not necessarily an aggregate of the causes of differences in health within populations.
3. Large benefits to population health may not improve the lives of all individuals.
4. The causes of population health are multilevel, accumulate throughout the life course, and are embedded in dynamic interpersonal relationships.
5. Small changes in ubiquitous causes may result in more substantial change in the health of populations than larger changes in rarer causes.
6. The magnitude of an effect of exposure on disease is dependent on the prevalence of the factors that interact with that exposure.
7. Prevention of disease often yields a greater return on investment than curing disease after it has started.
8. Efforts to improve overall population health may be a disadvantage to some groups; whether equity or efficiency is preferable is a matter of values.
9. We can predict health in populations with much more certainty than we can predict health in individuals.
twitter/@sandrogalea
sgalea@bu.edu