Statistics 209B / EPI 239 / Education 260A **Winter 2022**

Applications of Causal Inference Methods

Winter 2022 Flipped Instruction

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TA David Fager dfager {AT} stanford {DOT} edu
Course web page: http://rogosateaching.com/stat209//

Course Welcome and Logistics (first day stuff, to be posted soon in December, call it Week0)
Lecture slides, week 0 (pdf) **Audio companion, week 0**
For recreation of in-classroom experience, linked below are youtube versions of the music I play
before starting lecture and after lecture concludes. Some may wish to reverse that ordering.

Registrar's Information

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Statistics 209B (also EPI 239, EDUC 260A)   2 units
Title: Applications of Causal Inference Methods
Description: Application of potential outcomes formulation for causal inference to research settings including: mediation,
compliance adjustments, time-1 time-2 designs, encouragement designs, heterogeneous treatment effects, aggregated data,
instrumental variables, analysis of covariance regression adjustments, and implementations of matching methods.
see class website http://rogosateaching.com/stat209/
 suggested prerequisite: an introduction to causal inference methods such as  STAT209 (aut).
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Course Overview
Note: This course was structured before the new world order of March 2020 as one two-hour lecture class meeting per week.
Last year, Winter 2021, this course was done Remote Asynchronous. Lectures (pdf slides plus audio) and all supporting materials are online.
To see full course materials from Winter 2021 go here
This year, the course will be done in flipped mode, with lectures (pdf slides and audio) and all supporting materials linked in these pages.
Web page materials to be posted sequentially for that week's instruction-- i.e. Week 1 materials to be posted Jan 3, and so forth.
The Wednesday class meeting, not required, will be used in part for review and discussion of the prior weeks' materials.
More on this in the Week0 intro materials.
The **first in-class discussion section will be January 12, 2022**.

Brief Course Outline

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Unit 1. Extensions of RCT
  Analyzing Encouragement Designs
  Assessing Mediation in experimental studies
  Identifying Moderation in experimental studies (heterogeneous treatment effects)
  The wisdom of Compliance Adjustments (for binary and measured compliance);
  Analysis of Regression Discontinuity Designs (systematic assignment based on a covariate)
Unit 2. Legacy Methods for Causal Inference from Observational Data (good and bad)
  Regression adjustments (analysis of covariance) in observational studies
  Instrumental Variables methods for observational studies
  Failures of traditional Path Analysis (and Structural Equation Models)
  Interpreting associations: Spurious Correlation and Simpson's Paradox
  Multilevel data and Contextual Effects
  Reciprocal Causal Effects and non-recursive models
Unit 3. Historical and Modern Methods for Matching in Observational Data
  Case-Control, case-referent matching sudies
  Subclassification matching
  Propensity Score matching examples
Unit 4. Time-1, Time-2 data in experimental and observational studies.
  Experimental Designs     Cross-over designs
  Experimental Designs     Comparing groups on time-1, time-2 measurements: repeated measures anova vs lmer OR the t-test
  Observational studies    Economist's differences in differences (or diffs in diffs with matching) for observational studies.
  Observational studies    Lord's paradox; pre-post group comparisons.
  Observational studies    Exogenous Variables and Correlates of Change (use of lagged dependent variables)
  Additional Special topics  Interrupted Time-series designs;  Current implementations of value-added analysis
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Lectures, Course Files, and Readings
this page is where course content resides

Grading, Exams, and Credit Units
Stat209B/EPI239/Ed260A is listed as Letter or Credit/No Credit grading for 2-units
For Winter 2022 grading for the 2-units will be based on a 'take home'(i.e. do at home) Problem Set.
Also as you will see (or can view from 2021 pages), for each week's content a number of Review Questions with Solutions are posted.

Course Problem Set 2022 to be posted

Statistical computing
Class presentation will be in, and students are encouraged to use, R (occasionally, some references to SAS and Mathematica).
Current version of R is R version 4.1.2 released 2021-11-01
For references and software: The R Project for Statistical Computing Closest download mirrors in the past, UCLA and Berkeley, seem no longer avaliable, pick your fave anywhere in the world.
The CRAN Task Views provide useful overviews/navigation for the almost infinite set of R-packages. Task Views that cover contents of this course include CRAN Task View: Statistics for the Social Sciences; CRAN Task View: Econometrics and CRAN Task View: Psychometric Models and Methods .

Relevent Texts (optional).
Causal Inference in Statistics, Social and Biomedical Sciences: An Introduction, Guido Imbens and Don Rubin, 1st Edition (Cambridge University Press) Stanford access
Design of Observational Studies, Paul Rosenbaum, 1st Edition (Springer). Available online: Stanford access

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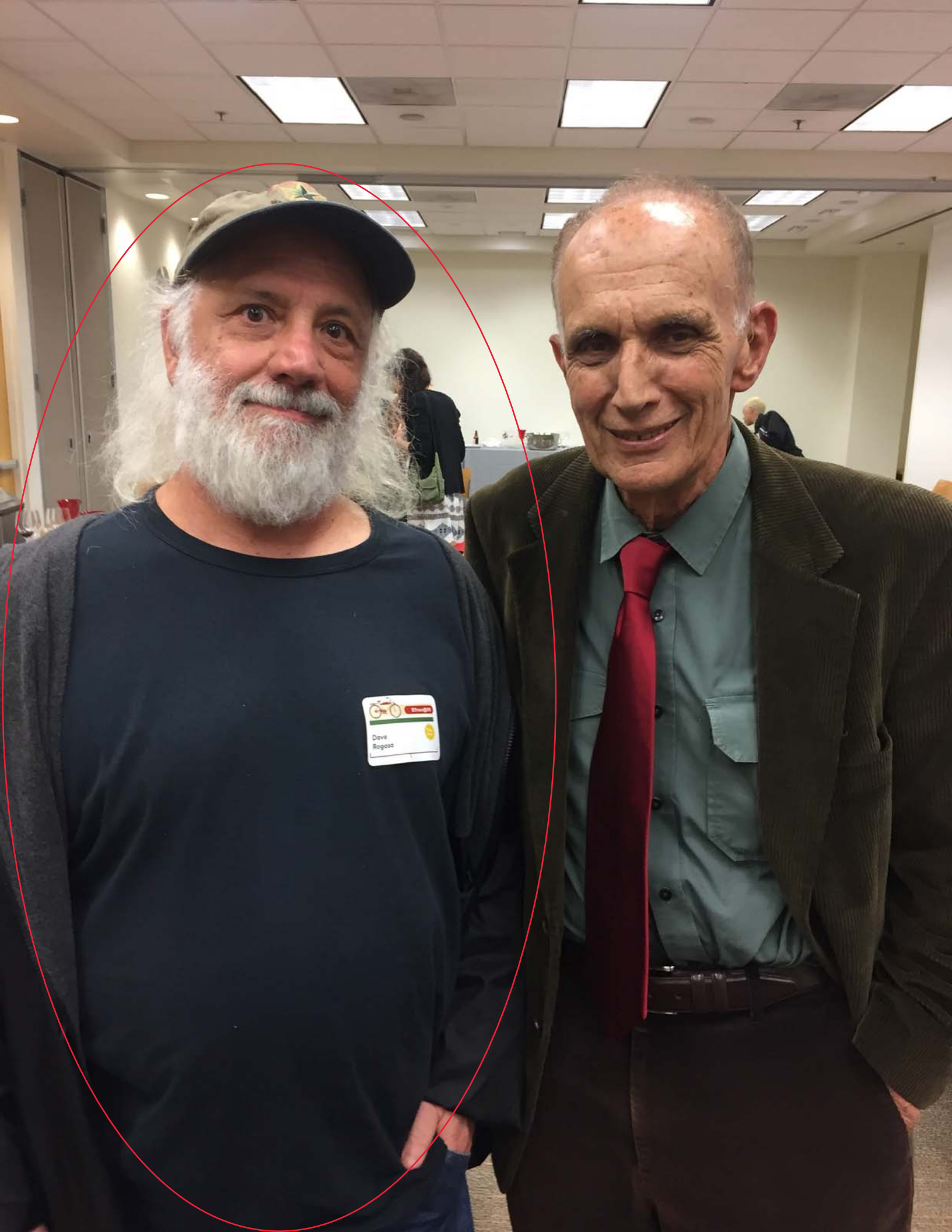
To see full course materials from legacy Stat209 (2005-2019) also stat266/epi292
Statistical Methods for Group Comparisons and Causal Inference go here


Dave
Rogosa

David Fager, STAT209B TA

Stat 209B-- Lectures, Course Files, and Readings

Week 0

Course introduction (slides and audio to be posted on main page)

Background readings (not required, but of interest if you haven't seen these before)

1. Correlation and Causation: A Comment, Stephen Stigler *Perspectives in Biology and Medicine*, volume 48, number 1 supplement (winter 2005)
2. Secret to Winning a Nobel Prize? Eat More Chocolate (Time)
   Publication: Chocolate Consumption, Cognitive Function, and Nobel Laureates Franz H. Messerli, M.D. N Engl J Med 2012; 367:1562-1564 October 18, 2012
3. *David Freedman chapters*.
   From Association to Causation: Some Remarks on the History of Statistics;
   Statistical Models for Causation: A critical review
   Statistical Models and Shoe Leather, *Sociological Methodology*, Vol. 21. (1991), pp. 291-313. JStor link

Week 1

OCCASIONAL NOTES

Chocolate Consumption, Cognitive Function, and Nobel Laureates

Franz H. Messerli, M.D.

Dietary flavonoids, abundant in plant-based foods, have been shown to improve cognitive function. Specifically, a reduction in the risk of dementia, enhanced performance on some cognitive tests, and improved cognitive function in elderly patients with mild impairment have been associated with a regular intake of flavonoids.[1,2] A subclass of flavonoids called flavanols, which are widely present in cocoa, green tea, red wine, and some fruits, seems to be effective in slowing down or even reversing the reductions in cognitive performance that occur with aging. Dietary flavanols have also been shown to improve endothelial function and to lower blood pressure by causing vasodilation in the peripheral vasculature and in the brain.[3,4] Improved cognitive performance with the administration of a cocoa polyphenolic extract has even been reported in aged Wistar–Unilever rats.[5]

Since chocolate consumption could hypothetically improve cognitive function not only in individuals but also in whole populations, I wondered whether there would be a correlation between a country's level of chocolate consumption and its population's cognitive function. To my knowledge, no data on overall national cognitive function are publicly available. Conceivably, however, the total number of Nobel laureates per capita could serve as a surrogate end point reflecting the proportion with superior cognitive function and thereby give us some measure of the overall cognitive function of a given country.

METHODS

A list of countries ranked in terms of Nobel laureates per capita was downloaded from Wikipedia (http://en.wikipedia.org/wiki/List_of_countries_by_Nobel_laureates_per_capita). Because the population of a country is substantially higher than its number of Nobel laureates, the numbers had to be multiplied by 10 million. Thus, the numbers must be read as the number of Nobel laureates for every 10 million persons in a given country.

All Nobel Prizes that were awarded through October 10, 2011, were included. Data on per capita yearly chocolate consumption in 22 countries was obtained from Chocosuisse (www.chocosuisse.ch/web/chocosuisse/en/home), Theobroma-cacao (www.theobroma-cacao.de/wissen/wirtschaft/international/konsum), and Caobisco (www.caobisco.com/page.asp?p=213). Data were available from 2011 for 1 country (Switzerland), from 2010 for 15 countries, from 2004 for 5 countries, and from 2002 for 1 country (China).

RESULTS

There was a close, significant linear correlation ($r=0.791$, $P<0.0001$) between chocolate consumption per capita and the number of Nobel laureates per 10 million persons in a total of 23 countries (Fig. 1). When recalculated with the exclusion of Sweden, the correlation coefficient increased to 0.862. Switzerland was the top performer in terms of both the number of Nobel laureates and chocolate consumption. The slope of the regression line allows us to estimate that it would take about 0.4 kg of chocolate per capita per year to increase the number of Nobel laureates in a given country by 1. For the United States, that would amount to 125 million kg per year. The minimally effective chocolate dose seems to hover around 2 kg per year, and the dose–response curve reveals no apparent ceiling on the number of Nobel laureates at the highest chocolate-dose level of 11 kg per year.

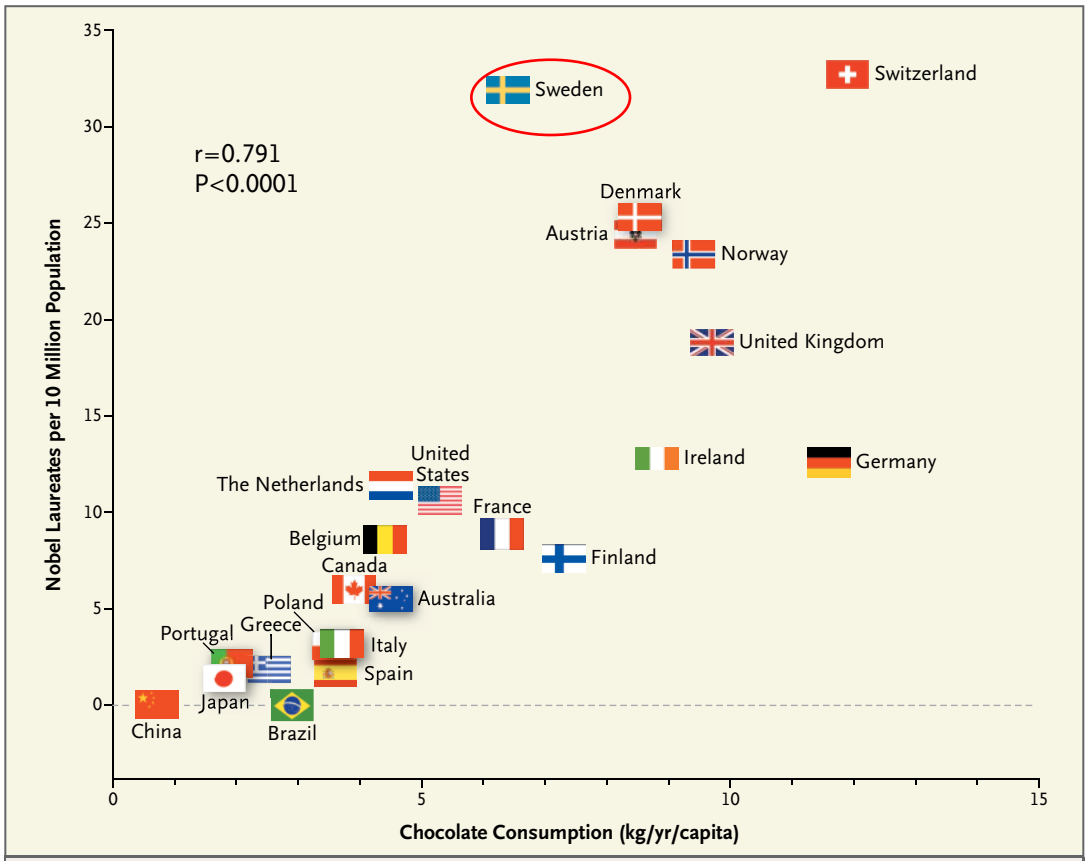


Figure 1. Correlation between Countries' Annual Per Capita Chocolate Consumption and the Number of Nobel Laureates per 10 Million Population.

DISCUSSION

The principal finding of this study is a surprisingly powerful correlation between chocolate intake per capita and the number of Nobel laureates in various countries. Of course, a correlation between X and Y does not prove causation but indicates that either X influences Y, Y influences X, or X and Y are influenced by a common underlying mechanism. However, since chocolate consumption has been documented to improve cognitive function, it seems most likely that in a dose-dependent way, chocolate intake provides the abundant fertile ground needed for the sprouting of Nobel laureates. Obviously, these findings are hypothesis-generating only and will have to be tested in a prospective, randomized trial.

The only possible outlier in Figure 1 seems to be Sweden. Given its per capita chocolate consumption of 6.4 kg per year, we would predict that Sweden should have produced a total of about 14 Nobel laureates, yet we observe 32. Considering that in this instance the observed number exceeds the expected number by a factor of more than 2, one cannot quite escape the notion that either the Nobel Committee in Stockholm has some inherent patriotic bias when assessing the candidates for these awards or, perhaps, that the Swedes are particularly sensitive to chocolate, and even minuscule amounts greatly enhance their cognition.

A second hypothesis, reverse causation — that is, that enhanced cognitive performance could stimulate countrywide chocolate consumption — must also be considered. It is conceivable that persons with superior cognitive function (i.e., the cognoscenti) are more aware of the health benefits of the flavanols in dark chocolate and are therefore prone to increasing their consumption. That receiving the Nobel Prize would in itself increase chocolate intake countrywide seems unlikely, although perhaps celebratory events associated with this unique

honor may trigger a widespread but most likely transient increase.

Finally, as to a third hypothesis, it is difficult to identify a plausible common denominator that could possibly drive both chocolate consumption and the number of Nobel laureates over many years. Differences in socioeconomic status from country to country and geographic and climatic factors may play some role, but they fall short of fully explaining the close correlation observed.

STUDY LIMITATIONS

WEEK6

The present data are based on country averages, and the specific chocolate intake of individual Nobel laureates of the past and present remains unknown. The cumulative dose of chocolate that is needed to sufficiently increase the odds of being asked to travel to Stockholm is uncertain. This research is evolving, since both the number of Nobel laureates and chocolate consumption are time-dependent variables and change from year to year.

CONCLUSIONS

Chocolate consumption enhances cognitive function, which is a sine qua non for winning the Nobel Prize, and it closely correlates with the number of Nobel laureates in each country. It remains to be determined whether the consumption of chocolate is the underlying mechanism for the observed association with improved cognitive function.

Dr. Messerli reports regular daily chocolate consumption, mostly but not exclusively in the form of Lindt's dark varieties.

Disclosure forms provided by the author are available with the full text of this article at NEJM.org.

From St. Luke's–Roosevelt Hospital and Columbia University, New York.

This article was published on October 10, 2012, at NEJM.org.

1. Nurk E, Refsum H, Drevon CA, et al. Intake of flavonoid-rich wine, tea, and chocolate by elderly men and women is associated with better cognitive test performance. J Nutr 2009;139:120-7.
2. Desideri G, Kwik-Uribe C, Grassi D, et al. Benefits in cognitive function, blood pressure, and insulin resistance through cocoa flavanol consumption in elderly subjects with mild cognitive impairment: the Cocoa, Cognition, and Aging (CoCoA) Study. Hypertension 2012;60:794-801.
3. Corti R, Flammer AJ, Hollenberg NK, Lüscher TF. Cocoa and cardiovascular health. Circulation 2009;119:1433-41.
4. Sorond FA, Lipsitz LA, Hollenberg NK, Fisher ND. Cerebral blood flow response to flavanol-rich cocoa in healthy elderly humans. Neuropsychiatr Dis Treat 2008;4:433-40.
5. Bisson JF, Nejdi A, Rozan P, Hidalgo S, Lalonde R, Messaoudi M. Effects of long-term administration of a cocoa polyphenolic extract (Acticoa powder) on cognitive performances in aged rats. Br J Nutr 2008;100:94-101.

DOI: 10.1056/NEJMon1211064

Correlation and Causation

a comment

Stephen Stigler

ABSTRACT Some purely methodological comments are made on the pitfalls and difficulties in making causal inferences from observational data, including in studies of disparity in medicine. The ideas of spurious correlation and measurement error are discussed with an eye towards their impact upon inferences about causality, and cautions are offered about over-reliance upon testing hypotheses.

STATISTICIANS HAVE LONG STRUGGLED to deduce causal relationships from correlation; that is, to determine a mechanistic relationship from purely empirical evidence of association. That is also the essential goal of the methods that are being discussed here. There are other important issues to be sure, such as whether or not the deduced causal relationship is illegal or immoral, but statistically that is secondary to the study of the nature of the relationship. The answer to the strict question "Can cause be deduced from correlation?" is generally "no." But necessity being the mother of invention, we do it anyway, by weakening the question to one permitting a more positive answer: "Under certain restrictive assumptions, can we conclude causation from correlation, beyond a reasonable doubt?" In that form a great deal of methodological progress has been made, including by one of this year's Nobel Prize–winning economists, Clive Granger. But the answer to the strict question remains "no," and it may be worthwhile recalling why that is so.

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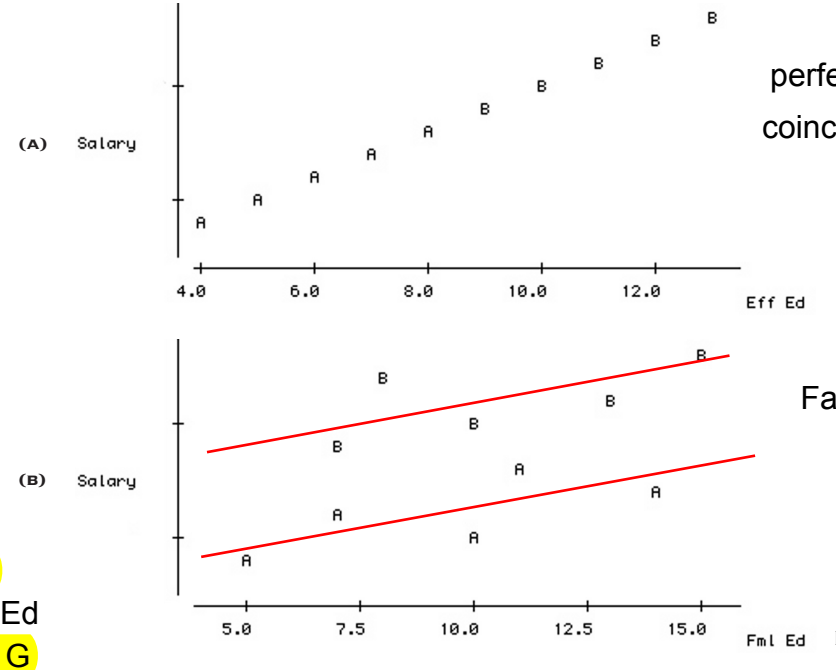

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FIGURE 4

*An illustration of measurement error bias. The salary of two groups of five individuals each (As and Bs) are compared on the basis of effective education (*top panel*) and formal education (which is effective education measured with error,* lower panel*). In the top panel the relationship is strong, with no sign of bias; salary is proportional to effective education. In the lower panel it appears that the Bs are paid more than the As, even allowing for differences in formal education, despite the fact that the salaries are the same in both panels.*

cf, Freedman Chap 1, Yule

showed that pellagra was the result of a diet deficiency. Terris (1964) reprints many of Goldberger's articles; also see Carpenter (1981). A useful reference on Pasteur is Dubos (1988). References on the history of cholera include Rosenberg (1962), Howard-Jones (1975), Evans (1987), Winkelstein (1995), Paneth et al. (1998). Today, the molecular biology of the cholera vibrio is reasonably well understood; see, for instance, Finlay, Heffron, and Fialkow (1989) or Miller, Mekalanos, and Fialkow (1989). For a synopsis, see Alberts et al. (1994, pp. 484, 738); there are recent surveys by Colwell (1996) and Raufman (1998). Problems with ecological inference are discussed in Freedman (2001).

4. REGRESSION MODELS IN SOCIAL SCIENCE

Legendre (1805) and Gauss (1809) developed the regression method (least absolute residuals or least squares) to fit data on the orbits of astronomical objects. In this context, the relevant variables are known and so are the functional forms of the equations connecting them. Measurement can be done to high precision, and much is known about the nature of the errors—in the measurements and the equations. Furthermore, there is ample opportunity for comparing predictions to reality.

By the turn of the century, investigators were using regression on social science data where these conditions did not hold, even to a rough approximation. One of the earliest such papers is Yule (1899), "An investigation into the causes of changes in pauperism in England, chiefly during the last two intercensal decades." At the time, paupers were supported either inside "poor-houses" or outside, depending on the policy of local authorities. Did the relief policy affect the number of paupers? To study this question, Yule offered a regression equation,

$$\Delta\text{Paup} = a + b \times \Delta\text{Out} + c \times \Delta\text{Old} + d \times \Delta\text{Pop} + \text{error}.$$

In this equation,

Δ is percentage change over time,
"Out" is the out-relief ratio N/D,
$N =$ number on welfare outside the poor-house,
$D =$ number inside,
"Old" is the percentage of the population over 65,
"Pop" is the population.

Data are from the English Censuses of 1871, 1881, 1891. There are two Δ's, one for 1871–81 and one for 1881–91.

Relief policy was determined separately by the local authorities in each "union," a small geographical area like a parish. At the time, there were about 600 unions, and Yule divided them into four kinds: rural, mixed, urban, metropolitan. There are $2 \times 4 = 8$ equations, one for each combination of time period and type of union. Yule assumed that the coefficients were constant for each equation, which he fitted to the data by least squares. That is, he estimated the coefficients a, b, c, and d as the values that minimized the sum of squared errors,

$$\sum \left(\Delta\text{Paup} - a - b \times \Delta\text{Out} - c \times \Delta\text{Old} - d \times \Delta\text{Pop}\right)^2.$$

The sum is taken over all unions of a given type at a given time-period.

For example, consider the metropolitan unions. Fitting the equation to the data for 1871–81 gave

$$\Delta\text{Paup} = 13.19 + 0.755\Delta\text{Out} - 0.022\Delta\text{Old} - 0.322\Delta\text{Pop} + \text{residual}.$$

For 1881–91, Yule's equation was

$$\Delta\text{Paup} = 1.36 + 0.324\Delta\text{Out} + 1.37\Delta\text{Old} - 0.369\Delta\text{Pop} + \text{residual}.$$

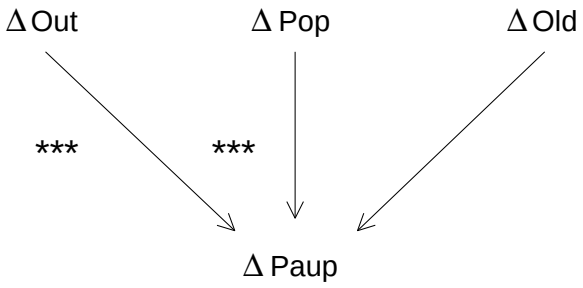
The framework combines the ideas of Quetelet with the mathematics of Gauss. Yule is studying the "social physics" of poverty. Nature has run an experiment, assigning different treatments to different areas. Yule is analyzing the results, using regression to isolate the effects of out-relief. His principal conclusion is that welfare outside the poor-house creates paupers—the estimated coefficient on the out-relief ratio is positive.

At this remove, the flaws in the argument are clear. Confounding is a salient problem. For instance, Pigou (a famous economist of the era) thought that unions with more efficient administrations were the ones building poor-houses and reducing poverty. Efficiency of administration is then a confounder, influencing both the presumed cause and its effect. Economics may be another confounder. At times, Yule seems to be using the rate of population change as a proxy for economic growth, although this is not entirely convincing. Generally, however, he pays little attention to economic activity. The explanation: "A good deal of time and labour was spent in making trial of this idea, but the results proved unsatisfactory, and finally the measure was abandoned altogether. [p. 253]"

The form of his equation is somewhat arbitrary, and the coefficients are not consistent over time and space. This is not necessarily fatal. However, if the coefficients do not exist separately from the data, how can they predict the results of interventions? There are also problems of interpretation. At best, Yule has established association. Conditional on the covariates, there is a positive association between ΔPaup and ΔOut. Is this association causal? If so, which way do the causal arrows point? These questions are not answered by the data analysis; rather, the answers are assumed a priori. Yule is quite concerned to parcel out changes in pauperism: so much is due to changes in the out-relief ratio, so much to changes in other variables, and so much to random effects. However, there is one deft footnote (number 25) that withdraws all causal claims:

"Strictly speaking, for 'due to' read 'associated with.'"

FIGURE 1. Yule's Model. Metropolitan Unions, 1871–81.



Yule's approach is strikingly modern, except there is no causal diagram and no stars indicating statistical significance. Figure 1 brings him up to date. An arrow from X to Y indicates that X

is included in the regression equation that explains Y. "Statistical significance" is indicated by an asterisk, and three asterisks signal a high degree of significance. The idea is that a statistically significant coefficient differs from 0, so that X has a causal influence on Y. By contrast, an insignificant coefficient is zero: then X does not exert a causal influence on Y.

The reasoning is seldom made explicit, and difficulties are frequently overlooked. Stringent assumptions are needed to determine significance from the data. Even if significance can be determined and the null hypothesis rejected or accepted, there is a much deeper problem. To make causal inferences, it must in essence be assumed that equations are invariant under proposed interventions. Verifying such assumptions—without making the interventions—is quite problematic. On the other hand, if the coefficients and error terms change when the right hand side variables are manipulated rather than being passively observed, then the equation has only a limited utility for predicting the results of interventions. These difficulties are well known in principle, but are seldom dealt with by investigators doing applied work in the social and life sciences. Despite the problems, and the disclaimer in the footnote, Yule's regression approach has become widely used in the social sciences and epidemiology.

Some formal models for causation are available, starting with Neyman (1923). See Hodges and Lehmann (1964, sec. 9.4), Rubin (1974), or Holland (1988). More recent developments will be found in Pearl (1995, 2000) or Angrist, Imbens and Rubin (1996). For critical discussion from various perspectives, see Goldthorpe (1998, 2001), Humphreys and Freedman (1996, 1999), Abbott (1997), McKim and Turner (1997), Manski (1995), Lieberson (1985), Lucas (1976), Liu (1960), or Freedman (1987, 1991, 1995). Ní Bhrolcháin (2001) presents some fascinating case studies. The role of invariance is considered in Heckman (2000) and Freedman (2002). The history is reviewed by Stigler (1986) and Desrosières (1993).

5. REGRESSION MODELS IN EPIDEMIOLOGY

Regression models (and variations like the Cox model) are widely used in epidemiology. The models seem to give answers, and create at least the appearance of methodological rigor. This section discusses one example, which is fairly typical of such applications and provides an interesting contrast to Snow on cholera. Snow used primitive statistical techniques, but his study designs were extraordinarily well thought out, and he made a huge effort to collect the relevant data. By contrast, many empirical papers published today, even in the leading journals, lack a sharply-focused research question; or the study design connects the hypotheses to the data collection only in a very loose way. Investigators often try to use statistical models not only to control for confounding, but also to correct basic deficiencies in the design or the data. Our example will illustrate some of these points.

Kanarek et al. (1980) asked whether asbestos in the drinking water causes cancer. They studied 722 census tracts in the San Francisco Bay Area. (A census tract is a small geographical region, with several thousand inhabitants.) The investigators measured asbestos concentration in the water for each tract. Perhaps surprisingly, there is enormous variation; less surprisingly, higher concentrations are found in poorer tracts. Kanarek et al. compared the "observed" number of cancers by site with the expected number, by sex, race, and tract. The "expected" number is obtained by applying age-specific national rates to the population of the tract, age-group by age-group; males and females are done separately, and only whites are considered. (There are about 100 sites for which age-specific national data are available; comparison of observed to expected numbers is an

on the same scale after standardization, which makes it easier to compare regression coefficients.

standardized vs unstandardized

Hooke's Law

According to Hooke's law, stretch is proportional to weight. If weight x is hung on a spring, the length of the spring is $a + bx + \epsilon$, provided x is not too large. (Near the elastic limit of the spring, the physics will be more complicated.) In this equation, a and b are physical constants that depend on the spring not the weights. The parameter a is the length of the spring with no load. The parameter b is the length added to the spring by each additional unit of weight. The ϵ is random measurement error, with the usual assumptions. Experimental verification is a classroom staple.

If we were to standardize, the crucial slope parameter would depend on the weights and the accuracy of the measurements. Let v be the variance of the weights used in the experiment, let σ^2 be the variance of ϵ, and let s^2 be the mean square of the deviations from the fitted regression line. The standardized regression coefficient is

$$\sqrt{\frac{\hat{b}^2 v}{\hat{b}^2 v + s^2}} \approx \sqrt{\frac{b^2 v}{b^2 v + \sigma^2}}, \tag{7}$$

as can be verified by examining the sample covariance matrix. Therefore, the standardized coefficient depends on v and σ^2, which are features of our measurement procedure not the spring.

Hooke's law is an example where regression is a very useful tool. But the parameter to estimate is b, the unstandardized regression coefficient. It is the unstandardized coefficient that says how the spring will respond when the load is manipulated. If a regression coefficient is stable under interventions, standardizing it is probably not a good idea, because stability gets lost in the shuffle. That is what (7) shows. Also see [4], ([11], p. 451).

Political Repression During the McCarthy Era

Gibson [28] tries to determine the causes of McCarthyism in the United States. Was repression due to the masses or the elites? He argues that elite intolerance is the root cause, the chie of evidence being a path model (Figure 3, r from the paper). The dependent variable measure of repressive legislation in each sta independent variables are mean tolerance sco each state, derived from the Stouffer sur masses and elites. The 'masses' are just respo in a probability sample of the population. include school board presidents, commanders American Legion, bar association presidents union leaders. Data on masses were availa 36 states; on elites, for 26 states. The two arrows in Figure 3 represent causal links: ma elite tolerance affect repression. The curved headed arrow in Figure 3 represents an asso between mass and elite tolerance scores. Each influence the other, or both can have some c cause. The association is not analyzed in the di

Gibson computes correlations from the av data, then estimates a standardized regressio tion,

$$\text{Repression} = \beta_1 \text{Mass tolerance} + \beta_2 \text{Elite tolerance} + \delta.$$

He says, 'Generally, it seems that elites, not m were responsible for the repression of the era. beta for mass opinion is −.06; for elite opinio −.35 (significant beyond .01)'.

The paper asks an interesting question, a data analysis has some charm too. However, a physics, the path model is not convincing hypothetical intervention is contemplated? I

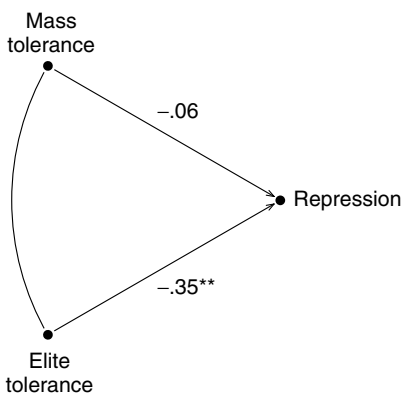


Figure 3 Path model. The causes of McCarthyis

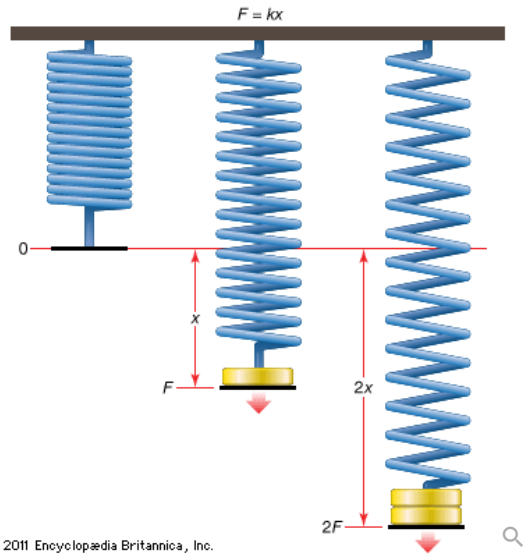
DAF text sec 5.3, w2 handout also

Hooke's law

PHYSICS

WRITTEN BY: The Editors of Encyclopædia Britannica
LAST UPDATED: 12-15-2017 See Article History

Hooke's law, law of elasticity discovered by the English scientist Robert Hooke in 1660, which states that, for relatively small deformations of an object, the displacement or size of the deformation is directly proportional to the deforming force or load. Under these conditions the object returns to its original shape and size upon removal of the load. Elastic behaviour of solids according to Hooke's law can be explained by the fact that small displacements of their constituent molecules, atoms, or ions from normal positions is also proportional to the force that causes the displacement.



Hooke's law, $F = kx$, where the applied force F equals a constant k times the displacement or change in length x.

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RELATED TOPI
- Robert Hool
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- Physics
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- Elastic limit
- Plasticity
- Ductility

The deforming force may be applied to a solid by stretching, compressing, squeezing, bending, or twisting. Thus, a metal wire exhibits elastic behaviour according to Hooke's law because the small increase in its length when stretched by an applied force doubles each time the force is doubled. Mathematically, Hooke's law states that the applied force F equals a constant k times the displacement or change in length x, or $F = kx$. The value of k depends not only on the kind of elastic material under consideration