On the many uses and abuses of regression models

John Carlin

Murdoch Children's Research Institute
& University of Melbourne

ISCB President's Invited Speaker, Newcastle, U.K.
22 August 2022 (reprised for ViCBiostat Seminar, 24-Nov-22)

A word or two of introduction

- A statistical "bomb-thrower"?

> "... the listed committee [*ASA Task Force on Statistical Significance and Replicability*] seems like a bunch of reasonable people, no bomb-throwers like us or Nicole Lazar or John Carlin or Sander Greenland or various others to represent the voice of radical reform..."
>
> – Andrew Gelman blog posting, February 21, 2020

A word or two of introduction

- A statistical "bomb-thrower"?

> "... the listed committee [*ASA Task Force on Statistical Significance and Replicability*] seems like a bunch of reasonable people, no bomb-throwers like

Probable Error

I don't mean to sound critical, but I am; so that's how it comes across

HOME ABOUT ACADEMIC PUBLICATIONS COMICS

https://mchankins.wordpress.com/2013/04/21/still-not-significant-2/

A word or two of introduction

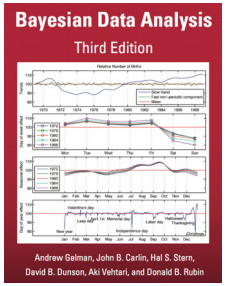
J. Austral. Math. Soc. (Series A) 36 (1984), 30–52

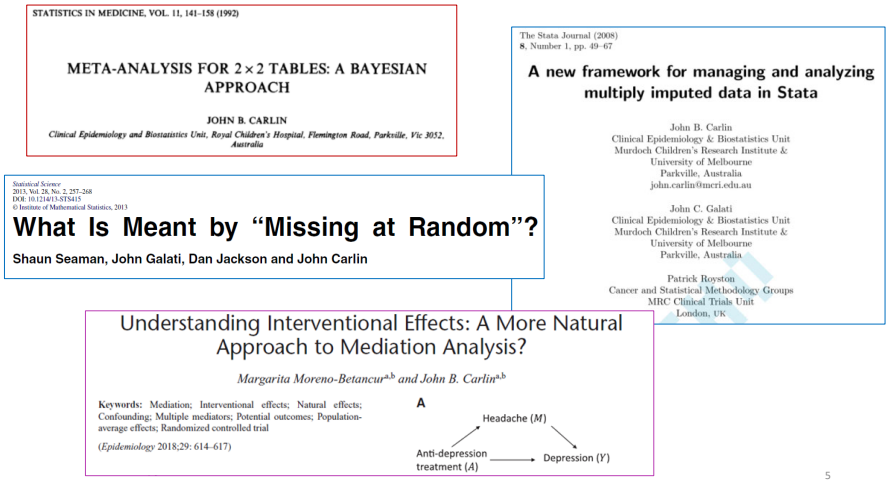
RECURSIVE CAUSAL MODELS

HARRI KIIVERI, T. P. SPEED and J. B. CARLIN

Sensitivity Analysis of Seasonal Adjustments: Empirical Case Studies

J. B. CARLIN and A. P. DEMPSTER*





STATISTICS IN MEDICINE, VOL. 11, 141–158 (1992)

META-ANALYSIS FOR 2×2 TABLES: A BAYESIAN APPROACH

JOHN B. CARLIN

Clinical Epidemiology and Biostatistics Unit, Royal Children's Hospital, Flemington Road, Parkville, Vic 3052, Australia

The Stata Journal (2008)
8, Number 1, pp. 49–67

A new framework for managing and analyzing multiply imputed data in Stata

John B. Carlin
Clinical Epidemiology & Biostatistics Unit
Murdoch Children's Research Institute &
University of Melbourne
Parkville, Australia
john.carlin@mcri.edu.au

John C. Galati
Clinical Epidemiology & Biostatistics Unit
Murdoch Children's Research Institute &
University of Melbourne
Parkville, Australia

Patrick Royston
Cancer and Statistical Methodology Groups
MRC Clinical Trials Unit
London, UK

Statistical Science
2013, Vol. 28, No. 2, 257–268
DOI: 10.1214/13-STS415
© Institute of Mathematical Statistics, 2013

What Is Meant by "Missing at Random"?

Shaun Seaman, John Galati, Dan Jackson and John Carlin

Understanding Interventional Effects: A More Natural Approach to Mediation Analysis?

Margarita Moreno-Betancur[a,b] and John B. Carlin[a,b]

Keywords: Mediation; Interventional effects; Natural effects; Confounding; Multiple mediators; Potential outcomes; Population-average effects; Randomized controlled trial

(*Epidemiology* 2018;29: 614–617)

A

Headache (*M*)

Anti-depression treatment (*A*)

Depression (*Y*)

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A talk about *practice* and *teaching*…

- … not about new methods or methodological research
  - … and ideas will be familiar to some, just not sufficiently connected to the 'mainstream'
- ***Practice*** is important: without active engagement in improving statistical standards, biostatisticians put their discipline at risk
- ***We should take responsibility for what is done in the name of (bio)statistics***
  - Bad science is a problem, often fed by bad statistics
- Need to engage in ***teaching and training*** at all levels to improve practice

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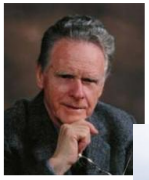
Arthur P. Dempster

Emeritus Professor of Theoretical Statistics

Advice to PhD students:
"Knowledge of techniques is not the important thing in statistics"



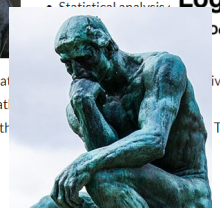
Research Interests

- Methodology and logic of applied statistics.
- Computational aspe
- Modelling and analy

- 1956: Ph.D. in Mat
- 1953: M.A. in Mat
- 1952: B.A. in Math

iversity

Toronto



Statistical Science
1998, Vol. 13, No. 3, 248–276

Logicist Statistics I. Models and Modeling

Dempster

2. WHAT "IS" A MODEL?

"Model" is used here interchangeably with the awkwardly long "mathematical model." The long form draws attention to abstract or purely mathematical content, while the short form suggests a type of replica, here a formal representation of objective reality through a corresponding mathematical structure. The term model implies, in addition to the abstract structure, a defined set of connections of the structure to the objective world,

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Journey of an applied statistician (me!)

- Training: BSc, Masters & PhD in Statistics, limited practical experience
- Motivation/interest: using statistical methods to 'make sense of data', i.e. answer questions in health & medical research
- On the job: a big gap between training and confidence in practice
- How to cope? Looked around and noticed that a lot of statistical analysis published in medical journals uses regression models… so that was it!
- How to succeed? Become skilled at fitting these models and back-engineering stories that sound meaningful to collaborators!

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Example 1

- Question:

How much is a child's kidney enlarged after acute infection?

- Use of regression?

Sonographic Measurement of Renal Enlargement in Children with Acute Pyelonephritis and Time Needed for Resolution: Implications for Renal Growth Assessment

Frederick E. Pickworth[1]
John B. Carlin[1]
Michael R. Ditchfield[1]
Margaret P. de Campo[1]
John F. de Campo[1]
David J. Cook[1]
Terry Nolan[3]
Harley R. Powell[4]
Robert Sloane[3]
Keith Grimwood[3]

OBJECTIVE. Failure of a kidney to grow satisfactorily in childhood is evidence of renal disease. Because kidneys may enlarge during an episode of acute pyelonephritis, concomitant renal length measurements cannot be used as baselines for growth assessment. This study was designed to determine the degree of renal enlargement in children with acute pyelonephritis and the time the enlargement takes to resolve after treatment is started to find the optimum time for obtaining baseline measurements.

SUBJECTS AND METHODS. In a cohort study, 180 children younger than 5 years old with their first proven acute urinary tract infection, with or without pyelonephritis, had renal scintigraphy and sonography within 15 days of starting treatment. The presence of cortical defects on scintigrams indicated pyelonephritis. The lengths of kidneys with and without scintigraphic defects (i.e., with and without pyelonephritis) were compared, adjusting for age and sex, and the length of kidneys with defects was related to time elapsed between the start of treatment and sonography.

RESULTS. Ninety-nine kidneys (28%) in 77 children (43%) had scintigraphic defects. Kidneys with defects were an average of 3.2 mm longer than kidneys without defects. Length and time interval between treatment and sonography in kidneys with defects correlated negatively, with mean length approaching that of kidneys without defects by 10–11 days.

CONCLUSION. Kidneys with acute pyelonephritis initially increase in length but return to normal on average by the 11th day of treatment. If poor renal growth is used as an indication of renal disease, sonography should be delayed or repeated at least 2 weeks after the start of treatment to determine the length of the uninflamed kidney.

AJR 1995;165:405–408

Example 1

Use of regression?

- *Kidney length increases with age*
- *Regression allows estimation of mean difference controlling for age… assuming difference ~constant!*

Nice descriptive summary of data

- *Aside: cubic curve used for age dependence*

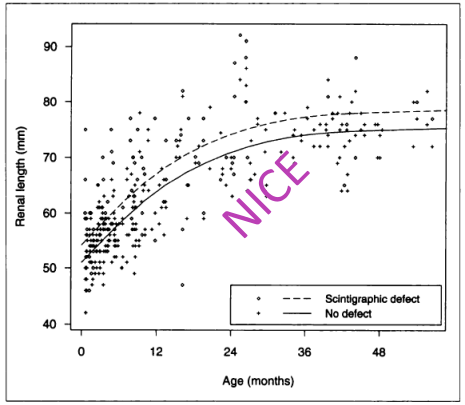

Fig. 1.—Scatter plot of renal length measured on sonograms versus age for kidneys with and without defects shown on scintigrams. Curved lines represent cubic model used in analysis of covariance calculations. Because curves are parallel, there is a similar absolute increase in renal length at all ages.

Example 2

- Question:

Can clinical factors predict successful gas enema for intussusception?

- Use of regression?

Gas Enema for the Reduction of Intussusception: Relationship Between Clinical Signs and Symptoms and Outcome

M. Katz[1]
E. Phelan[1]
J. B. Carlin[2]
S. W. Beasley[3]

OBJECTIVE. The aim of this study was to establish the extent to which the clinical features of intussusception can be used to predict successful outcome of gas enema and to determine whether the nonsurgical management of intussusception in children can be improved by refining the criteria used to select patients for gas enema.

SUBJECTS AND METHODS. Clinical data on 282 consecutive episodes of intussusception (255 patients) were collected prospectively from January 1987 to July 1991. Gas enema was performed in 273 episodes, in which the clinical signs and symptoms were studied by using logistic regression. Nine patients had primary surgery.

RESULTS. Gas enema was successful in 216 (79%) of 273 enemas attempted. Fifty-seven patients had surgery after unsuccessful enema. Univariate analysis showed significant associations between successful enema and duration of signs and symptoms less than 12 hr, no rectal bleeding, absence of small-bowel obstruction, presence of a palpable mass, and normal hydration. Multivariate analysis showed that dehydration, small-bowel obstruction, and duration of signs and symptoms longer than 12 hr were significant predictors of unsuccessful enema; yet, in these groups the rate of success still justified attempted enema. Even in severe dehydration, the successful enema reduction rate was 31%.

CONCLUSION. Our data suggest that although the factors identified had some predictive value in determining the outcome of attempted enema reduction, they could not be used to indicate patients in whom enema reduction should not be attempted. All patients with intussusception should have a gas enema if the absolute contraindications to enema (i.e., peritonitis or perforation) are absent.

AJR 1993;160:363–366

Example 2

- Question:

Can clinical factors predict successful gas enema for intussusception?

- Use of regression?

Logistic regression "to determine which variables were predictive… forward selection procedure was used…"

TABLE 4: Results of Logistic Regression Analysis with Successful Gas Enema as Outcome for Children with Intussusception

Predictor Variable[a]	*p* Value[b]	Odds Ratio[c]	95% Confidence Interval
Dehydration level	<.001		
1–4%		0.32	(0.13, 0.80)
5%		0.13	(0.05, 0.33)
6–10%		0.10	(0.02, 0.42)
Duration of symptoms >12 hr	.03	0.42	(0.02, 0.90)
Small-bowel obstruction	.005		
1–2 fluid levels		0.78	(0.32, 1.90)
>3 fluid levels		0.24	(0.10, 0.57)
Palpable mass present	.03	2.43	(1.07, 5.50)

Note.—Baseline odds of successful gas enema for well-hydrated patients who had signs and symptoms for less than 12 hr, no obstruction, and no palpable mass were 10.1.

[a]Variables in Table 1 that are omitted from this table showed no significant contribution to the multivariate model. No significant interactions were found between the independent variables.

[b]Likelihood ratio test for variable when entered last.

[c]Odds ratio comparing given level of each variable with baseline: for example, at dehydration level 1, odds of success are .32 times the odds for normal hydration, assuming all other variables remain constant.

MISGUIDED

Example 3

- Question:

Estimate "strength of association" of numerous factors with risk of childhood asthma

- Use of regression?

Paediatric and Perinatal Epidemiology 1993, 7, 67–76

The associations between childhood asthma and atopy, and parental asthma, hay fever and smoking

Mark A. Jenkins*, John L. Hopper*, Louisa B. Flander*, John B. Carlin† and Graham G. Giles‡
*Faculty of Medicine, Epidemiology Unit, The University of Melbourne, Parkville, Victoria, †Clinical Epidemiology and Biostatistics Unit, Royal Children's Hospital, Parkville, Victoria, and ‡Anti Cancer Council of Victoria, 1 Rathdowne St, Carlton, Victoria, Australia

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Example 3

- Question:

Estimate "strength of association" of numerous factors with risk of childhood asthma

- Use of regression?

"Relationships between [Y] and explanatory variables [$X_1, X_2, \ldots$] were analysed by multiple logistic regression"

Table 2. Odds ratios and 99% confidence intervals for child's asthma after adjustment for all other factors in the model

Risk factor	n=7368
Maleness	1.56 (1.30–1.86)
Hay fever	3.86 (3.12–4.78)
Eczema	2.04 (1.63–2.55)
Hives	1.34 (1.09–1.65)
Allergy to foods or medicines	1.70 (1.26–2.30)
Maternal asthma	2.63 (2.08–3.31)
Paternal asthma	2.52 (1.99–3.19)
Maternal smoking	1.26 (1.05–1.51)

WRONG

Table 2 fallacy!!!

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A snapshot of current practice in clinical research

Pediatrics Zsp258=@Mwyi$$Nyri$5466-

- 18 research articles
- 11/18 report results based on regression analysis
  - Others: 6/7 descriptive aims (2 purely qualitative), 1/7 RCT
- Of the 11:
  - 2: essentially descriptive (trends over time)
  - 1: time trends compared between "groups"
  - 4: regression to estimate a causal effect controlling for confounders
  - 4: "investigate associations", "identify risk factors"…
- CLAIM: purpose (and therefore value) of 4-5 (of 11) uses of regression analysis are questionable

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Regression abuse

Claim: regression models are poorly understood by most (non-statistician) users…

- "Exploring risk factors…": data-driven regression modelling seen as valid approach for illuminating cause and effect
  - E.g. from an anonymous reviewer (*J Cystic Fibrosis*)

  "It might also be interesting to include some multiple regression models with various health [markers] predicting QoL scores in the same model to understand the relative contribution of each marker on QoL."
- "Adjustment" = statistical magic to ensure quality of conclusions?

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Regression models: what are they?

- Represent the variation in a "response" or "outcome" as "systematic + random" or "smooth + error"

$$Y = \beta_0 + \beta_1 X + \epsilon$$ - Simple, univariate

$$Y = \beta_0 + \beta_1 X_1 + \cdots + \beta_p X_p + \epsilon$$ - Multiple, multivariable

- Why are these models so compelling, but poorly used and understood?
- Can we find clues in the way we train people to use them…?

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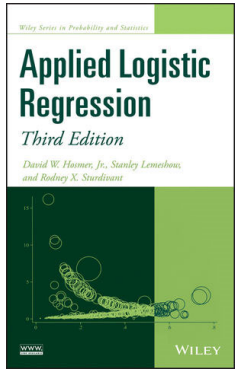
How do we (currently) teach regression?

- Classic texts

e.g. Hosmer & Lemeshow (1989, 2000, 2013)

From the Introduction:

"Before beginning a thorough study of the logistic regression model it is important to understand that the goal of an analysis using this model is the same as that of any other regression model used in statistics, that is, to find the best fitting and most parsimonious, clinically interpretable model to describe the relationship between an outcome (dependent or response) variable and a set of independent (predictor or explanatory) variables."



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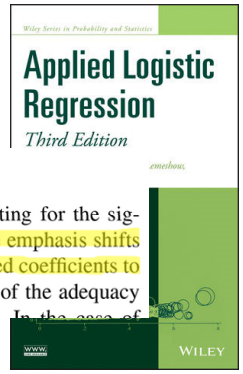
How do we (currently) teach regression?

Contents



3.1 INTRODUCTION

In Chapters 1 and 2 we discussed the methods for fitting and testing for the significance of the logistic regression model. After fitting a model the emphasis shifts from the computation and assessment of significance of the estimated coefficients to the interpretation of their values. Strictly speaking, an assessment of the adequacy

NO!!
We should interpret the coefficients *before* fitting the model

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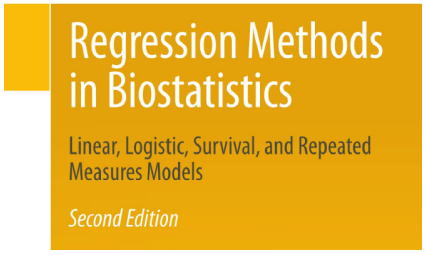
How do we (currently) teach regression?

- Contemporary texts

e.g. Vittinghoff et al (Springer, 2nd ed. 2012)



"The book describes a family of statistical techniques that we call *multipredictor* regression modeling. This family is useful in situations where there are multiple measured factors (also called predictors, covariates, or independent variables) to be related to a single outcome (also called the response or dependent variable). The applications of these techniques are diverse, including those where we are interested in prediction, isolating the effect of a single predictor, or understanding multiple predictors."

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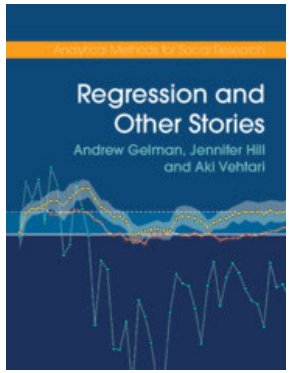
How do we (currently) teach regression?

Gelman, Hill & Vehtari
Regression and Other Stories (2020)

- Emphases the importance of purpose and tentativeness of models, but still ambiguous about whether the model or the purpose comes first



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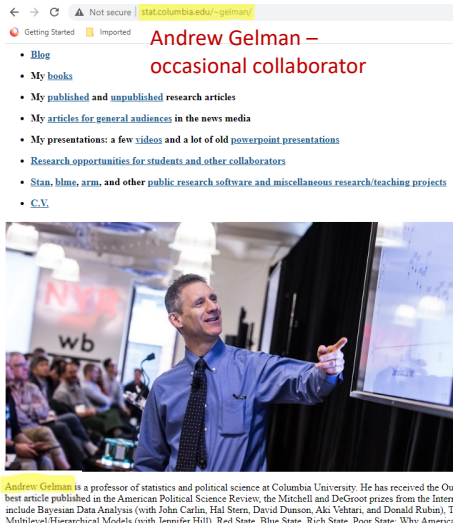
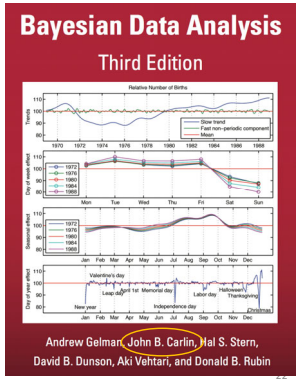


Andrew Gelman – occasional collaborator

Am I a Bayesian?

- Models are everything?



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How do we (currently) teach regression?

- First define the general model,

$$Y = \beta_0 + \beta_1 X_1 + \cdots + \beta_p X_p + \epsilon$$

  - Establishes mathematical framework with clear notation
- Then discuss applications…
- This feeds the **true model myth**, that *lurking within every dataset is an underlying "true model" that we should find…*
  - Seductive – mathematical facts can be claimed AND scientific conclusions may be possible, IF the model is true
  - Students internalise that once you have the "correct" model everything else follows…

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Regression models: why so important?

Two possibilities?

1) Natural phenomena follow "laws" that can be captured by regression models…
   - N.B. Assumes all variables measured and the model identifiable in whatever finite sample you might have!
   - Could then determine the "independent effect" of each variable on *Y*?
   - But surely this is never true of health and disease in populations, which are complex, with a lot of variability…
2) Regression models provide useful tools to study aspects of phenomena…
   - What exactly do we mean? How?
   - To answer, we need to revisit the purpose of statistical analysis!

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Three types of research question

Hernán et al (*CHANCE*, 2019)
"three tasks in data science: description, prediction, counterfactual prediction"

- Descriptive
  - Summarising and describing phenomena
- Prediction
  - Turning inputs into output: if we measure *a*, *b*, *c*, *d*, what value of *Y* should we expect?
- Causal
  - What value of *Y* should we expect if we change input *X*? (counterfactual prediction)

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Three purposes for regression methods

- Descriptive: simple ("univariate") regression *describes* average rate of change in *Y* with one-unit change in *X*
- Predictive: multiple regression seems a good place to start, with inputs/predictors the "independent variables"
- Causal: role for regression not so immediately obvious…? (the "effect of *X* on *Y*"?)

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Three examples revisited: three purposes?

Example 1

- Question:

How much is child's kidney enlarged after acute infection?

- Use of regression:

Estimate (mean) difference between infected and not, adjusting for age and sex

DESCRIPTIVE

Example 2

- Question:

Can clinical factors predict successful gas enema for intussusception?

- Use of regression:

Logistic regression "to determine which variables were predictive… forward selection procedure was used…"

PREDICTIVE (but…)

Example 3

- Question:

Estimate "strength of association" of numerous factors with risk of childhood asthma

- Use of regression:

"Relationships between [Y] and explanatory variables $[X_1, X_2, \ldots]$ were analysed by multiple logistic regression"

CAUSAL??

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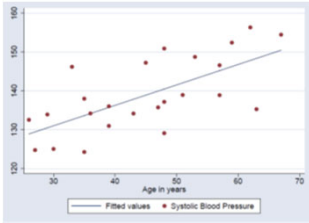
How should we teach regression?



Simple linear regression

- Scatterplot depicts (co-)variation of *Y* and *X*
  - Regression line *describes* average rate of change in *Y* with one-unit change in *X*
- Why not *X* vs. *Y*? Correlation or regression?
- Regression describes variation of *Y* (*on average*) as function of *X*
  - What form of function? *Straight line?* (may need more flexible functions…)
- For what other purposes could this be useful?
  - Simple *prediction*: if we only know *X*, what do we expect for *Y*?
  - *Causal inference*: not clear how??
- Finally, need statistical inference for rate of change, simple prediction (mean, individual value), etc

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How should we teach regression?

Multiple regression

Begin with purpose/questions, build theory as needed around these…

Introduce for each of the 3 purposes…

Descriptive purpose/question:

- Scatterplot *Y* vs. age for two groups (e.g. infected, not infected)
  - E.g. Example 1 (renal lengths)
- Regression as curve fitting/ smoothing
- May be useful to describe difference between groups using a simple model
- Inference for average difference between groups, adjusted? (for age, in example)
- All as exemplifying a general approach…

Fox MP et al, "On the Need to Revitalize Descriptive Epidemiology" *Am J Epi* 2022

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How should we teach regression?

Multiple regression: for prediction

- Multiple *X*'s available for *prediction* of *Y*
- Standard independent-predictors linear model ($Y = \beta_0 + \beta_1 X_1 + \cdots + \beta_p X_p + \epsilon$) a useful starting point
- Strategies for building models
  - Emphasise dependence on sample size!
  - Selection of variables, considering interactions, non-linearities etc
- Validation (internal/external)
- Coefficients not useful!! Traditional inference (tests) not useful!
  - Instead need to consider measures of prediction accuracy etc.

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How should we teach regression?

Regression for causal inference

- First requires clarity of purpose: what are we seeking to estimate?
- Potential outcomes & target trial: define the causal effect as the true difference we would obtain in the ideal study (a perfect infinite RCT)
  - Difference in means, risk difference, RR, OR, …
  - Key *statistical* issue: the target parameter!
- Then map from the ideal to the actual study, invoking causal assumptions…
- A regression model for the outcome *may* enable us to estimate the target effect… how?

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Regression for a causal purpose: how?

- Begin by defining the target parameter…

  i.e. causal effect (estimand) of interest…

  = (say) Mean difference in a continuous outcome Y:

  $$\delta = \mu^{(1)} - \mu^{(0)}$$

  where $\mu^{(x)} = E\left(Y^{(x)}\right)$ = mean value in population under treatment/exposure condition x $(= 0,1)$

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Regression for a causal purpose: how? (RCT)

- **Suppose target trial is feasible to conduct as a real RCT**
- If a **perfect** RCT, then the target parameter is (trivially) *identified* as

$$\delta = E\left(Y^{(1)}\right) - E\left(Y^{(0)}\right) = E(Y|X=1) - E(Y|X=0)$$

- … which in turn is unbiasedly *estimated* by

$$\hat{\delta} = \hat{E}(Y|X=1) - \hat{E}(Y|X=0) = \bar{Y}_1 - \bar{Y}_0$$

- Inference for $\hat{\delta}$?
  - Can get from standard "*t*-test", or *equivalently* using regression estimation (`regress y i.trt` in Stata-speak)

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Regression for causal purpose (beyond RCT)

- **If target trial not feasible**: observational study (or an 'imperfect' RCT) may emulate it
- Target parameter (estimand) remains the same but is no longer identifiable without *causal assumptions*
  - Encode these in a causal diagram (DAG), which guides analysis plan that minimises biases
  - In particular, must try to control confounding…
  - …by standardisation (g-computation, weighting etc), or conditioning (blocking back-door paths)
- Conditioning = holding confounders (***Z***) constant: regression is one approach…

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Regression for causal purpose (beyond RCT)

- How does regression provide control of a confounder?
- Need assumptions! Causal identifiability (consistency, exchangeability, positivity) ⇒

$$\delta = E_Z(E(Y \mid X=1, Z=z) - E(Y \mid X=0, Z=z))$$

Now, *IF* (we further assume) $E(Y \mid X=x, Z=z) = \beta_0 + \beta_1 x + \beta_2 z$

THEN $E(Y \mid X=1, Z=z) - E(Y \mid X=0, Z=z) =$

$= (\beta_0 + \beta_1 + \beta_2 * z) - (\beta_0 + \beta_2 * z) = \beta_1$

So, under these assumptions,

$\delta = \beta_1$ = difference in means between exposure groups *at every value of Z…*

= desired target effect (*IF the effect is indeed constant for all z*)

`regress y i.trt z` in Stata-speak

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Regression for causal purpose (beyond RCT)

Extends in two ways:

1. Control for multiple confounders

$$E(Y \mid X=x, Z_1=z_1, Z_2=z_2, \ldots) = \beta_0 + \beta_1 x + \eta_1 z_1 + \eta_2 z_2 + \cdots$$

- N.B. the equation now encodes many more assumptions:
  - Effect is constant across all strata of the confounders!
  - Default linear specification for non-categorical confounder effects

2. Apply to different target estimands

- Risk ratio: log-link regression ("GLM") for binary ("binomial") outcome
- Odds ratio (if you must!): logistic regression

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The causal revolution

- Upswell of interest in development of methods among biostatisticians
  - Newer “g-methods” also use regression models within them
- Standard practice lags behind
  - Yet to recognise that majority of published research addresses causal questions
  - Statistical tradition largely responsible: “correlation does not equal causation”…

  Hernán, M. A. (2018). "The C-Word: Scientific Euphemisms Do Not Improve Causal Inference From Observational Data." *American Journal of Public Health* **108(5): 616-619.**
- In summary, most published data analysis both:
  (a) addresses causal questions and (b) uses regression analysis…
  BUT with limited clarity of purpose or method!
- Biostatisticians should take responsibility for improving the standards of analysis planning and interpretation!

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Regression for causal purpose: done wrong…

An unfortunate but still common approach:

- Fit multiple regression model using all “risk factors of interest”, including “adjustment for covariates”
- Present estimates of coefficients (after variable selection) as “effects mutually adjusted for each other”

THIS HAS NO LOGICAL BASIS!

- The “Table 2 fallacy”

  - Greenland, S. and D. Westreich (2013). "The Table 2 Fallacy: Presenting and Interpreting Confounder and Modifier Coefficients." *American Journal of Epidemiology* **177(4): 292-298.**

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Assoc. Prof. Margarita Moreno-Betancur

LEAD INVESTIGATOR

Australia

Margarita is co-lead of the Clinical Epidemiology and Biostatistics Unit (CEBU) at the MCRI and the University of Melbourne. Since completing her PhD in Biostatistics at Université Paris-Sud in 2014 she has developed an integrated program of methodological and collaborative research, supported by an NHMRC Investigator Grant (2022-26) and previously an ARC DECRA (2019-22). Her methodological areas of interest are causal inference, missing data and survival analysis, and she has contributed to epidemiological research projects in a range of areas, particularly in life course and social epidemiology in her role as co-convenor and methodology lead of the Melbourne Children's LifeCourse Initiative.

Find out more about Margarita's work:

https://moreno-betancur.github.io/

twitter: @_MargaritaMB

Institutional profiles:

University of Melbourne

Murdoch Children's Research Institute

A major contributor to my recent thinking

Responsible for many of the ideas presented, in particular the terms

- “true model myth”
- “regression abuse”

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Concluding messages

- Extensive reform needed in the practice and teaching of regression methods in biostatistics, epidemiology and health science!
- Regression is not a method for “fitting models” but a tool for answering questions
  - “All models are wrong, but some models are useful…”
    (George Box, 1970s)
  - Useful for what?
    Until the purpose is defined, the construction of models should wait!

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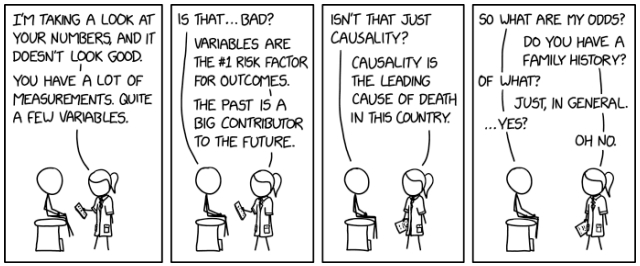
The causal revolution



We can do better than this!!

https://xkcd.com/2620/

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