# A Methodologic Assessment

## from a Social Epidemiologist


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# Today's Talk

Key Findings from a methodologic review

- A robust literature – on par with cigs and cancer
- Compelling evidence for good and bad effects
- Exposure measurement is good, and will get better
- Outcome measures are diverse J

# Today's Talk

Key Findings from a methodologic review

- Effect modification by recognized groups (measures?)
- Contextual effects
- Causal inference in observational studies
- Too few field interventions

# Effect Modification

**Effect estimates vary by given characteristics**

Stratify or estimate regression “interactions”

# Contextual Effects

Contexts shape (psycho-social) reaction to stimuli

Pre-disposing and fundamental causes (not proximal)

Cannot estimate independent contexts effects w/ regression models, including so-called multilevel model!

# Causality?

A rooster's crow predicts the sunrise,
but does not cause it

# Counterfactual Causality

# Counterfactual Causality

| Subject | Tx | Outcome |
|---|---|---|
| Ellen | BAD | 6 |
| Ellen | GOOD | 10 |

**The causal effect of Tx (on Ellen) is 10-6 = 4**

# Counterfactual Causality

| Subject | Tx | Outcome |
|---|---|---|
| Ellen | BAD | Poor |
| Ellen | GOOD | ? |

Counter-
factual!

**Cannot observe counterfactual/potential outcome**

**It's literally a <u>missing data</u> problem**

# Counterfactual Causality

| Subject | Tx | Outcome |
|---|---|---|
| Ellen | Good | Good |
| *P* | Bad | Bad |

Does good media Tx cause good outcomes?

Only if **Paris** is a good <u>substitute</u> for **Ellen!**

# Counterfactual Causality

The substitution step is the most important.

Good substitutes are "exchangeable" with the unobservable counterfactuals

Bias occurs when substitutes are not exchangeable with unobservable counterfactuals

# Randomization

Randomizing yields exchangeable comparison group to substitute for unobservable counterfactuals

**This is why randomization eliminates confounding in expectation**

# Observational Studies

In experiments we theorize from cause to effect
(from known intervention to outcome)

If we intervene and randomize, we're in good shape.
But simply intervening (quasi-experiment) is also helpful.

Absent intervention,
we must theorize from effect to cause which is not easy,
especially in presence of confounding.
Results could be due to anything and what of unmeasured things?

Appreciate that all observational studies
have an analog in both
counterfactual reasoning and experimental design.

# Propensity Scores

**Propensity Score: The probability of exposure**

In simple two-arm experiments, prob(E) = 0.50

Since everyone has same prob(E),
subjects are exchangeable and thus
good counterfactual substitutes.

# Propensity Scores

In actual pscore analyses, one models EXPOSURE not outcome/dependent variable… No gaming!

The predicted probability of exposure (often from logistic regression) is the propensity score.

Persons with same pscore are assumed to be exchangeable/substitutable.

Thus, match on pscore!

# Propensity Score

Rubin – If complete separation in PS? “You can say nothing about causal effects.”

Rosenbaum – Sharply distinct treatments that could happen to anyone.

If your substitute (ie, comparison group) does not reflect treatment group, then all inferences are based on (off-support) model assumptions.

# IV

## Aim to address <u>unmeasured</u> confounding

Key link to natural experiments

Find a variable (instrument) the predicts EXPOSURE but is unrelated to outcome (ie, disturbances). Estimate with 2SLS.

Most estimates are ITT (assignment)
Pscore matching yield ATT/TOT (uptake)
IV estimates are LATE (instrument)

* Caution: poor instruments often make things worse *

# CV

## Cross validate models – the only True test!

Fit your best model in one sample/data set.

Take estimated equation (ie, model) and use it to literally predict outcomes in another (independent) sample.

If your model is good, observed and predicted outcomes ought to be reasonably close.

Box's analogy to criminal investigation.

# Longitudinal Designs

Good for discrete random exposures

Good for appreciating natural (disease) progression

Not so good if exposures are chosen

Not so good for "over-determined" exposures

Not so good if confounders vary with time (MSMs?)

Is our interest in

Robinson Crusoe

or someone in a
social setting?

# No Interference

## SUTVA

"Treatment" only affects targets,
no interactions allowed.

Social dynamics / disequilibria?

# Field Experiments?

- Too few, but the best/only way to see what affects what
- Messy, but that's the point

# GRTs

Randomize things to <u>groups</u>

**Canonical design for social interventions**

**Account for – even hope for! – interactions**

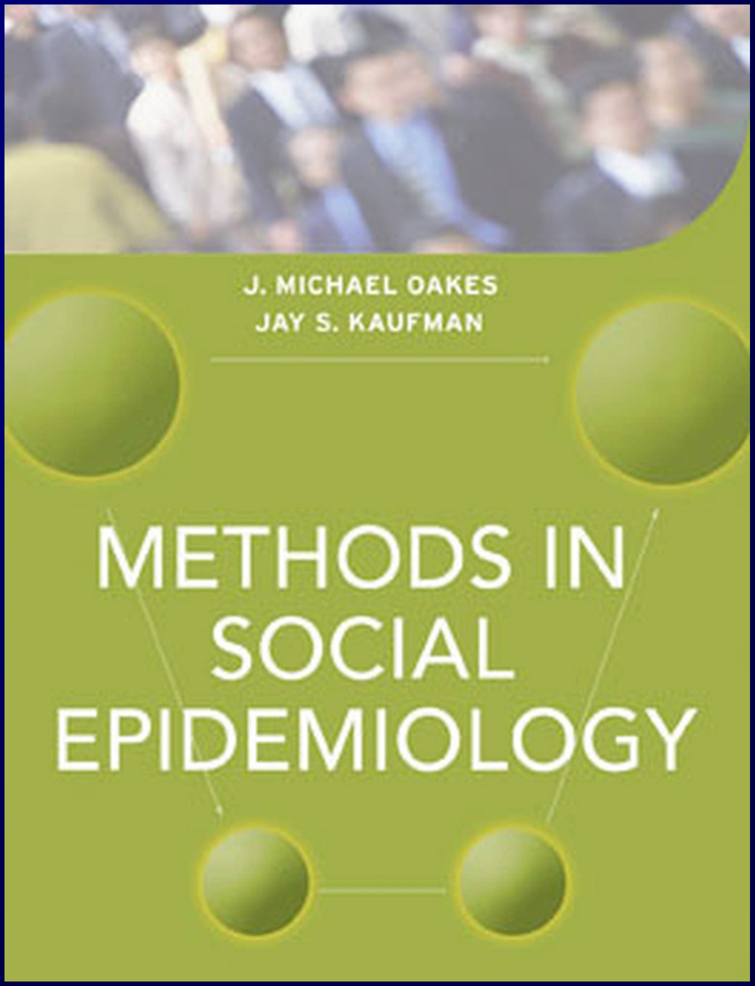
**It's NOT contamination!**

**Help the social process (eg, norms) work!**

# Community Trials

**Downside:**

- VERY expensive
- Tons of work
- Not everything is manipulable
- Ethics
- Poor historical success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METHODS IN SOCIAL EPIDEMIOLOGY