


CAFOs: WHAT'S ALL THE FUSS ABOUT PUBLIC HEALTH?


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WHAT'S AN ENGINEER DOING MESSING WITH PUBLIC HEALTH?



THE SKEPTICS' GAMBITS

- ◆ "Wolf!"




THE SKEPTICS' GAMBITS

- ◆ "Wolf!"
- ◆ "Fool me once...fool me twice"




THE SKEPTICS' GAMBITS

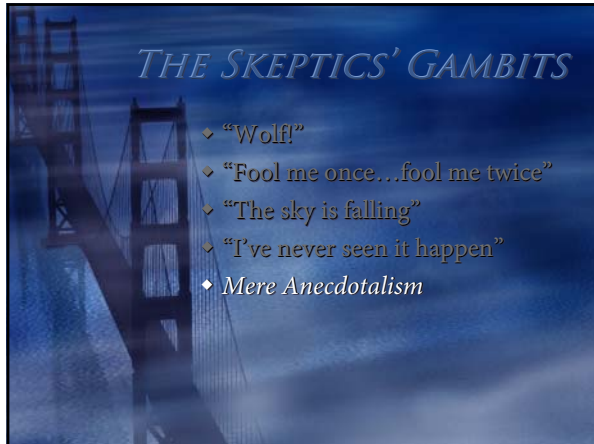


- ◆ "Wolf!"
- ◆ "Fool me once...fool me twice"
- ◆ "No, seriously, this time I really mean it"

THE SKEPTICS' GAMBITS

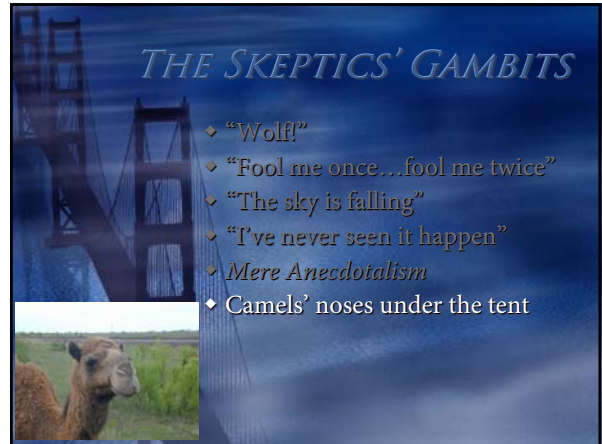
- ◆ "Wolf!"
- ◆ "Fool me once...fool me twice"
- ◆ "The sky is falling"
- ◆ "I've never seen it happen"





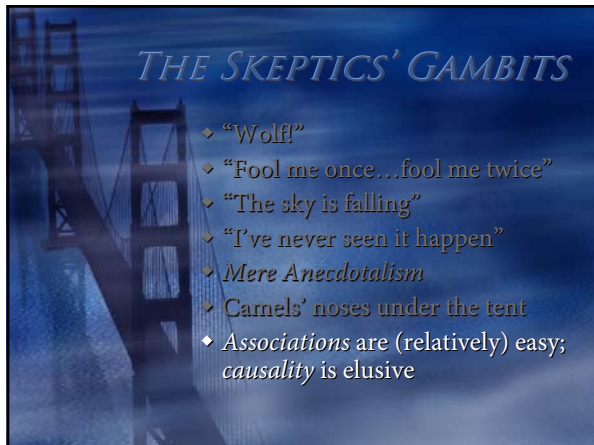
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- ◆ *Mere Anecdotalism*



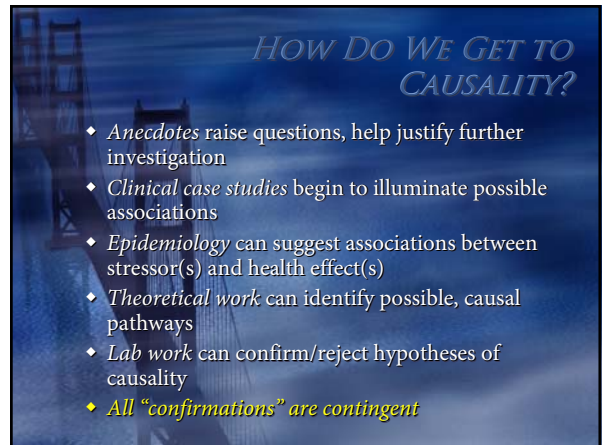
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- ◆ Camels’ noses under the tent



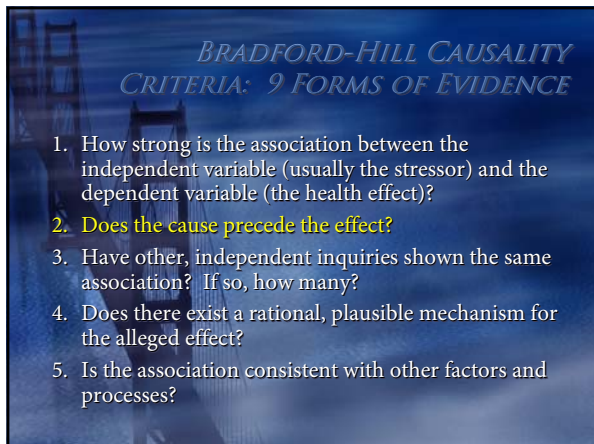
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- ◆ *Mere Anecdotalism*
- ◆ Camels’ noses under the tent
- ◆ Associations are (relatively) easy; causality is elusive



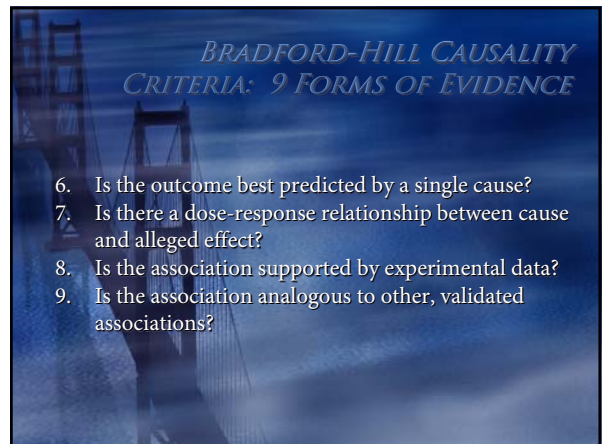
HOW DO WE GET TO CAUSALITY?

- ◆ *Anecdotes* raise questions, help justify further investigation
- ◆ *Clinical case studies* begin to illuminate possible associations
- ◆ *Epidemiology* can suggest associations between stressor(s) and health effect(s)
- ◆ *Theoretical work* can identify possible, causal pathways
- ◆ *Lab work* can confirm/reject hypotheses of causality
- ◆ All “confirmations” are contingent



BRADFORD-HILL CAUSALITY CRITERIA: 9 FORMS OF EVIDENCE

1. How strong is the association between the independent variable (usually the stressor) and the dependent variable (the health effect)?
2. Does the cause precede the effect?
3. Have other, independent inquiries shown the same association? If so, how many?
4. Does there exist a rational, plausible mechanism for the alleged effect?
5. Is the association consistent with other factors and processes?



BRADFORD-HILL CAUSALITY CRITERIA: 9 FORMS OF EVIDENCE

6. Is the outcome best predicted by a single cause?
7. Is there a dose-response relationship between cause and alleged effect?
8. Is the association supported by experimental data?
9. Is the association analogous to other, validated associations?

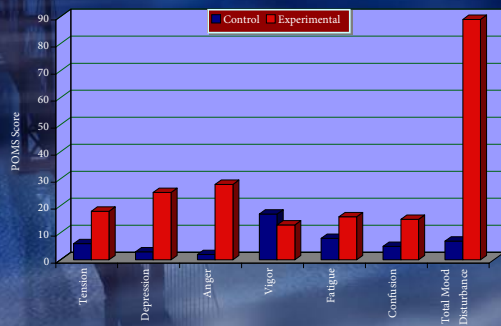
PHILLIPS AND GOODMAN (2004)

- ◆ Statistical significance should not be mistaken for evidence of a substantial association
- ◆ Association does not prove causation
- ◆ Precision should not be mistaken for validity
- ◆ Evidence of causation is not sufficient to suggest that action should be taken
- ◆ Uncertainty about causation is not sufficient to suggest that action should not be taken

Shusterman (1992)

“Any differential regulatory response to environmental odor pollution...based upon the distinction between community annoyance reactions and health effects *is a matter of legal, not scientific, interpretation.*”

Schiffman et al. (1995)



Thu et al. (1997)

- Health responses separated into 4 clusters
 - Respiratory symptoms ($P < 0.02$)
 - Nausea, weakness, dizziness, fainting ($P < 0.04$)
 - Headaches and plugged ears ($P < 0.06$)
 - Burning eyes, runny nose and sore throat ($P < 0.12$)
- “Little evidence to suggest” that anxiety or depression were elevated in CAFO neighbors

AREAS OF IMMEDIATE CONCERN

- Dust and NH_3 appear to have a 2- to 3-fold synergistic effect in large animals
- Endotoxin appears to be a major player
- Species differences apparent, but may simply be artifact of liquid vs. solid manure handling
- Bioaerosols demand greater attention; defense mechanisms may change exposure pathway
- Keep an eye on chronic, low-level H_2S
- Quasi-epidemiological studies of odor and public health are suggestive but need to be strengthened

Recommendations

- CAFO industry should not take psychological responses lightly
- Observed physiological associations are variable, but fairly compelling
- Researchers should pay close attention to the weaknesses in studies conducted to date
- Researchers need to avoid holding forth with too much certitude on associations that were not explored



Recommendations

- Studies need to account for the extent of exposure, not merely the presence of a stressor
- CAFOs should acknowledge (a) credible circumstantial evidence or (b) reasonable proposed causality as a basis for research