CAUSALITY & CORRELATION

ETHAN LIGON

1. Where does causality come from?

1.1. **How can we draw inferences regarding causality?** We can infer joint probability distributions over observable variables, but by themselves these probability distributions can tell us *nothing* about cause.

(1) No causes in... It immediately follows that cause *cannot* be inferred simply by reference to data. As philosopher Cartwright (1994) puts it in her critique of Bayesian networks, "No causes in, no causes out." (This is a position perhaps first taken by Hume, who framed "cause" as an idea.)

(2) Ragnar Frisch (1930) "The scientific answer to 'what is cause'... must read: it is such and such a way of thinking."

1.2. **Identification Distinct from Causality.** If data/probability distributions tell us nothing about causality absent a model, then it follows that the *analysis* of causality resides entirely in the specification and analysis of the model.

(1) Identification (Causal) Identification has to do with whether unspecified aspects of the model can be pinned down (at least in principle) by trying to make model predictions match things observable in TRW. The issue of identification is conceptually distinct from the issue of causality.

(2) Frisch (Again) A well-specified model implies a joint probability distribution over some set of variables. Then identification depends on the "correspondence between the model world of probability and the real world of frequency..."

1.3. **Notation for Causality.** Structural approach in economics encodes causality in its syntax and in conventions for labeling variables; i.e., in a model written as

$$y = f(x, u)$$

a contemporary economist would read this as "y is caused by x and u", even though the mathematical equation itself carries no such causal implication.

2. Four Cs

2.1. **Correlation does not imply causation.** Consider the regression equation

$$y = a + bx + e,$$

with e *defined* by the condition that $\mathbb{E}(e|x) = 0$; for simplicity assume $\text{var}(x) = \text{var}(y)$.

(1) Marginal Causal Effect? It's tempting to define the 'marginal' causal effect of x on y as $\partial y / \partial x = b$, the least squares regression coefficient. But these are random variables!
(2) Reverse Regression Consider the 'reverse regression'

$$x = c + dy + e'$$

Given our assumptions about equal variances, estimating this yields $d = b$. So which way does the causation go?

2.2. **Conditioning does not imply control.** Consider the regression equation

$$y = a + bx + e,$$

with e *defined* by the condition that $\mathbb{E}(e|x) = 0$. Then we can define the conditional expectation $\mathbb{E}(y|x) = bx$.

(1) Careful! It's tempting to read this regression causally, inferring that a one unit increase in x implies a b unit increase in y. But we're conditioning on a random variable! We can't just go adding numbers to random variables. Even if we can change the 'location' of the *marginal* distribution of x what effect does this have on the joint distribution of (x, y)?

3. Marshall, Frisch, Haavelmo, & the Cowles Commission

3.1. **Structural Models (Cowles Commission).** A structural model can be expressed as a triple (U, V, F), where:

- U is a set of *exogenous* variables;
- V a set of *endogenous* variables; and
- F is a set of functions such that $V_i = F_i(U, V_{-i})$.

3.2. **Causality in Structural Models.** Note that this is entirely a property of the model! Frisch (1930) observes that "the main aspect of of the problem of scientific causality is the *direction* of causality."

(1) Direct cause A variable $X \in U \cup (V \setminus Y)$ is a *direct cause* of Y if it appears in the function describing Y, i.e., $Y = F_Y(X, \cdots)$.
(2) Cause A variable $X \in U \cup (V \setminus Y)$ is a *cause* of Y if it directly causes either Y or any other cause of Y.

3.3. **Graphical Models (Pearl).** Every structural model is associated with a directed graph, which summarizes causal relationships.

- A directed graph is is comprised of *nodes* and *directed edges*.
- The graph corresponding to a structural model (U, V, F) will have a node for every variable in $U \cup V$.
- If, in the structural model, a variable X directly causes a variable Y then there will an edge directed from X to Y.

3.4. **The Canonical Model of Economics: Demand & Supply.** An example of a *structural* model of demand & supply. A market equilibrium exists if there exists a pair (p^*, q^*) such that

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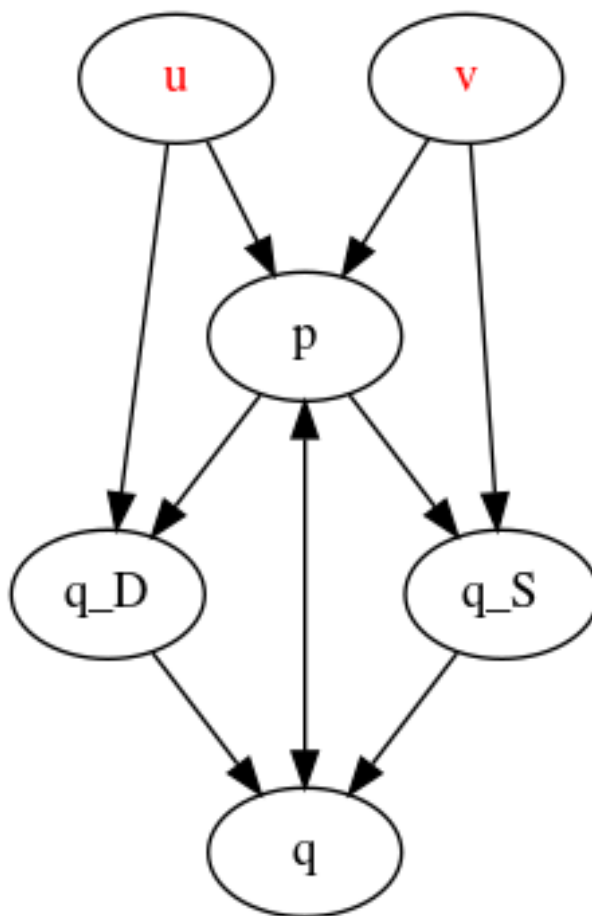
(2) Left column

$$q_D = q_D(p, u) \Big\}$$
$$q_S = q_S(p, v) \Big\}$$

and markets clear,

$$q_D(p^*(u, v), u) = q_S(p^*(u, v), v) = q^*(u, v).$$

Model is closed (or "completely specified") if we specify the functions (q_D, q_S) and the distributions of u and v, say F_u and F_v.

3.5. **Some Questions Answered by the Canonical Model.**

Control: What is the expected demand if we *set* the price $p = p_0$?

Observe: What is the expected demand if we *observe* $p = p_0$?

Counterfactual: If prices and quantities are observed to be (p_0, q_0), what **would** demand be if we **were** to *change* the price to p_1, *ceteris paribus*?

(1) NB: "Observed" implicates identification

3.6. **Controlling Price.**

(1) Right column Expected demand if we *set* price to p_0, no longer treating price as a random variable. Note that *setting* the price has no effect on the distribution of u, so:

$$\mathbb{E}(q_D(p_0)) = \int q_D(p_0, u) dF_u(u).$$

(2) Left column

3.7. **Observing Price.**

(1) Right column Expected demand if we *observe* a realization of $p = p_0$:

$$\mathbb{E}(q^*|p = p_0) =$$

$$\mathbb{E}\left[q^*(u, v)|q_D(p_0, u) = q_S(p_0, v)\right].$$

Given that we observe, say, a high price p_0 the *conditional* expected value of u will also be higher, and the conditional value of v will be lower (if q_D and q_S are monotonically increasing in u and v, respectively).

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3.8. **Price Change, *Ceteris Paribus*.**

(1) Right column If (p^*, q^*) are *observed* to be (p_0, q_0), what would demand be if we were to change the price to p_1, *ceteris paribus*?
   - If we observe $(p^*, q^*) = (p_0, q_0)$ we can maybe infer (u_0, v_0) (using, e.g., monotonicity). Nothing's random anymore! Then *ceteris paribus* we have the change

$$\Delta \qquad = \qquad q_D(p_1, u_0) \qquad - \qquad q_D(p_0, u_0).$$

Note that answer here doesn't depend on supply at all, except to infer u_0.

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4. Recursive Models

4.1. **Non-equilibrium models.** Our canonical supply & demand model is an *equilibrium* model. We saw that in a graphical description of this model there was a *cycle* corresponding to an equilibrium condition when we expressed the model in terms of equations; these are *simultaneous*.

(1) Different expressions of same idea
   - Recursive model; Cowles Commission

- Directed Acyclical Graphs (DAGs); Pearl et al
- Triangular models; Imbens & others
- Markovian models; Statistics

(2) Equilibrium models have cycles Thus can't be expressed exclusively in these terms.

4.2. **Nice results for recursive models.**

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4.3. **Recursive Model.**

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(2) Middle column
(3) Left column

5. MODERN APPROACHES TO CAUSALITY

5.1. **Introduction: Modern Approaches to Causality.** Three contemporary people from three different fields very influential in developing methods to draw causal inferences:

James J. Heckman/Economics (Heckman 2010): "Generalized Roy model", or generally a defender of the notion of causality embedded in "structural" economic models.

Donald Rubin/Statistics (Imbens and Rubin 2015): "Potential outcomes model" or "Neyman-Rubin model".

Judea Pearl/Computer Science (Pearl 2009): "do-calculus", "Structural causal model", graphical models.

6. RUBIN

6.1. **Idea of the Potential Outcomes Model (Neyman-Rubin).** Idea is that people (or "units") are "assigned" to two or more different groups—say a treatment (1) and a control (0). We are interested in an outcome for unit i conditional on this assignment; e.g., the pair (y_i^0, y_i^1).

"Causal effect of treatment": $y_i^1 - y_i^0$;

"Fundamental problem of causal inference" (Holland 1986): In this tradition "problem" is essentially missing data: we can't observe outcomes for unit i in both states.

This leads to methods to try to impute what missing data **would** have been had assignment been different. This means we need a counterfactual model!

7. Pearl

7.1. **Idea of the Structural Causal Model (Pearl).** Causal structure can be expressed in least restrictive fashion.

(1) Do-calculus Pearl develops economists' *ceteris paribus* idea expressed as his "do-calculus". Some nice formal results about identification in structural models.
(2) Directed Acyclical Graphs (DAGs) Analysis focuses on *acyclical* graphs. For this class strong results on non-parametric identification available. Implies that models with DAGs need *only* specify causal structure.

7.2. **Pearl's Inference Engine.** Pearl describes an "inference engine", which takes as inputs a model embedding causal assumptions (A), a set of questions (Q) that could be answered if one knew the exact form of the fully specified theoretical model, and a dataset (D). Outputs from the inference engine are a set A* of data-independent implications of the model; a set C of claims which *respond* to Q, depend on data D, and are true conditional on the model assumptions A.

7.3. **Stages in the Inference Engine.** There are three distinct activities involved in operation of the engine: (i) Model specification; (ii) Causal inference (identification); (iii) Statistical inference; and (iv) different sorts of conclusions, including conditional claims (C) and possibly model testing.