RECONSTRUCTING CAUSAL NETWORKS FROM DATA

- Overview and Developments (Part I)

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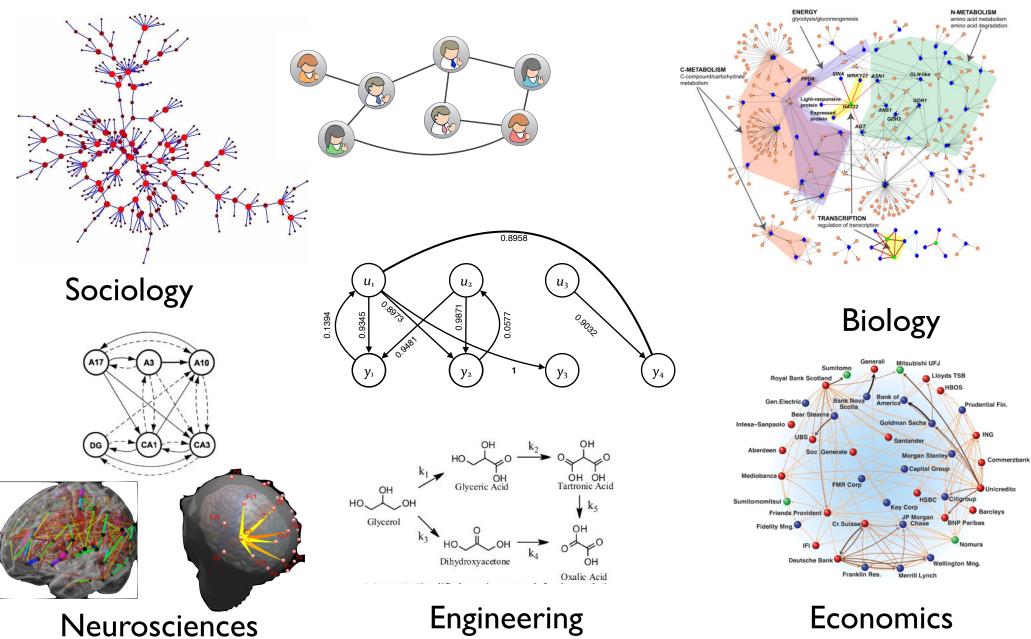
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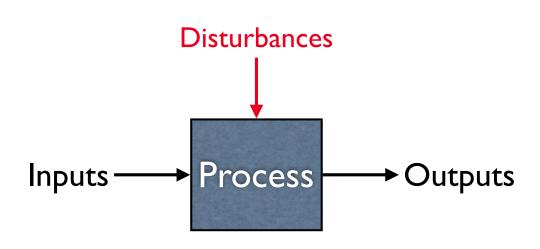
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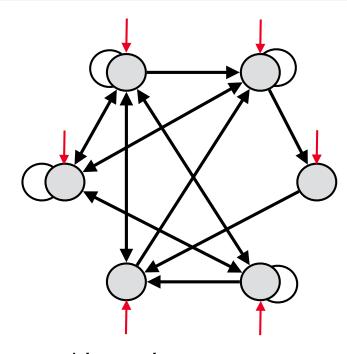
COMPLEX NETWORKS IN DIFFERENT FIELDS



AN IMMINENT PARADIGM SHIFT



Classical representation



Network representation

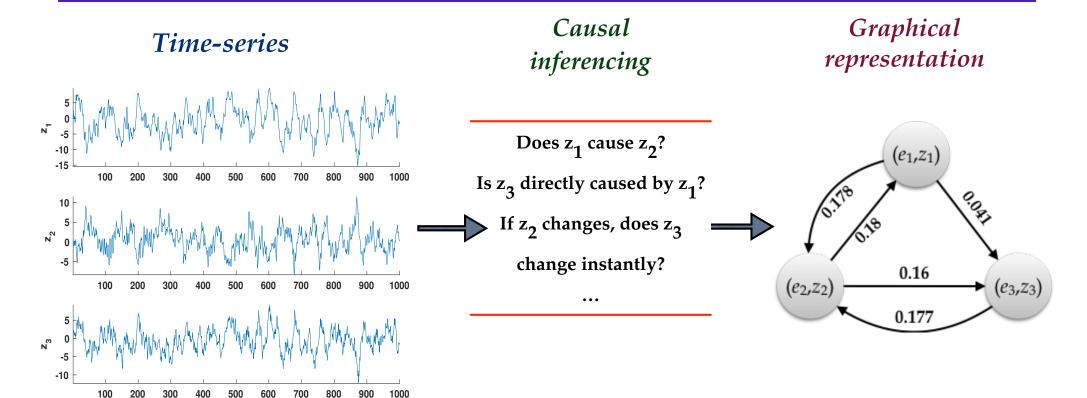
Benefits:

- Can handle broader class of systems and situations
- Offers new perspectives and paradigms for analysis of process systems

Challenge: Added layer of complexity in n/w representation.

Directionality of relations also have to be identified

DATA TO DIRECTIONALITY



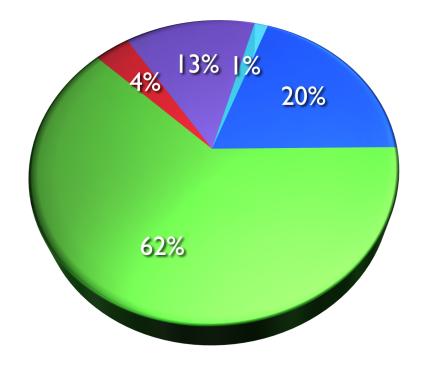
What does z_i "cause" z_j mean? How do we infer such causal relationships? Concepts, assumptions, methods, inferences, etc.? What do these graphical model encode?

Sample time

RESEARCH ACTIVITY

- A quick search of "causality" and "time-series" results in ~ 0.3M results.
- On "Granger causality", one observes nearly 0.07M search results.



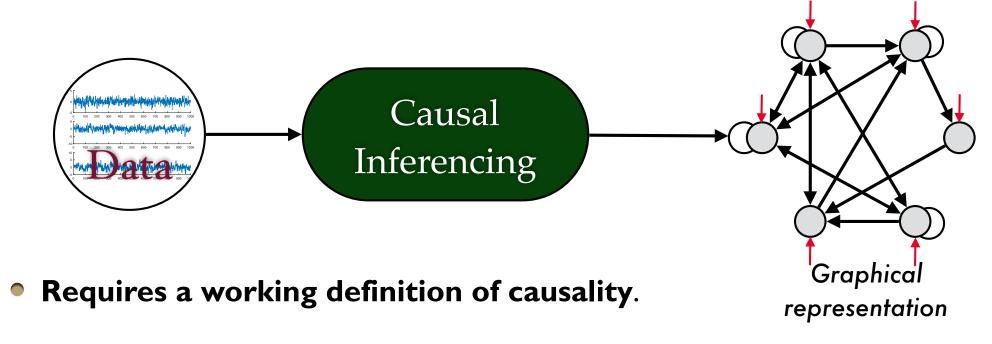


ROAD MAP

- Historical Developments: From Econometrics to Physical Sciences
- Formal Concepts
 - Causality
 - Wiener-Granger Causality
- Reconstruction of GC Networks for Stochastic Processes
 - ▶ Time-domain methods
 - ▶ Frequency-domain methods

PROBLEM STATEMENT

Given multivariate time-series, identify the "causal" relations between the (measured) signals



- A method for inferring causality from data
- Formalism of encoding causal inferences in a graphical form

DIFFERENT FLAVOURS

- Determine causal relations only
 - Structure determination (Boolean problem)
- Determine causal relations and their strengths
 - Weighted causal network determination
- Determine causal and dynamical relations
 - Joint causality and modelling problem
- Advanced problems
 - ▶ Determine interconnection of networks in a multidimensional scenario
 - Determine causality under constraints and mixed (deterministic plus stochastic) processes.

WHY DATA-DRIVEN?

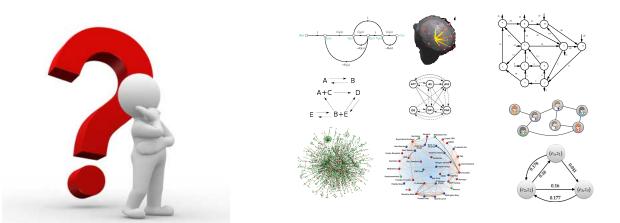
First-principles models are rarely available or in complicated forms

Difficult to translate first-principles models, flow sheets, PnID into graphical representations

Determining structure from data has three important advantages:

- I. Can calculate strengths of connectivity:
 - 1. Knowing the intensity and characteristics of a coupling can only be determined experimentally
 - 2. A data-driven approach can give us the **effective connectivity.**
- 2. Can account for stochastic effects and uncertainties
- 3. Facilitates inclusion of partial knowledge

KNOW YOUR APPLICATION / NETWORK!

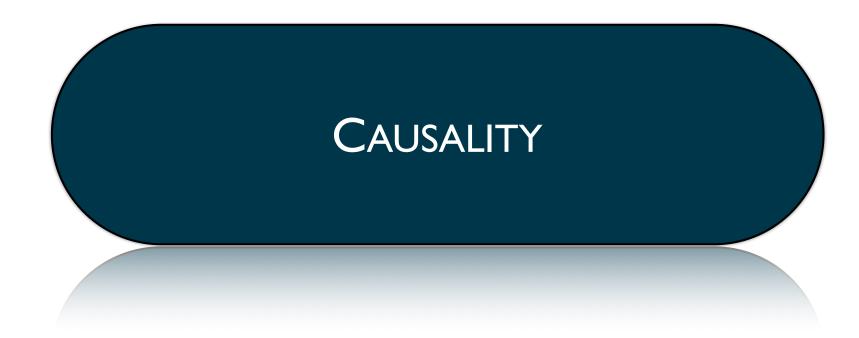


- **Type of network** (characteristics of nodes, edges, directed / undirected, etc.)
- Assumptions on process (stochastic, deterministic, dynamic, etc.)
- Information encoded (independence, conservation laws, causality, etc.)
- Ability to reconstruct from data (uniqueness, confounding, etc.)
- Type and amount of data required (steady-state, time-series,
- Computational burden

•

SCOPE OF THIS TALK

- Multivariate discrete-time stochastic processes
- Jointly stationary
- Linear / non-linear processes
- Granger-causal networks



CAUSALITY

Causality is a physical concept based on the cause-effect relationship

Three different forms of causality: Strict, instantaneous and feedback

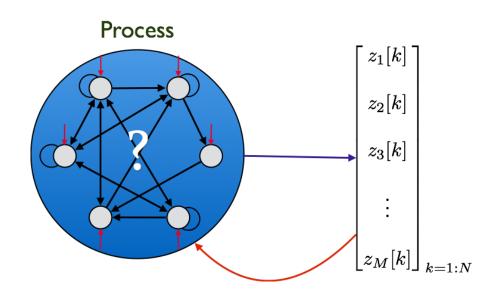
- Strict/Lagged: $z_2[k] = az_1[k-l]$
- ② Instantaneous:

$$z_2[k] = a_0 z_1[k] + a_1 z_1[k-l]$$

Seedback/Bidirectional:

$$z_2[k] = az_1[k - l_1]$$
 and $z_1[k] = bz_2[k - l_2]$

Can we detect all three forms of causality from data?



DIRECT OR INDIRECT?

• Example:

$$z_1[k] = a_{11}z_1[k-1] + e_1[k]$$

$$z_2[k] = a_{21}z_1[k-1] + a_{22}z_2[k-1] + e_2[k]$$

$$z_3[k] = a_{32}z_2[k-1] + z_{33}z_3[k-1] + e_3[k]$$

- Does $z_1[k]$ cause $z_3[k]$?
- As per Granger causality, NO.
 As per Sims causality, YES.

Does it mean we have a contradiction?

Clearly, a working definition of causality is needed for practice - but we can expect no definition to be complete in all respects!

MOTIVATING A DEFINITION OF CAUSALITY

 Determining "cause of an effect" is obviously much more difficult than studying "effect of a cause"

Even more challenging is answering "what is meant by a cause?"

- Most of the literature is devoted to studying "effect of a cause"
- Does it mean we can never determine if X causes Y?

Yes, we can, but in an indirect (back-door) way.

Assume X to be the cause and determine if it affects Y.

Challenge: X could affect Y in many ways - its amplitude, or mean, or variance, or distribution, etc.

A QUOTE

Granger (1980) - Testing for causality: a personal viewpoint

"Attitudes towards causality differ widely, from the defeatist one that it is impossible to define causality, let alone test for it, to the populist viewpoint that everyone has their own personal definition and so it is unlikely that a generally acceptable definition exists. It is clearly a topic in which individual tastes predominate, and it would be improper to try to force research workers to accept a definition with which they feel uneasy. My own experience is that, unlike art, causality is a concept whose definition people know that they do not like but few know that they do like. It might therefore be helpful to present a definition that some of us appear to think has some acceptable features so that it can be publicly debated and compared with alternative definitions."

A FEW IMPEDIMENTS

- Early literature focussed on deterministic situations
 - ▶ *If X occurs, then Y should occur (necessity)*
 - ▶ *If Y occurs, then X must have occurred (sufficiency).*
- Not a practically useful one in reality, our knowledge has uncertainties.
- Another challenge: causation can change with space and time
- Finally, will a statistical inference imply / agree with science?

AGAIN A QUOTE

Granger (1980) - Testing for causality: a personal viewpoint

"The philosophers and others have provided a variety of definitions, but no attempt to review them will be made here, as most are of little relevance to statisticians. Once a definition has been presented, it is very easy for someone to say "but that is not what I mean by causation." Such a remark has to be taken as a vote against the particular definition, but it is entirely destructive rather than constructive. To be constructive, the critic needs to continue and provide an alternative definition. What is surely required is a menu of definitions that can be discussed and criti- cized but at least defended by someone."

NOTIONS OF CAUSALITY

Formal and model-based:

- Intervention Causality (Eichler and Didelez, 2007)
- Structural Causality (White & Lu, 2010)

Empirical (data-based):

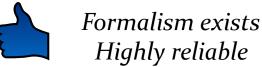
- Wiener-Granger Causality (Wiener, 1956; Granger; 1959)
- Akaike Causality (Akaike, 1968)
- Sims Causality (Sims, 1972)
- Convergent cross-mapping
- ...

CAUSAL INFERENCES

Two Broad Data Classes

Interventional Data

- User intervention
- Outcomes recorded for controlled perturbations in causal variables
- Randomized experiments





Infeasible for several processes

Observed Data

- No user intervention
- Outcomes recorded for natural / routine perturbations in causal variables
- Little to no control on excitation, levels of randomness, etc.

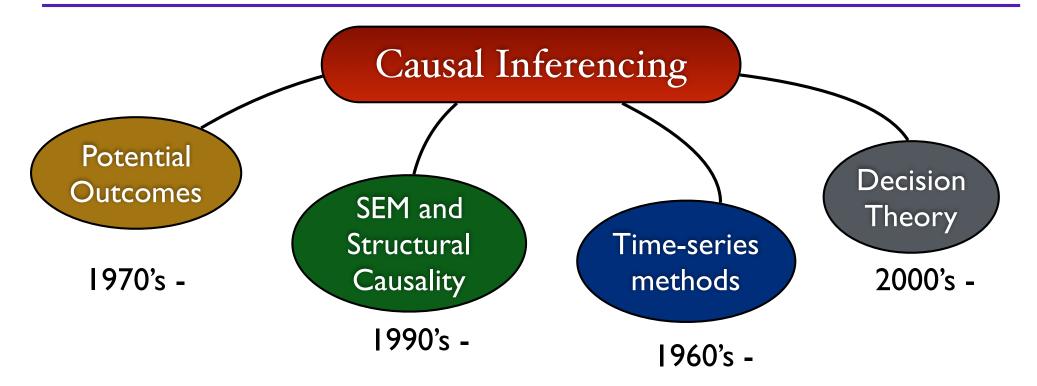


Highly practical Formalism exists



Less reliable Low generalisability

HISTORICAL DEVELOPMENTS



- Contributions from social sciences, econometrics, statistics, computer sciences, neuroscience and (recently) engineering
- Different notions, settings, prior knowledge, etc.
- Theory at its infancy. No unified, mature and comprehensive framework yet!

Read: 🔟

Causality - Statistical Perspectives and Applications (2012)

ISSUES AND CHALLENGES

- Is it possible to reconstruct networks uniquely from data?
 - ▶ Requires a formal problem formulation (structural identifiability?)
- Confounding
 - Unavoidable can minimize by choosing a sufficiently large control volume
- Assumptions
 - Deterministic / Stochastic nature, linearity, stationarity, . . .
- Strength of connectivity
 - ▶ Appropriate measure; estimation algorithms, significance levels, . . .
- Constraints (on variables)
 - ▶ Conservation laws; balance equations, . . .
- Limitations:
 - ▶ Similar to other data-driven techniques excitation, singularities of covariance matrix, . . .

WIENER-GRANGER-CAUSALITY

Two assumptions:

- cause precedes its effect
- 2 cause contains unique information about the future values of its effect

Introduce, $I^*[k]$: information set **up to** 'k', $I^*_{-zi}[k]$: information **excluding** z_i .

The series $\{z_i[k]\}$ does not (Granger) cause $\{z_j[k]\}$ if

$$z_j[k+1] \perp \!\!\! \perp \mathcal{I}^{\star}[k] \mid \mathcal{I}^{\star}_{-z_i}[k]$$

for all $k \in \mathbb{Z}$, else $\{z_i[k]\}$ is said to cause the series $\{z_j[k]\}$

Alternative definition / viewpoint:

- Granger non-causal if and only if **conditional distribution** of $z_j[k]$ is unchanged whether $z_i[k]$ is included or not in the information set.
- Granger non-causal if both sets contain ``same" information about $z_i[k]$.

WGC: FIRST RELAXATION

- Clearly, it is not possible to measure all variables relevant to $z_j[k]$ in the universe!
- A working definition that includes the available knowledge is required.

Suppose only the series z_i , z_j and ${f w}$ have been observed.

Then, the series z_i does not (Granger) cause z_j w.r.t. ${f v}=(z_i,z_j,{f w})$ if and only if

$$z_j[k+1] \perp \mathbf{v}^k \mid \{z_j^k, \mathbf{w}^k\}$$

for all $k \in \mathbb{Z}$, else z_i is said to Granger-cause z_i w.r.t. \mathbf{v} .

Tests for conditional independence are usually replaced with tests of prediction

WGC: PREDICTION-BASED DEFINITION

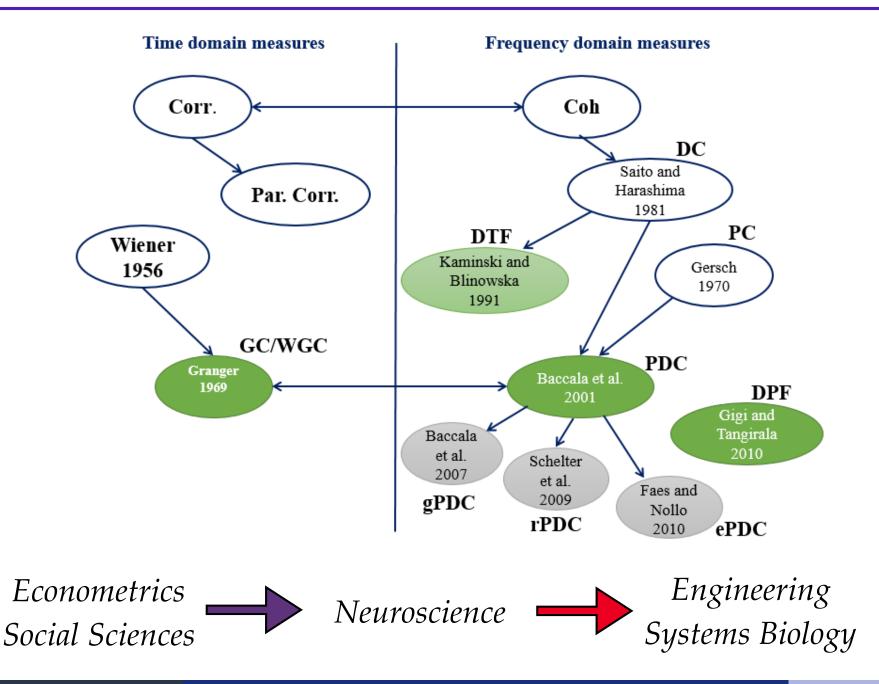
(Bivariate) Granger causality [Granger, 1969]

Suppose we wish to test if a random signal $z_1[k]$ Granger causes another random signal $z_2[k]$. Predict $z_2[k]$ using its own past and construct another forecast by incorporating past of $z_1[k]$. If the forecast is improved then $z_1[k]$ is said to "Granger cause" $z_2[k]$.

Extensions to multivariate, feedback and instantaneous causality are on similar lines.

The definition is not confined to a "method" of prediction!

Overview of Methods (for Testing GC)



GRANGER-CAUSAL GRAPHS

Let G=(V,E) be the path diagram associated with a stationary Gaussian process $\{\mathbf{z}[k]\}$

Then,

- 1. The directed edge $a \longrightarrow b$ is absent in the graph if and only if z_a is Granger-noncausal for z_b with respect to the full process \mathbf{z} .
- 2. The undirected edge a--b is absent in the graph if and only if z_a and z_b are not contemporaneously correlated with respect to the full process z.

• Example:

$$z_1[k] = 0.6z_1[k-1] + e_1[k]$$

$$z_2[k] = 0.9z_2[k-1] + e_2[k]$$

$$z_3[k] = 0.2z_1[k-1] + 0.5z_2[k-1] + 0.8z_3[k-1] + e_3[k]$$

