Causal Modeling

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Motivations

Artificial Intelligence / Machine Learning

A Case of Irrational Scientific Exuberance

- Underspecified goals — Big Data cures everything
- Underspecified limitations — Big Data can do anything (if big enough)
- Underspecified caveats — Big Data and Big Brother

Wanted: An AI with common decency

- Fair — no biases
- Accountable — models can be explained
- Transparent — decisions can be explained
- Robust — w.r.t. malicious examples

ML & AI, 2

In practice

- Data are ridden with biases
- Learned models are biased (prejudices are transmissible to AI agents)
- Issues with robustness
- Models are used out of their scope

More

- C. O'Neill, *Weapons of Math Destruction*, 2016
- Zeynep Tufekci, *We're building a dystopia just to make people click on ads*, Ted Talks, Oct 2017.

ML yields discriminative or generative modelling

Given a training set iid samples $\sim P(X, Y)$

$$\mathcal{E} = \{(\mathbf{x}_i, y_i), \mathbf{x}_i \in \mathbb{R}^d, i \in [[1, n]]\}$$

Find

- Supervised learning: $\hat{h} : X \mapsto Y$ or $\widehat{P}(Y|X)$
- Generative model $\widehat{P}(X, Y)$

Predictive modelling might be based on correlations

If umbrellas in the street, Then it rains

The implicit big data promise:

If you can predict what will happen,
then how to make it happen what you want ?

Knowledge $\rightarrow$ Prediction $\rightarrow$ Control

ML models will be expected to support *interventions*:
Intervention $do(X = a)$ forces variable X to value a

- health and nutrition
- education
- economics/management
- climate

The implicit big data promise, 2

Intervention Pearl 2009

Direct cause $X \rightarrow Y$ iff

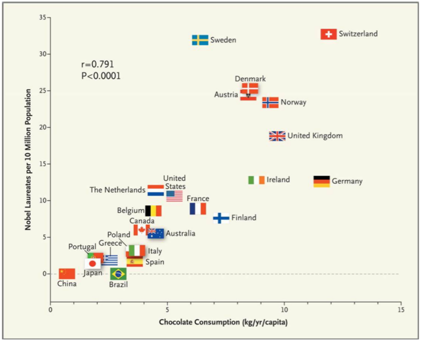
$$P_{Y|\mathrm{do}(X=a,\mathbf{Z}=\mathbf{c})} \neq P_{Y|\mathrm{do}(X=b,\mathbf{Z}=\mathbf{c})}$$

Example C: Cancer, S : Smoking, G : Genetic factors

$$P(C|do\{S=0, G=0\}) \neq P(C|do\{S=1, G=0\})$$

$$\begin{tikzcd} S \arrow[r] & C & G \arrow[l] \\ \mathit{Intervention} \arrow[u, dotted] & & \end{tikzcd}$$

Correlations do not support interventions



F. H. Messerli: *Chocolate Consumption, Cognitive Function, and Nobel Laureates*, N Engl J Med 2012

Causal models are needed to support interventions

Consumption of chocolate enables to predict # of Nobel prizes
but eating more chocolates does not increase # of Nobel prizes

Predictive model $\not\to$ Causal model

Consider

$$\begin{aligned} X, E_Y, E_Z &\sim \text{Uniform}(0,1), \\ Y &\leftarrow 0.5X + E_Y, \\ Z &\leftarrow Y + E_Z, \end{aligned}$$

with $E_Y, E_Z \sim \mathcal{N}(0,1)$ (noise)

Predicting Y

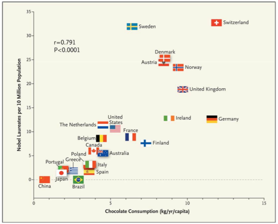
$$\widehat{Y} = 0.25X + 0.5Z$$

If interpreted as a causal model, suggests that Y depends on Z.

Issue

Causes can often be predicted from their effects

When correlations do not imply causality



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Country wealth

Chocolate consumption

Number of Nobel prizes

Tentative explanation: confounders

- Both effects of a same cause, $C \not\perp\!\!\!\perp N$.
- But **C** and **N** are conditionally independent given W

$$C \perp\!\!\!\perp N | W$$

Causality and paradoxes

Facts

- If mother smokes, child weight tends to be small
- Tiny child, more health problems
- However, tiny child AND mother smokes $>$ tiny child

Interpretation mother smoking beneficial to child's health ?

Explaining away
Many possible causes for small child weight
Many of these severely affect child's health (genetic diseases)
Compared to these, mother smoking is rather a good news...

An AI with common decency

Desired properties

- Fair — no biases
- Accountable — models can be explained
- Transparent — decisions can be explained
- Robust — w.r.t. malicious examples

Relevance of Causal Modeling

- Decreased sensitivity wrt data distribution
- Support interventions — clamping variable value
- Hopes of explanations / bias detection

Causal Discovery

HOW

- Gold Standard: perform randomized controlled experiments
- But these experiments are often costly, unethical or unfeasible
- Our setting: observational causal discovery
  From data, infer causal model.

WHAT FOR

- Understandable, interpretable, more robust models
- Prioritize confirmatory experiments: enabling some control
- Generate new data: privacy and domain-compliant, e.g. for medical training

Motivating applications

Human resources

1. Autonomy / Satisfaction / Productivity
2. Quality of life at work / Economic profitability of firms

Joint project with 'La Fabrique de l'industrie'

Kalainathan et al. 18

Health and Life habits

1. Diet / Diabetes type 2.

Joint project Nutriperso with INRA

State of the art

Causal Modelling

The Causal Discovery Setting

Assume random variables

$$X_1, \dots X_d : \text{ random variables}$$

and a sample of their joint distribution

$$\mathcal{D} = \{\mathbf{x}_i, i = 1 \dots n\}$$

to be given.

Formal background: Overview

1. Key concepts
2. Framework
3. Approaches

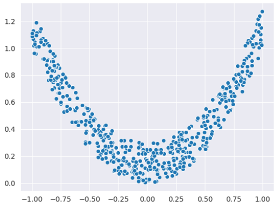
Key concepts: 1. Dependence among pairs of variables

Independent variables X and Y ($X \perp\!\!\!\perp Y$)

$$X \perp\!\!\!\perp Y \text{ iff } P(X, Y) = P(X).P(Y)$$

Dependency tests

- Correlation — limited to linear dependencies



$$Y = X^2 + E$$
$$Correlation(X, Y) \approx 0$$

Key concepts: 1. Dependence among pairs of variables

Independent variables X and Y ($X \perp\!\!\!\perp Y$)

$$X \perp\!\!\!\perp Y \text{ iff } P(X, Y) = P(X).P(Y)$$

Dependency tests

- Correlation — limited to linear dependencies
- HSIC, Hilbert-Schmidt Independence Criterion

Gretton et al. 05

$$HSIC(\underset{XY}{Pr}, \mathcal{F}, \mathcal{G}) := ||C_{XY}||^2$$

where $||\cdot||$ denotes the Hilbert-Schmidt norm, and C_{XY} a kernel based covariance operator and $\mathcal{F}, \mathcal{G}$ two RKHSs.

Key concepts: 2. Conditional Dependence/Independence

Conditional independence a.k.a. hidden confounder

Conditional dependence a.k.a. V-structure

$$
\begin{array}{ccc}
X = \text{Complex machine} & & Y = \text{Inexperienced Worker} \\
& \searrow \quad \swarrow & \\
& Z = \text{Accident} &
\end{array}
$$

X and Y are independent; but given $Z = \textit{true}$ they are not independent (either the machine is complex or the worker is inexperienced...)

Definition of causal relationship

Definition of intervention

$$do(X = 1) \text{ forces variable } X \text{ to value } 1$$

Pearl 09

Definition of causal relationship

X is a direct cause of Y ($X \rightarrow Y$) iff
all other variables Z being constant,

$$P_{Y|\mathrm{do}(X=1,\ldots,Z=c)} \neq P_{Y|\mathrm{do}(X=0,\ldots,Z=c)}$$

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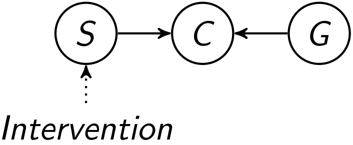


Markov equivalence class and V-structure

Markov Equivalent Class: $A \perp\!\!\!\perp C | B$ and $A \not\perp\!\!\!\perp C$

$A \rightarrow B \rightarrow C$ $\quad$ $A \leftarrow B \leftarrow C$ $\quad$ $A \leftarrow B \rightarrow C$

V-Structure: $A \not\perp\!\!\!\perp C | B$ and $A \perp\!\!\!\perp C$

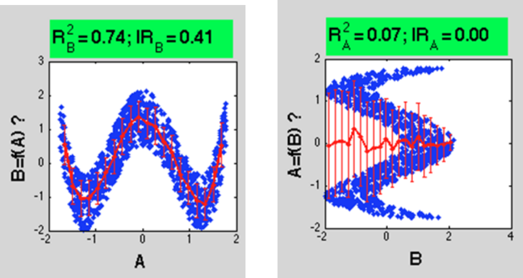
$$\begin{tikzcd} A \arrow[dr] & & C \arrow[dl] \\ & B & \end{tikzcd}$$

Spirtes et al. 00, 16

Key concepts: 3. Causality with distributional asymmetry

Leveraging Occam's razor principle; Janzig 19

$\rightarrow$ the causal model as the one being the simplest model that fits the data.

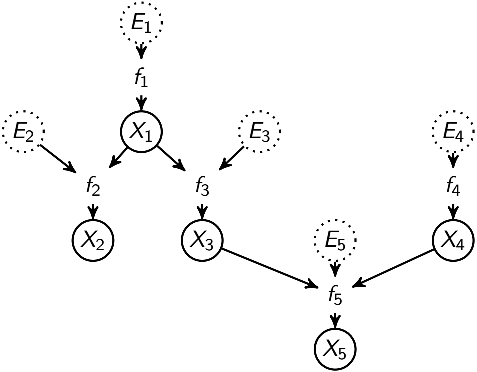


Framework: Functional Causal Models (FCMs)

Given $X_1, ..X_d$,

$$X_i = f_i(X_{\text{Pa}(i;\mathcal{G})}, E_i), \forall i \in [1, d]$$

with $X_{\text{Pa}(i;\mathcal{G})}$ the set of parents of X_i in $\mathcal{G}$ (= causes of X_i),
E_i a random independent noise variable modeling the unobserved other causes,
f_i a deterministic function: the causal mechanism



$$\begin{cases} X_1 = f_1(E_1) \\ X_2 = f_2(X_1, E_2) \\ X_3 = f_3(X_1, E_3) \\ X_4 = f_4(E_4) \\ X_5 = f_5(X_3, X_4, E_5) \end{cases}$$

Functional Causal Models, 2

Markov decomposition

$$P(X_1, \ldots, X_d) = \Pi P(X_i | X_{\mathrm{Pa}(i;\mathcal{G})})$$

Usual Assumptions

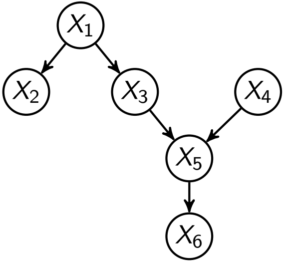
Causal Sufficiency: no unobserved confounders

Causal Markov: all d-separations in the causal graph $\mathcal{G}$ imply conditional independences in the observational distribution P

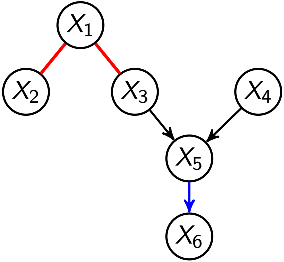
Causal Faithfulness: all conditional independences in P imply d-separations in $\mathcal{G}$.

Key approach 1: Constraint-based methods

Constraint-based methods, through V-Structures and constraint propagation, output a **CPDAG** (Completed Partially Directed Acyclic Graph).



(a) The exact DAG of $\mathcal{G}$.



(b) The CPDAG of $\mathcal{G}$.

Ex: Peter-Clark Algorithm (PC) Spirtes et al. 00

Non-linear extensions (CI tests): PC-HSIC (KCI-test), PC-RCIT

Zhang 12, Strobl 17

Key approach 2: Score-based methods

Objective function to optimize such as the Bayesian Information Criterion (BIC):

$$BIC(\mathcal{G}) = -2 \ln L + k * \ln n$$

with L: Likelihood of the model, k: number of parameters, n: Number of samples

The graph is optimized with the operators:

- add edge
- remove edge
- revert edge

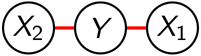
Ex: Greedy Equivalence Search (GES) Chickering 02

Limitations

- Computational cost dependent on the type of test/scoring method used
- Data hungry
- Identifiability issues

Example

$$X_1, E_{X_1}, E_{X_2} \sim \text{Uniform}(0,1), X_1 \perp\!\!\!\perp E_{X_1},\ \ Y \perp\!\!\!\perp E_{X_2}$$
$$Y \leftarrow 0.5X_1 + E_{X_1},$$
$$X_2 \leftarrow Y + E_{X_2},$$



Here $X_1 \perp\!\!\!\perp X_2 | Y$. No V-structure

Key approach 3: Global optimization

Assuming linear causal mechanisms, the causal mechanisms can be formulated in terms of linear algebra.

$$\mathbf{X} = B^T \mathbf{X} + E$$

And estimate the B matrix, through ICA for LiNGAM

Shimizu 06, Hyvarinen 99

$\rightarrow$ Graphical models — Pearl 09, Friedman 08

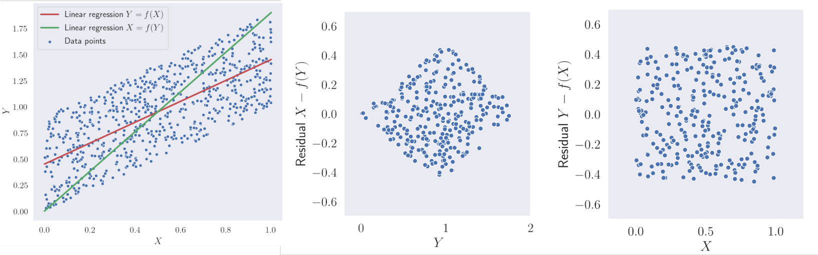
Ex: Max-Min Hill-Climbing (MMHC) — Tsamardinos 06

Concave penalized Coordinate Descent (CCDr) — Aragam 15

Key approach 4: Exploiting asymmetries in the distribution

→ If no v-structure available or causal discovery with 2 variables: leverage assymetries in the distributions.
Additive noise model (ANM): Hoyer 09

$$Y = f(X) + E$$



Original data **Residuals of X=g(Y)** **Residuals of Y=f(X)**

Ex: Post Non-Linear model (PNL), GPI

Zhang 10, Stegle 10

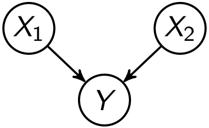
Limitations of asymmetry-based approaches

- Restrictive assumptions on the type of causal mechanisms
- Does not take into account conditional independence relations.

Zhang 09

Example

$$X_1, X_2, E_{X_1} \sim \text{Gaussian}(0,1), X_1 \perp\!\!\!\perp E_{X_1},\; X_2 \perp\!\!\!\perp E_{X_1}$$
$$Y \leftarrow 0.5X_1 + X_2 + E_{X_1}$$



(X_1, Y) and (X_2, Y) are perfect symmetric pairwise distribution (after rescaling)
However $X_1 \not\perp\!\!\!\perp X_2 | Y$: A V-structure may be identified

ey approach 5: A machine learning-based approach

Guyon et al, 2014-2015

Pair Cause-Effect Challenges

- Gather data: a sample is a pair of variables (A_i, B_i)
- Its label ℓ_i is the "true" causal relation (e.g., age "causes" salary)

Input

$$\mathcal{E} = \{(A_i, B_i, \ell_i), \ell_i \textit{ in } \{\rightarrow, \leftarrow, \perp\!\!\!\perp\}\}$$

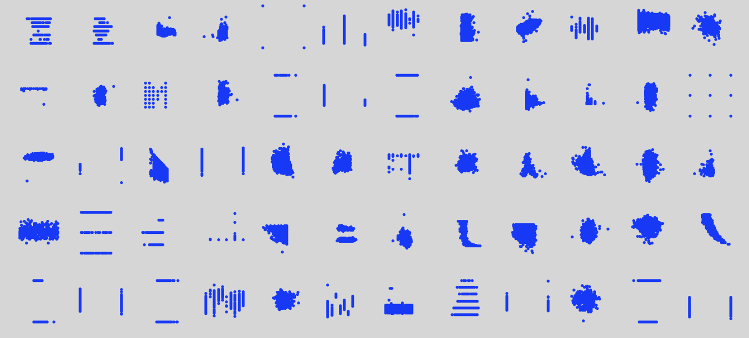
Example A_i, B_i	Label ℓ_i
A_i causes B_i	$\rightarrow$
B_i causes A_i	$\leftarrow$
A_i and B_i are independent	$\perp\!\!\!\perp$

Output

using supervised Machine Learning

$$\text{Hypothesis} : (A, B) \mapsto \text{Label}$$

Key approach 5: A machine learning-based approach, 2



The Cause-Effect Pair Challenge

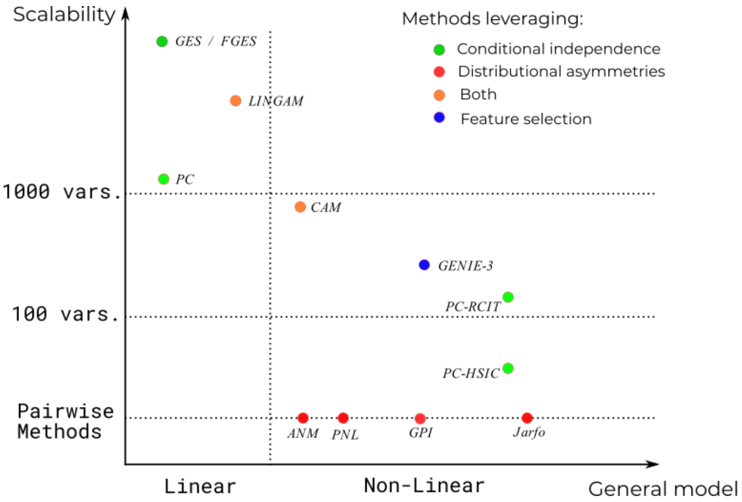
Learn a causality classifier (causation estimation)

- Like for any supervised ML problem from images ImageNet 2012

More

- Guyon et al., eds, *Cause Effect Pairs in Machine Learning*, 2019.

State of the art: summary



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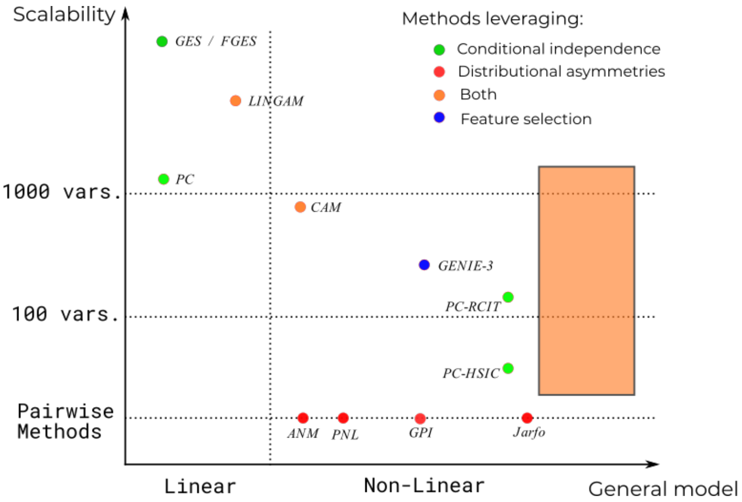


Causal Generative Neural Networks

Causal Generative Neural Networks (CGNN): Overview

Assumptions:

- Input: Graph skeleton with L edges
- Continuous data: $X_1 \ldots, X_d$ real valued

Problem posed:

- Combinatorial optimization problem of dimension L
- For each candidate in $\{-1, 1\}^L$, find each causal mechanism

Approach:

- Causal mechanisms f_i approximated as a neural net.
- Loss function: Maximum Mean Discrepancy (MMD) (distance original vs generated data);
- Hyperparameter: number n_h of neurons in f_i

Modeling FCMs with generative neural networks

- Idea: approximate the continuous mechanisms $f_1, \ldots, f_d$ with a set of one hidden layer neural networks $\hat{f} = (\hat{f}_1, \ldots, \hat{f}_d)$

Modeling FCMs with generative neural networks

- Idea: approximate the continuous mechanisms $f_1, \dots, f_d$ with a set of one hidden layer neural networks $\hat{f} = (\hat{f}_1, \dots, \hat{f}_d)$
- Estimate FCMs C as $\hat{C} = (\hat{\mathcal{G}}, \hat{f})$:

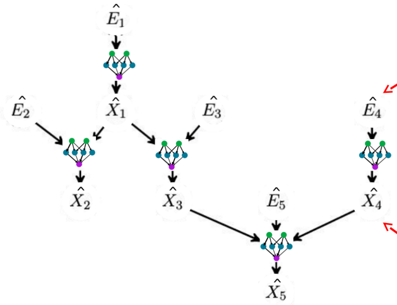
$$\hat{X}_i \leftarrow \hat{f}_i(\hat{X}_{\mathrm{Pa}(i;\widehat{\mathcal{G}})}, E_i), E_i \sim \mathcal{N}(0,1) \tag{1}$$

Generative neural networks as a FCM

Each function in a NN

$$\hat{X}_i \leftarrow \hat{f}_i(\hat{X}_{\mathrm{Pa}(i;\hat{\mathcal{G}})}, \hat{E}_i)$$

"Inputs" are noise var.



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Generated variables

For each candidate $(\hat{G}, \hat{f})$, generate samples $\hat{\mathbf{X}}$;
Loss = difference between original distribution, generated distribution

Learning Metric: Maximum Mean Discrepancy (MMD)

Kernel-based loss evaluating a "distance" between empirical distributions:

Gretton 05

- Generated data $\hat{\mathbf{X}} = \hat{\mathbf{x}}_i, i = 1 \dots n'$
- True data $\mathbf{X} = \mathbf{x}_i, i = 1 \dots n$

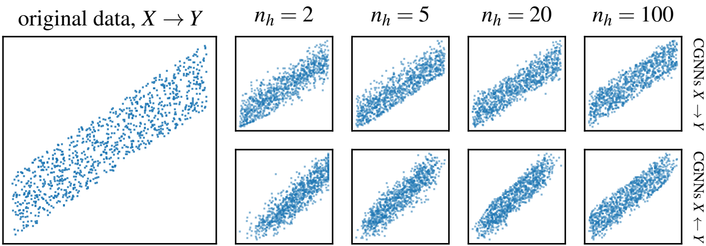
$$MMD(\hat{\mathbf{X}}, \mathbf{X}) = \frac{1}{n^2} \sum_{i,j} k(\mathbf{x}_i, \mathbf{x}_j) + \frac{1}{n'^2} \sum_{i,j} k(\hat{\mathbf{x}}_i, \hat{\mathbf{x}}_j) - \frac{2}{nn'} \sum_{i,j} k(\hat{\mathbf{x}}_i, \mathbf{x}_j)$$

with $k(\mathbf{u}, \mathbf{v}) = \sum_\ell exp^{-\frac{\gamma_\ell}{d}||\mathbf{u}-\mathbf{v}||^2}, \ \gamma_\ell \in \{10^{-2}, \dots, 10^2\}$

A linear approximation $\widehat{MMD}$ leveraging random projections has been proposed

Lopez-Paz et al. 16

Adjusting number of hidden units n_h



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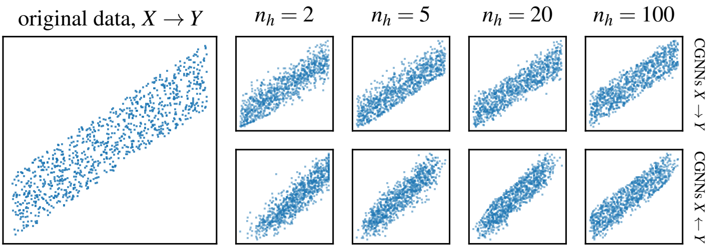

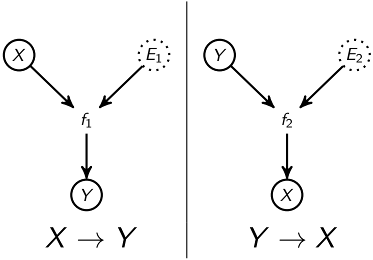

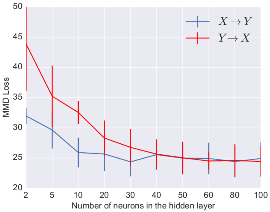

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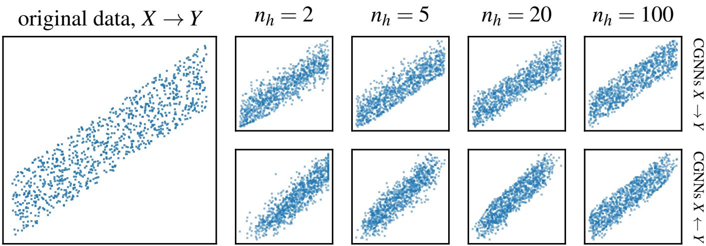

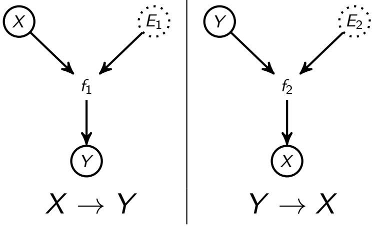

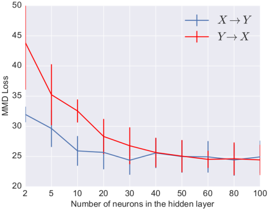

$\Rightarrow$ Causal direction not identifiable if n_h too high

General algorithm

Input = Continuous Data + Graph skeleton

1. Init: Pairwise orientation + DAG recovery (remove cycles heuristic)
2. Iteratively until the stopping criterion is met:
   - Reverse an edge at random that does not create a cycle
   - Retrain CGNN using backpropagation
   - If the resulting MMD loss is better, replace the current best solution

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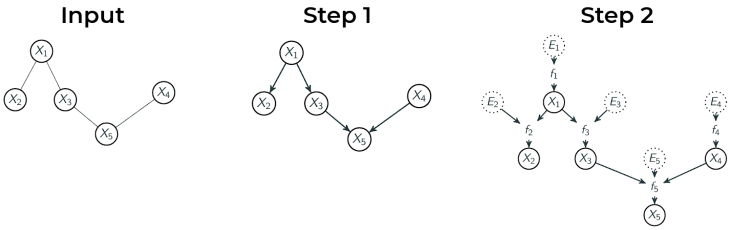

Experimental setting

- Benchmarks:
  - Simulated data: $X_i = f_i(X_{\mathrm{Pa}(i;\mathcal{G})}, E_i), \forall i \in [1, d]$, with f_i: Polynomials, Gaussian processes with additive and multiplicative noise
  - Biological data : SynTReN Gene expression, Real protein network

Sachs 05

- All methods are given the true skeleton
- Performance indicator: Area under the Precision Recall Curve (number of identified edges)

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  Sachs 05
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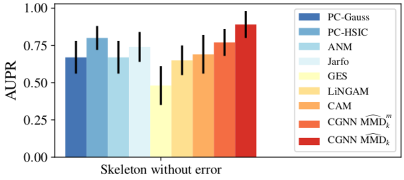
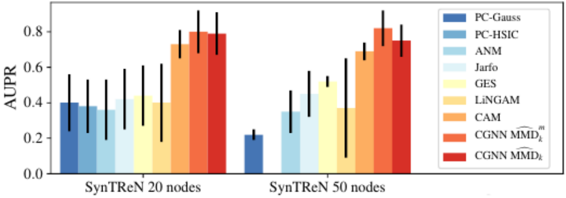
- Performance indicator: Area under the Precision Recall Curve (number of identified edges)
- Baselines:
  - PC, PC-HSIC (KCI-test) — Spirtes 00, Zhang 11
  - ANM — Hoyer 09
  - Jarfo — Fonollosa 16
  - GES — Chickering 02
  - LiNGAM — Shimizu 06
  - CAM — Buhlman 14
- CGNN: $n_h \in [5, 20], epochs = 2000, \ell_r = 0.01$
- $\widehat{MMD}_k^m$, $m = 300$ (Linear approx of MMD)

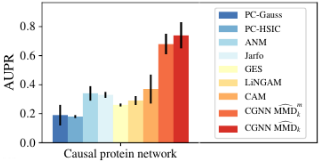
Experimental validation: Generated datasets



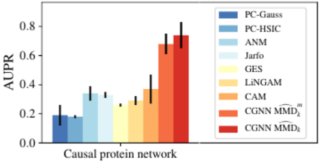


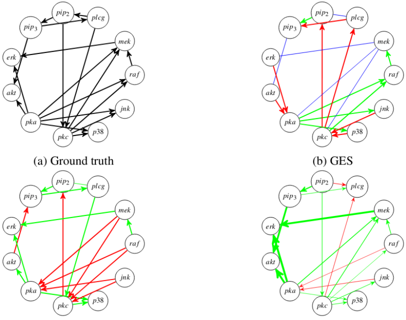
All methods are given the true skeleton.

Experimental validation: Real data



Experimental validation: Real data





(a) Ground truth (b) GES

(c) CAM (d) CGNN

Color: green: ok ; red: wrong; blue: unknown, Edge width: confidence

CGNN

PROS:

- **[UNIVERSALITY]** power of NN (universal approximators)
- **[UNIFICATION]** unification of causal discovery principles (CI and DA)

CONS:

- **[SKELETON KNOWLEDGE NEEDED]** the method requires the initial knowledge of the graph skeleton (though edge orientation is robust against skeleton mistakes)
- **[COMPUTATIONAL COST]** the method is computationally costly (30h for 50 variables) which in practice required us to perform sub-optimal greedy optimizations
- **[SENSITIVITY]** the method is sensitive to hyper-parameter selection (including number of neurons)

Structural Agnostic Modeling

Structural Agnostic Model (SAM): Overview

Assumptions:

- Continuous data
- Causal sufficiency (no hidden confounder)

Goal:

- Learn end-to-end the graph structure and the causal mechanisms

Approach:

- A global loss
- accounting for structural and functional complexity
- accounting for model fitness through an adversarial mechanism

Finding the causes for each variable

$$X_j = f_j(X_{-j}, E_j), \tag{2}$$

Finding the causes for each variable

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Goal: Find the causes = a sparse network it generates

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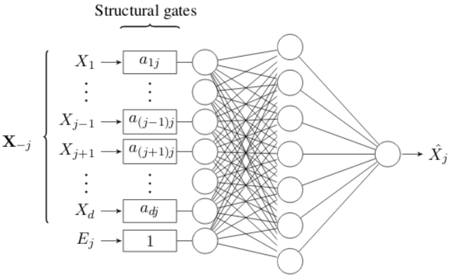
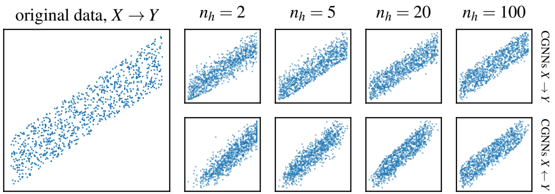


→ Enforcing sparsity through L_0 penalization

Leray 99, Maddison 16, Jang 16

Regularization of the complexity of the mechanisms



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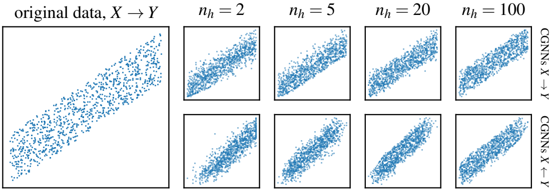
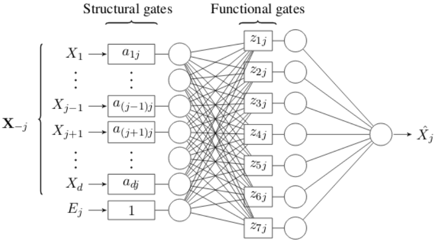




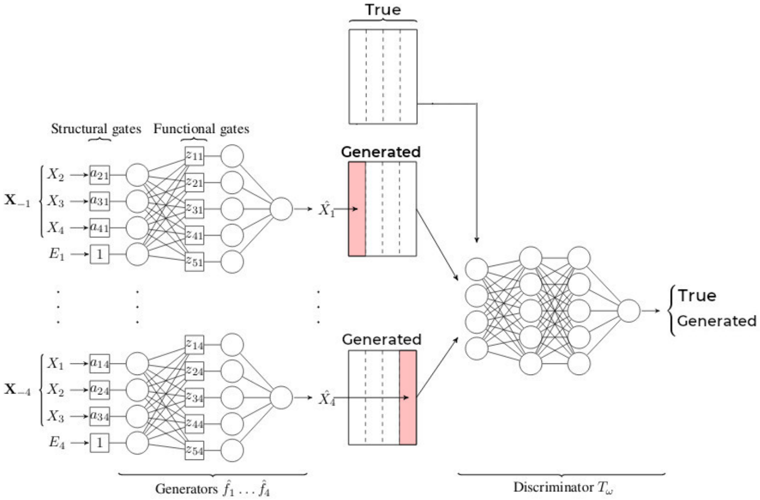
$\rightarrow$ Enforcing the sparsity of the mechanisms through L_0 penalization

General architecture and loss of SAM

$\rightarrow$ Adversarial loss goodfellow2014generative

General architecture and loss of SAM

→ Adversarial loss goodfellow2014generative



Loss of SAM

Learning criterion to minimize:

$$S(\widehat{\mathcal{G}}, \hat{f}, D) = \underbrace{-\mathbb{E}_{x \sim p(x)} \left[\log\, q(x, \theta, \widehat{\mathcal{G}}) \right]}_{\substack{\text{Log likelihood} \\ \text{estimated by the discriminator}}} + \underbrace{\lambda_A \|A\|_1 + \lambda_Z \|Z\|_1}_{\text{Regularization}}, \quad (3)$$

where

- $\|A\|_1 = \sum_{i,j=1..d} a_{i,j}$: total number of **edges in** $\widehat{G}$
  $\rightarrow$ Structural complexity.
- $\|Z\|_1 = \sum_{j=1,\ldots,d} \sum_{h=1,\ldots,n_h} z_{j,h}$: total number of **active units in** $\widehat{f}$
  $\rightarrow$ Functional complexity.

Final learning objective

$$
\begin{aligned}
S(\widehat{\mathcal{G}}, \hat{f}, D) = & \underbrace{\sum_{j=1}^{d} I(X_j, X_{\overline{\mathrm{Pa}(j;\widehat{\mathcal{G}})}} | X_{\mathrm{Pa}(j;\widehat{\mathcal{G}})}) + \lambda_A \|A\|_1}_{\text{Structural score}} \\
& + \underbrace{\sum_{j=1}^{d} D_{KL}[p(x_j | x_{\mathrm{Pa}(j;\widehat{\mathcal{G}})}) \parallel q(x_j | x_{\mathrm{Pa}(j;\widehat{\mathcal{G}})}, \theta_j)] + \lambda_Z \|Z\|_1}_{\text{Functional score}} \\
& + \underbrace{\lambda_D \sum_{k=1}^{d} \frac{\mathrm{tr}\ A^k}{k!}}_{\text{Acyclicity constraint}}
\end{aligned}
$$

Zheng 18

with I the mutual information and D_{KL} the Kullback-Leibler divergence

Properties of the score

Theorem 1: Identification to the Markov Equivalence Class

Under Causal Markov and faithfulness assumptions, the DAG $\widehat{G}$ minimizing the structural score belongs to the Markov equivalence class of the true graph G (CPDAG of G)

Properties of the score

Theorem 1: Identification to the Markov Equivalence Class

Under Causal Markov and faithfulness assumptions, the DAG $\widehat{G}$ minimizing the structural score belongs to the Markov equivalence class of the true graph G (CPDAG of G)

Theorem 2: Identification of the DAG

Under additional assumptions, the DAG $\widehat{G}$ minimizing **also** the functional score is exactly the DAG G

Experimental setting

- Benchmarks:
  - Simulated data (20 and 100 Variables):
    $X_i = f_i(X_{\mathrm{Pa}(i;\mathcal{G})}, E_i), \forall i \in [1, d]$,
    f_i: Linear, Gaussian processes with additive (GP AM) and multiplicative noise (GP Mix), Sigmoid functions (Sigmoid AM/Sigmoid Mix), Neural networks with randomized weights (NN).
  - Biological data : SynTReN Gene expression , Real protein network Sachs 05
- Performance indicator: Area under the Precision Recall Curve

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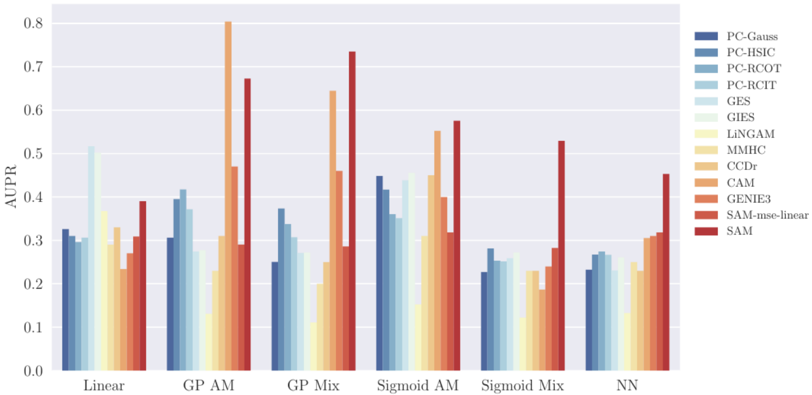
  - Biological data : SynTReN Gene expression , Real protein network — Sachs 05
- Performance indicator: Area under the Precision Recall Curve
- Baselines:
  - PC, PC-HSIC (KCI-test) — Spirtes 00, Zhang 11
  - PC-RCIT/RCOT — Strobl 17
  - ANM — Hoyer 09
  - Jarfo — Fonollosa 16
  - GES — Chickering 02
  - LiNGAM — Shimizu 06
  - CAM — Buhlman 14
  - MMHC — Tsamardinos 06
  - CCDr — Aragam 17
  - GENIE3 — Irrthum 10

Experimental setting (2)

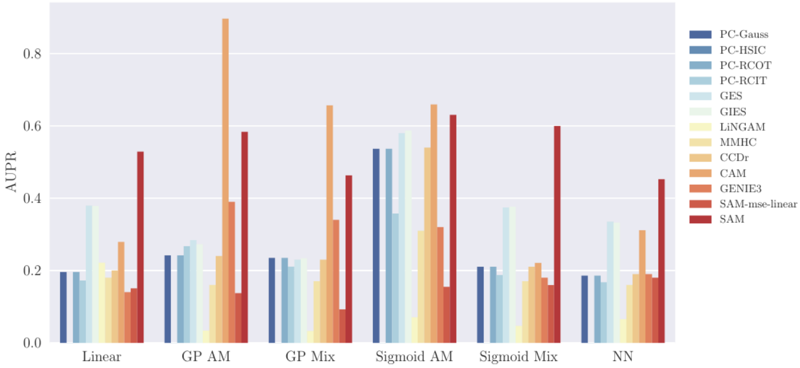
- Hyperparameters of SAM:
  - $\ell_r = 0.01$
  - $\lambda_A = 0.01$
  - $\lambda_z = 10^{-5}$
- Lesion study (impact of neural vs linear mechanims and mean square error vs adversarial loss):
  - SAM-mse-linear: Linear mechanisms and a MSE loss
  - SAM-linear: Linear mechanisms and a GAN Setting
  - SAM-mse: Non-linear mechanisms and a MSE Loss

Experimental results: Generated datasets (20 variables)

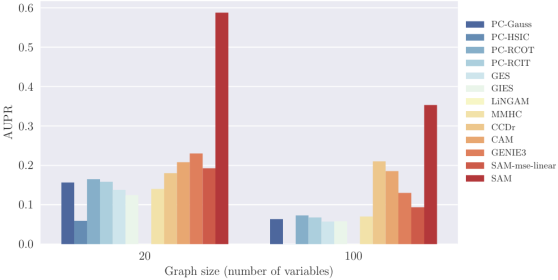


CAM is especially tailored for Gaussian processes with additive noise; and GES for linear mechanisms

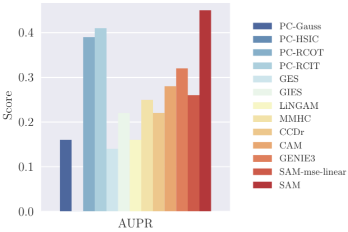
Experimental results: Generated datasets (100 variables)



Results on biological data

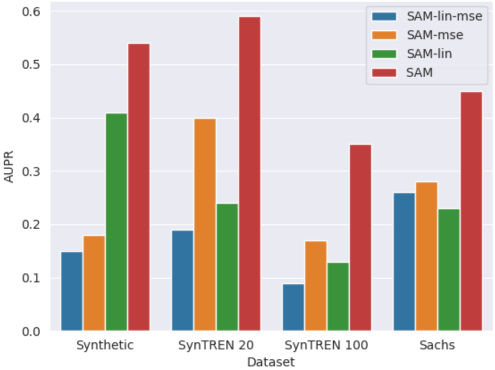


Syntren Dataset



Sachs dataset

Ablation studies



Both the non-linear mechanisms and the adversarial network are required to attain maximum performance

Computational time (graph of 100 variables)

AP	Time in s. (CPU)	Time in s. (GPU)
PC-Gauss	13	
PC-HSIC	-	
PC-RCOT	31 320	
PC-RCIT	46 440	
GES	1	
GIES	5	
MMHC	5	
LiNGAM	5	
CAM	45 899	
CCDr	3	
GENIE3	511	
SAM-lin-mse	3 076	74
SAM-mse	18 180	118
SAM-lin	24 844	1 980
SAM	24 844	2 041

Applications

Applications: 1. Human Resources

Causal Modeling and Human Resources

Known:

- A Quality of life at work — employee's perspective
- B Economic performance — firm's perspective
- ... are correlated

Question: Are there causal relationships ?

$A \rightarrow B$; or $B \rightarrow A$; or $\exists C \ / \ C \rightarrow A$ and $C \rightarrow B$

Data

- Polls from Ministry of Labor
- Gathered by Group Alpha Secafi (trade union advisor)
- Tax files + social audits for 408 firms

Economic sectors: low tech, medium-low, medium-high and

Variables

Economic indicators

- Total number of employees
- Capitalistic intensity, Total payroll, Gini index
- Average salary (of workers, technicians, managers)
- Productivity, Operating profits, Investment rate

People

- Average age, Average seniority, Physical effort,
- Permanent contract rate, Manager rate, Fixed-term contract rate, Temporary job rate, Shift and night work, Turn-over
- Vocational education effort, duration of stints, Average stint rate (for workers, technicians, managers);

Variables, cont'd

Quality of life at work

- Frequency & Gravity of work injuries, Safety expenses, Safety training expenses
- Absenteism (diseases), Occupational-related diseases
- Resignation rate, Termination rate, Participation rate
- Subsidy to the works council

Men/Women

- Percentage of women (employees, managers)
- Wage gap between women and men (average, for workers, technicians, managers)

General Causal Relations

Access to training $\nearrow$

- $\searrow$ Gravity of work injuries
- $\searrow$ Occupational-related diseases

Termination rate $\nearrow$

- $\nearrow$ Absenteism (diseases)

Percentage of managers $\nearrow$

- $\nearrow$ Access to training
- $\searrow$ Shift or night working hours

Age $\nearrow$

Global relations between QLW and performance ?

Failure

- Nothing conclusive

Interpretation

- Exist confounders (controlling QLW and performance) $C \to A$ and $C \to B$
- One such confounder is the activity sector
- In different activity sectors, causal relations are different (hampering their identification)
- $\Rightarrow$ Condition on confounders

Low-tech sector

- Resignation rate $\nearrow$, Productivity $\searrow$
- Average salary $\nearrow$, Productivity $\nearrow$ very significant
- Occupational-related diseases $\nearrow$, Productivity $\searrow$
- Temporary job rate $\nearrow$, Gravity of work injuries $\nearrow$
- Permanent contract rate $\nearrow$, Safety training $\searrow$
- Duration training stints $\nearrow$, Termination rate $\searrow$

Outcomes & Limitations

Causal modeling and exploratory analysis

- Efficient filtering of plausible relations (several orders of magnitude);
- Complementary w.r.t. visual inspection (experts can be fooled and make sense of correlations & hazards);
- Multi-factorial relations ? yes; but even harder to interpret.

Not a ready-made analysis

- Causal relations must be
  - interpreted
  - confirmed by field experiments; polls; interviews.

Applications: 2. Food and Health

A data-driven approach to individual dietary recommendations

Context

- Long-term goal: Personalized dietary recommendations
- Requirement: identify risk index associated to food products
- At a coarse-grained level (lipid, protein, glucid), nothing to see
- At a fine-grained level: 300+ types of pizzas, ranging from ok to very bad.

The wealth of Kantar data

- $\sim$22,000 households $\times$ 10 years (this study: 2014)
- 19M total purchases/year (180,000 products)
- Socio-demographic attributes, varying size

Beware: data rarely collected as should be...

Raw description can hardly be used for meaningful analysis

- 170,000 products for 22,000 households
- Data gathered with (among others) marketing goals
  where bought, which conditioning
- Most products are sold by 1 vendor
- Most families are going to one vendor

Manual pre-processing

- Consider 10 categories of interest, e.g. bio/non-bio; alcohol yes/no; fresh/frozen
- Merge products with same categories
- 170,000 $\rightarrow \approx$ 4,000 products

Example: for beer, we only selected as features of interest: colour (blonde, black, etc.); has-alcohol (yes, no); organic (yes, no)

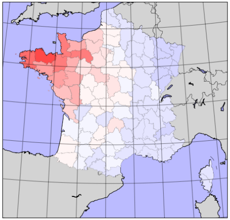
Methodology

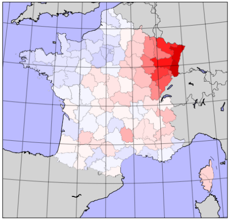
Dimensionality reduction

1. Borrowing Natural Language Processing tools, with
   vector of purchase ≈ document
   food product ≈ word
2. Using Latent Dirichlet Association to extract "dietary topics"

Blei et al. 03

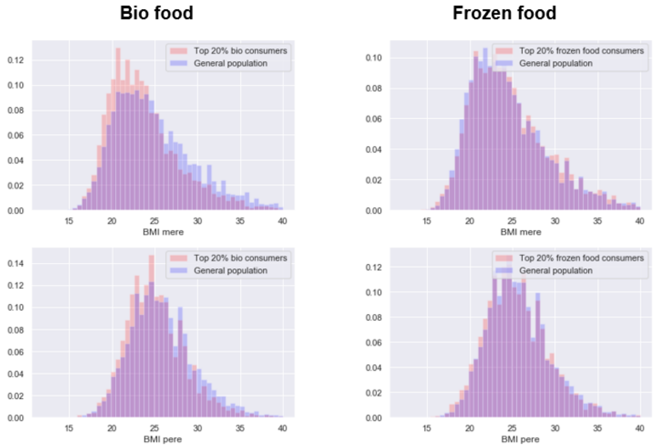
Some topics can be directly interpreted The darker the region, the more present the topic (NB: regions are not used to build topics)





Focus: impact of topics on BMI

Left: Bio/organic topic
Right: Frozen food topic
Top row: Women
Bottom row: Men



High weight of Bio topic is correlated with lower BMI ($p < 5\%$)
(particularly so for women)

Does A (eat bio) cause B (better BMI) ?

Three cases

- A does cause B (bio food is better)
- Confounder: exists C that causes A and B (rich/young/educated people tend to consume bio products and have lower BMI);
- Backdoor effects: exists C correlated with A which causes B (people eating bio also tend to eat more greens, which causes lower BMI);

Goal: Find out which case holds

Causal models

- Ideally based on randomized controlled trials

Imbens Rubins 15

Proposed Methodology

Taking inspiration from Abadie Imbens 06

Target population: “Bio” people $=$ top quantile coordinate on bio topic.

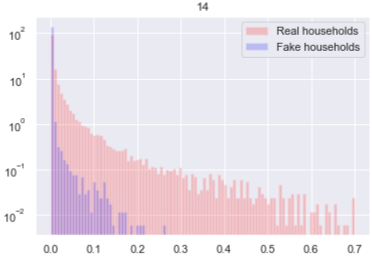
RCT would require a control population

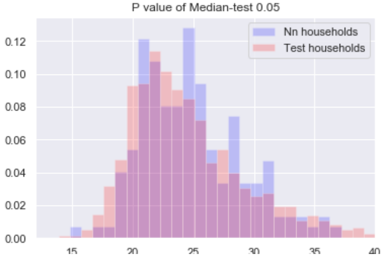
Building a control population finding matches

- For each bio person, take her consumption z (basket of products)
- Create a falsified consumption z' (replacing each bio product with same, but non-bio, product)
- Find true consumption z“ nearest to z' (in LDA space)
- Let the true person with consumption z“ be called ”falsified bio“

Compare bio and ”falsified bio“ populations wrt BMI

Bio vs Falsified Bio populations





Left

- Projection on the Bio topic (in log scale)
- (Falsified bio population not 0: the bio topic contains e.g. sheep yogurt).

Right

- BMI Histograms of both bio and falsified bio populations
- Statistically significant difference

Next

Chasing confounders

- Discriminating bio from "falsified bio" populations w.r.t. socio-professional features: accuracy $\approx$ 60%
- Candidate confounder: mother education level (on-going study)

Next steps

- Confirm conjectures using longitudinal data (2015-2016)
- Interact with nutritionists / sociologists
- Extend the study to consider the impact of, e.g.
  - Price of the food
  - Amount of trans fats
  - Amount of added sugar

Discussion

Perspectives: Causality analysis and Big Data

Finding the needle in the haystack

- Redundant variables (e.g. in economics) $\rightarrow$ un-interesting relations
- Variable selection
- Feature construction dimensionality reduction

Beyond causal sufficiency

- Confounders are all over the place (and many are plausible, e.g. age and size of firm; company ownership and shareholdings)
- When prior knowledge available, condition on counfounders
- Use causal relationships on latent variables Wang and Blei, 19
  to filter causal relationships on initial variables

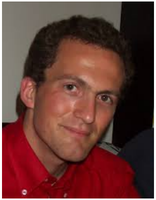
A python package for observational causal discovery

All the presented framework is available on GitHub at :
https://github.com/Diviyan-Kalainathan/CausalDiscoveryToolbox
It includes multiple algorithms as well as tools for graph structure.
Accepted at JMLR - Open Source Software

Kalainathan Goudet 19







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