# Causal EM for counterfactual inference, with an application to palliative care

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IDSIA is a research institute on Artificial Intelligence founded in 1988 in Lugano, Switzerland



121 people = 16 Professors, 51 Researchers, 45 PhD students, 9 Research assistants

# Istituto Dalle Molle di studi sull'intelligenza artificiale



Thanks to Italian philantropist Angelo Dalle Molle (1908-2002)



«The progress of science in general, and that of the emerging computer science in particular, should not subjugate people, but rather benefit them»



## Lugano





# Headache

 $X \longrightarrow Y$ 

• You take an aspirin (X=1) and your headache vanishes (Y=1)

- What is the probability that this has been due to the aspirin?
  - Data says: P(Y=0|X=0)=0.5 and P(Y=0|X=1)=0.1
- What if I had **not** taken the aspirin, would have headache stayed?
- Probability of necessity (PN):  $P(Y_{X=0} = 0 | Y = 1, X = 1)$
- We need a fully specified structural causal model (SCM) to compute it - What if not? - Yet P(X,Y) is available  $f_X(U) =: X \xrightarrow{\downarrow} f_Y$   $Y := f_Y(X,V)$   $f_X(U) =: X \xrightarrow{\downarrow} f_Y$   $Y := f_Y(X,V)$   $f_X(U) =: X \xrightarrow{\downarrow} f_Y$   $Y := f_Y(X,V)$   $f_X(U) =: X \xrightarrow{\downarrow} f_Y$   $Y := f_Y(X,V)$  $F_X(U) =: X \xrightarrow{\downarrow} f_Y$   $Y := f_Y(X,V)$



#### Causal inference (via *credal nets* = sets of Bayesian nets)

- Select number of states for U and V
- No knowledge:
  - Conservative specification (canonical partition)
    |U| = 2, |V| = 4



- Propagate P(X,Y) back to find out P(U) and P(V)
- We get P(U) = P(X) and P(V) = [t, 0.4 + t, 0.5 t, 0.1 t], t∈[0,0.1] (we call this a credal set = a set of distributions)
- Create its twin net where PN = P(Y' | X'=0,X=1,Y=1)
- Run an *exact* credal net algorithm to finally get 4/9 <= PN <= 5/9</li>
   One can *in principle* solve all counterfactuals with this methodology





# Problem

 The previous exact approach works well with Markovian structural causal models (SCMs)

- So-so 🙂 with quasi-Markovian ones
- And does not 😕 for non-quasi-Markovian SCMs



- Thm.: Causal inference (interventions) is NP-hard even in polytree-SCMs
  - Hardly surprising if you're in credal nets
- Let's approximate!
- Idea:

the exogenous variables are missing at random



## EM for Causal Computation (EMCC)

- Say we have a data set D of iid (x,y) instances
- Randomly initialise P(U) and P(V)
- Run the EM up to convergence



- Cor.: At convergence, P(U) and P(V) belong to their corresponding credal sets
  - EM samples the space of compatible fully specified SCMs!
  - On each of them, we can compute PN on its twin graph via Bayesian nets
- Repeat k times: random initialisation + EM up to convergence + BN algorithm
  - You get {P<sub>i</sub>(U),P<sub>i</sub>(V)} and PN<sub>i</sub>, for i=1, ..., k
  - A set of k points inside the interval [4/9,5/9]
- Take min and max and you get an **inner** approximation:  $[a,b] \subseteq [4/9,5/9]$ 
  - k=20 already gives a pretty good approximation

works for any semi-Markovian SCM with categorical endogenous variables



#### Why does it work?

- Thm.: The (log-)likelihood is unimodal
- Cor.: The global optimum points are IFF  $P(U_1)$ , ...,  $P(U_m)$  in their resp. credal sets



#### How well does it work?

(code available at github.com/idsia/credici)



- Remember  $[a,b] \subseteq [a^*,b^*]$
- k=20–30 runs already fairly good approximation
- Corollary 3 If a = b, i.e., all k runs in ρ are equal, then P(a\* = b\*|ρ) = 1 + 9/3<sup>k</sup> 8/2<sup>k</sup>
   9 equal runs => identifiable at 99% confidence

#### An application in palliative care



- Data from 116 patients about all the Boolean variables in the network =>  $X_1$ , ...,  $X_{12}$
- No latent confounders => structural causal model is Markovian => U<sub>i</sub> -> X<sub>i</sub> (i=1, ...,12)
  - Use the conservative specification
- Compute *PNS* := *P*(*Death*<sub>X=yes</sub> = home, *Death*<sub>X=no</sub> = hospital) w.r.t. the controllable X's
- EMCC: Triangolo 27–35%; family's awareness 4–11%; patient's awareness 3–11%
  - By the very Triangolo variable we can change the fate for ~30% of patients

#### EMCC extended to selection bias

• Consider (*X*,*Y*,*Z*) = (Treatment, Outcome, Gender) with SCM and data as shown



- Treated males (X=1,Z=1) and untreated females (X=0,Z=0) systematically not reported
  - Case of selection bias
  - Can we still say something about the overall population?

#### **EMCC** extended to selection bias

• Consider (X,Y,Z) = (Treatment, Outcome, Gender) with SCM and data as shown



• EMCC is applied as before with the only difference that the iteration becomes

$$P_{t+1}(U) \leftarrow \frac{d_0 P_t(U|S=0) + \sum_{\boldsymbol{x} \in \mathcal{D}_1} P_t(U|\boldsymbol{x})}{(d_0 + d_1)}$$

Main results hold as before: Experiments:
 Thm. 1: The (log-)likelihood is unimodal
 Cor. 1: The global optimum points are IFF P(U<sub>1</sub>), ..., P(U<sub>m</sub>) in their resp. credal sets

r = 10

r = 20

r = 30

#### Conclusions

- The EMCC is based on simple ideas and tools
  - It should not be too difficult to join it to other models
  - Or to extend it to the continuous case
  - It might lead to `simple', while general, ways to join causal inference with machine learning
- It delivers guaranteed inner approximations
  - Outer ones are safer but tend to be more difficult to yield without becoming loose
  - Yet EMCC is anytime and can easily be made parallel
  - And we can yield credible intervals to increase safety with some guarantee
- More work is certainly needed on all these fronts

For now, we have automated counterfactual computation also under selection bias