# Graphical Models for Causal Inference

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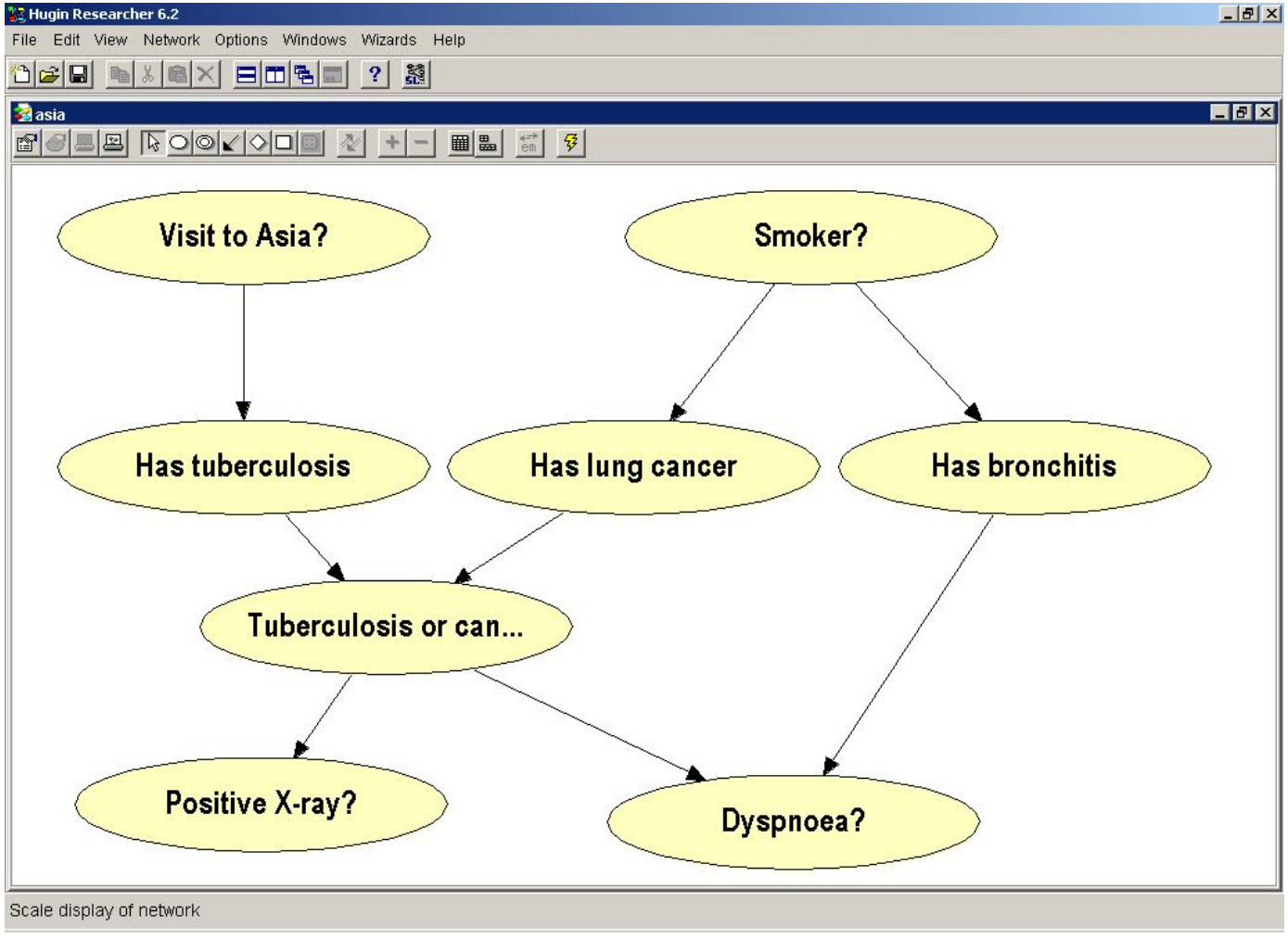
# Overview

- Causal interpretation of Bayesian networks
- Structural equation systems
- Assessment of treatment effects
- Intervention diagrams and LIMIDS
- Identifiability of causal effects
- Potential responses and mapping variables
- Discovery of (causal) structure

# Why are Bayesian networks sensible?




Causal interpretation!

# Intervention vs. observation

Causal interpretations are tied to the notion of *conditioning by intervention*

$$P(X = x \,|\, Y \leftarrow y) = p(x \,||\, y), \tag{1}$$

which in general is quite different from conventional conditioning or *conditioning by observation* which is

$$P(X = x \,|\, Y = y) = p(x \,|\, y) = p(x, y)/p(y).$$

A causal interpretation of a Bayesian network involves giving (1) a simple form.

# Causal Bayesian network

We say that a BN is *causal w.r.t. atomic interventions at* $B \subseteq V$ if it holds for any $A \subseteq B$ that

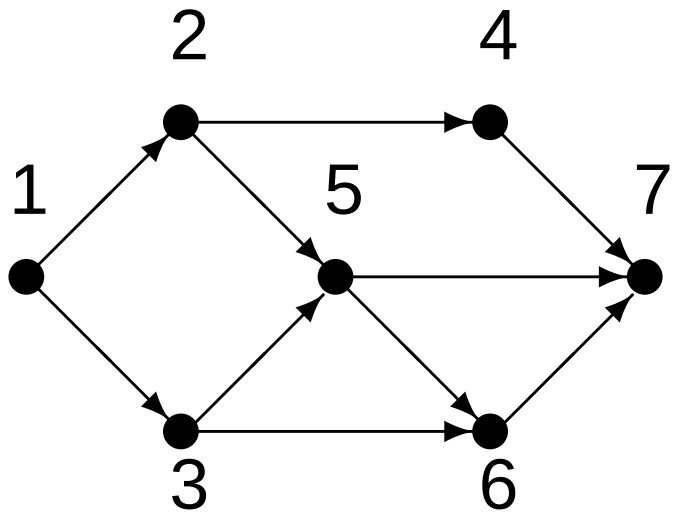
$$p(x \,||\, x^*_A) = \prod_{v \in V \setminus A} p(x_v \,|\, x_{\mathrm{pa}(v)}) \Big|_{x_A = x^*_A}$$

For $A = \emptyset$ we obtain standard factorisation.

Note that *conditional distributions* $p(x_v \,|\, x_{\mathrm{pa}(v)})$ are *stable under interventions* which do not involve $x_v$.

Such assumption must be justified in any given context.

# Intervention vs. observation in example




$$
\begin{aligned}
p(x \,||\, x_5^*) &= p(x_1)p(x_2 \,|\, x_1)p(x_3 \,|\, x_1)p(x_4 \,|\, x_2) \\
&\times \; p(x_6 \,|\, x_3, x_5^*)p(x_7 \,|\, x_4, x_5^*, x_6)
\end{aligned}
$$

whereas

$$
\begin{aligned}
p(x \,|\, x_5^*) &\propto p(x_1)p(x_2 \,|\, x_1)p(x_3 \,|\, x_1)p(x_4 \,|\, x_2) \\
&\times \; p(x_5^* \,|\, x_2, x_3)p(x_6 \,|\, x_3, x_5^*)p(x_7 \,|\, x_4, x_5^*, x_6)
\end{aligned}
$$

# Structural equation systems

DAG $\mathcal{D}$ can also represent structural equation system:

$$X_v \leftarrow g_v(x_{\mathrm{pa}(v)}, U_v), v \in V, \tag{2}$$

where $g_v$ are fixed functions and $U_v$ are independent random disturbances.

Intervention in structural equation system can be made by *replacement*, i.e. so that $X_v \leftarrow x_v^*$ is replacing the corresponding line in ‘program’ (2).

Corresponds to $g_v$ and $U_v$ being unaffected by the intervention.

# Justification by structural equations

*Intervention by replacement in structural equation system implies $\mathcal{D}$ causal for distribution of $X_v, v \in V$.*

Occasionally used for *justification* of CBN.

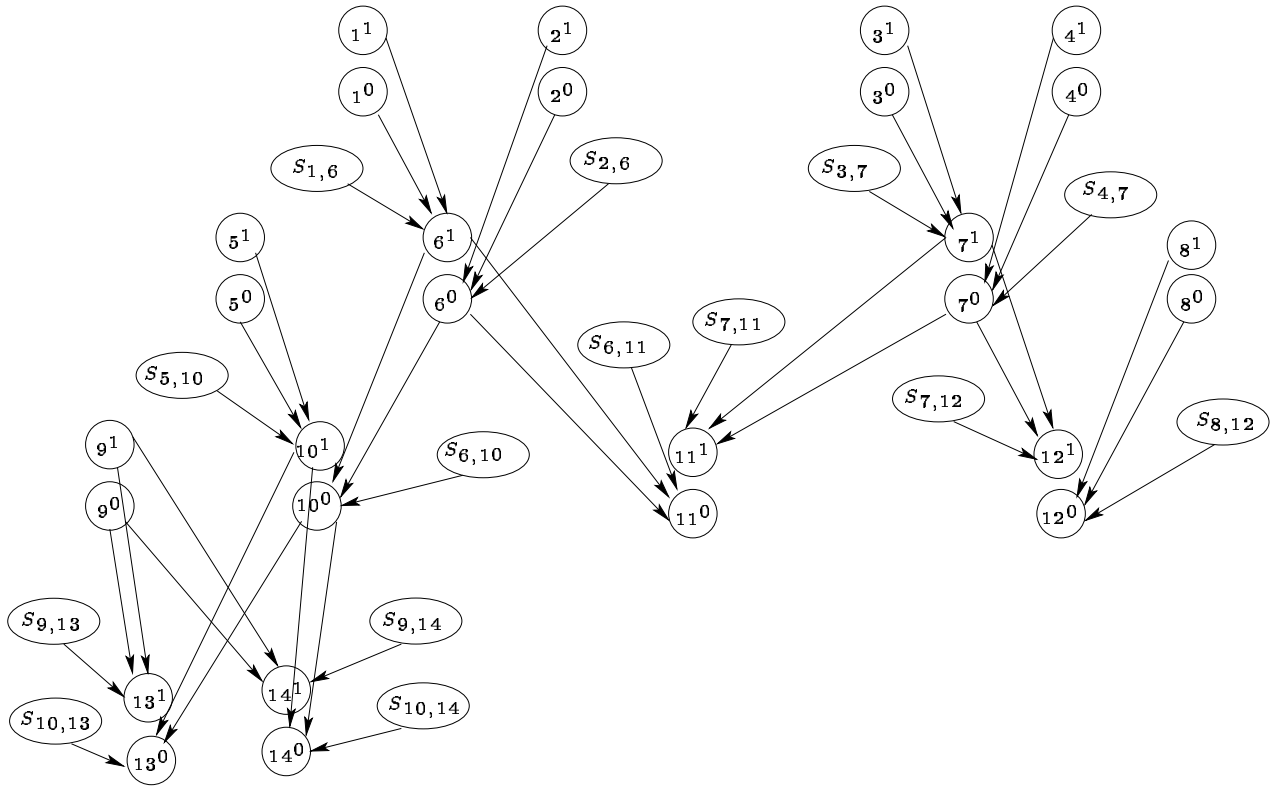
Ambiguity in choice of $g_v$ and $U_v$ makes this problematic.

May take *stability of conditional distributions as a primitive* rather than structural equations.

Structural equations more expressive when choice of $g_v$ and $U_v$ can be externally justified.

Nodes $U_v, v \in A$ can be adjoined to the network as additional parents of $X_v$.

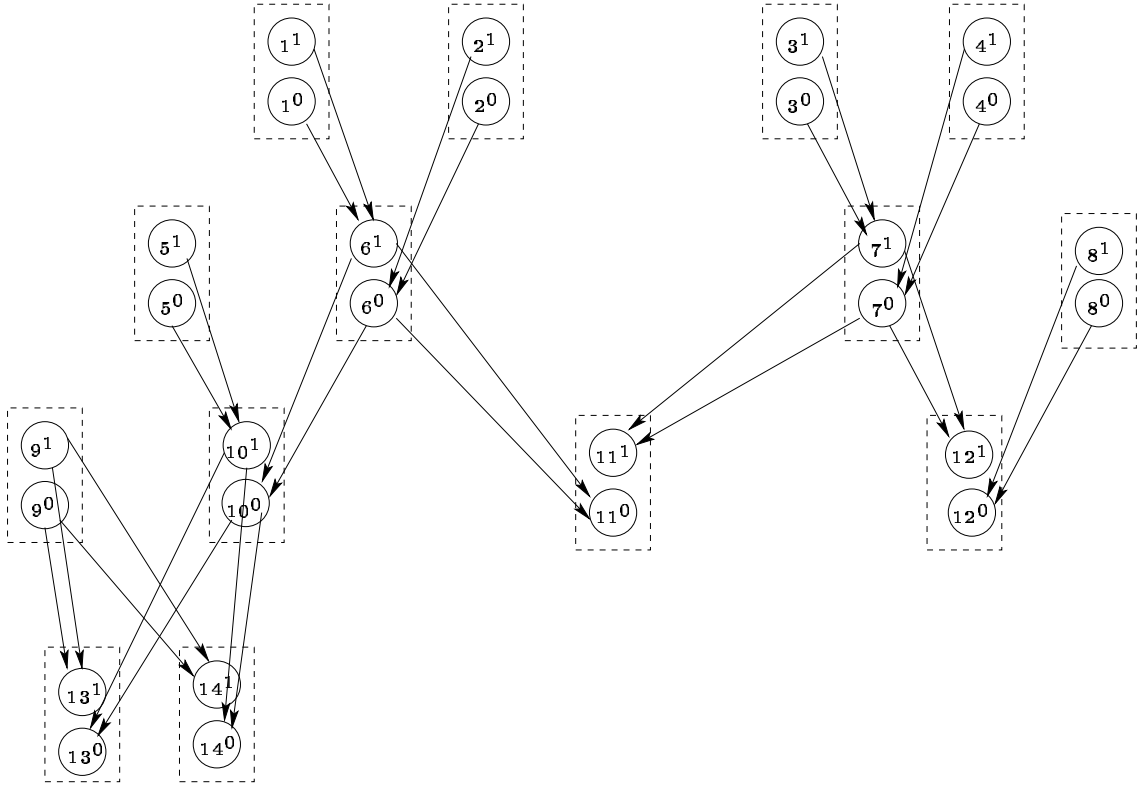
# Genetic segregation network




Circles represent *alleles*. Ovals represent *segregation indicators:* 1 for paternal transmission, 0 for maternal.

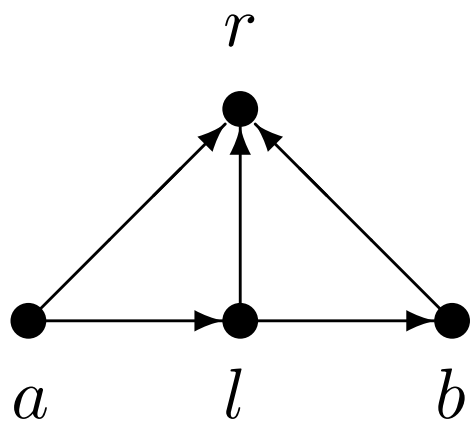
Relationships deterministic!

# Allele network




Causal Markov property follows from deterministic representation as segregation network, equivalent to structural equation model.

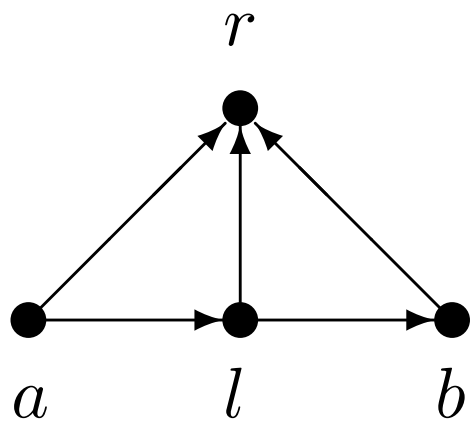
# Assessment of treatment effect




$a$ - treatment with AZT; $l$ - intermediate response (possible lung disease); $b$ - treatment with antibiotics; $r$ - survival after a fixed period.

Predict survival if $X_a \leftarrow 1$ and $X_b \leftarrow 1$, assuming stable conditional distributions.

# G-computation




$$
\begin{aligned}
p(1_r \,||\, 1_a, 1_b) &= \sum_{x_l} p(1_r, x_l \,||\, 1_a, 1_b) \\
&= \sum_{x_l} p(1_r \,|\, x_l, 1_a, 1_b) p(x_l \,|\, 1_a).
\end{aligned}
$$

# More complex interventions

Intervene with *strategy* $\sigma_A = \{\pi_v, v \in A\}$ for choosing the 'treatments' $x_v, v \in A$ depending on the outcome of other variables in $\mathrm{pa}^*(v)$.

Stability of conditional distributions gives

$$p(x \,||\, \sigma) = \prod_{v \in A} \pi_v(x_v \,|\, x_{\mathrm{pa}^*(v)}) \prod_{v \in V \setminus A} p(x_v \,|\, x_{\mathrm{pa}(v)}).$$

Typically, $\mathrm{pa}^*(v) \neq \mathrm{pa}(v)$. Graph $\mathcal{D}^* = (V, E^*)$ must be DAG for intervention to make sense.

Variables in $\mathrm{pa}^*(v)$ must be observed before intervention on $X_v$ is implemented.

# Limited Memory Influence Diagrams

A *Limited Memory Influence Diagram* (LIMID) is a BN of *chance nodes*, *decision nodes* and *utility nodes*.

- Chance nodes $\Gamma$ represented with circles
- Decision nodes $\Delta$ represented with squares.
- Utility nodes $\Upsilon$ represented with diamonds.
- Parents of decision nodes are observed before decision taken.

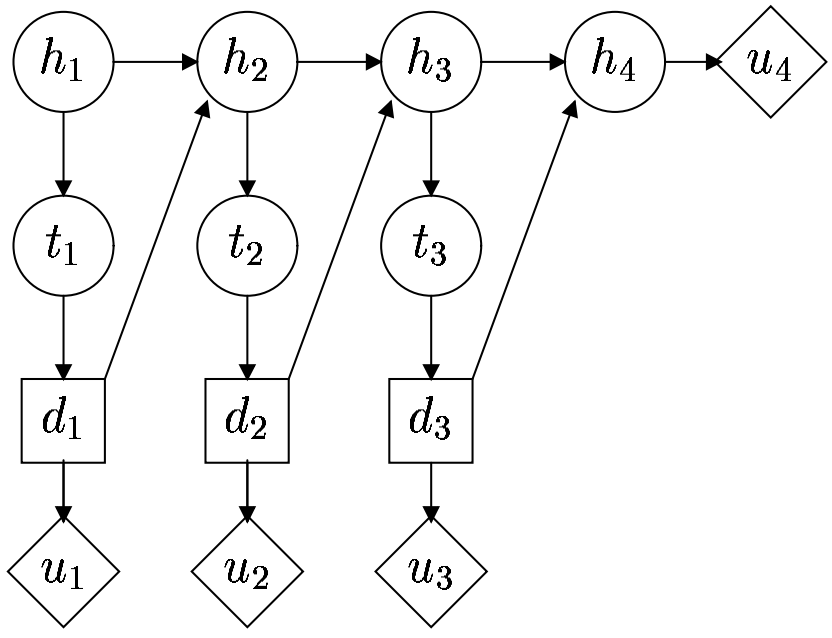
Relaxes traditional assumptions of influence diagrams, where decisions are taken in specified order and previous decisions and observations remembered.

# Limited Memory Influence Diagram

$h$ is health

$t$ is test

$d$ is treat or not




$t_1$ observed when $d_1$ is taken. Then $t_2$ is observed and $d_2$ is taken, etc.

# Intervention diagram

Augment each node $v \in A$ where intervention is contemplated with additional parent variable $F_v$.

$F_v$ has state space $\mathcal{X}_v \cup \{\phi\}$ and conditional distributions in the intervention diagram are

$$p'(x_v \mid x_{\mathrm{pa}(v)}, f_v) = \begin{cases} p(x_v \mid x_{\mathrm{pa}(v)}) & \text{if } f_v = \phi \\ \delta_{x_v, x_v^*} & \text{if } f_v = x_v^*, \end{cases}$$

where $\delta_{xy}$ is Kronecker's symbol

$$\delta_{xy} = \begin{cases} 1 & \text{if } x = y \\ 0 & \text{otherwise.} \end{cases}$$

$F_v$ is *forcing* the value of $X_v$ when $F_v \neq \phi$.

# Intervention diagrams

In more general setup, $F_v$ can have parents and decision policies $\pi$ can be specified.

Intervention diagrams similar to LIMIDS, but without utility nodes.

$F_v$ correspond to *decision nodes* in LIMIDS, only with special relation to its child $v$.

When $F_v$ has no parents it holds that

$$p(x) = p'(x \,|\, F_v = \phi, v \in A),$$

but also

$$\begin{aligned} p(x \,||\, x^*_B) &= P(X = x \,|\, X_B \leftarrow x^*_B) \\ &= P'(x \,|\, F_v = x^*_v, v \in B, F_v = \phi, v \in B \setminus A), \end{aligned}$$

# Identifiability of causal effects

Treatment variable $t$, response $r$, set of observed covariates $C$, unobserved variables $U$.

*When and how can* $p(X_r \,||\, x_t)$ *be calculated from* $p(x_t, x_r, x_C)$, the latter in principle being observable from data?

Answer can be found by analysing intervention diagram.

Simplest cases known as *back-door* and *front-door* criteria and formulae.

# Back-door criterion and formula

$\mathcal{D}'$ denotes $\mathcal{D}$ augmented with $F_t$.

Assume $C \supseteq C_0$, where $C_0$ satisfies

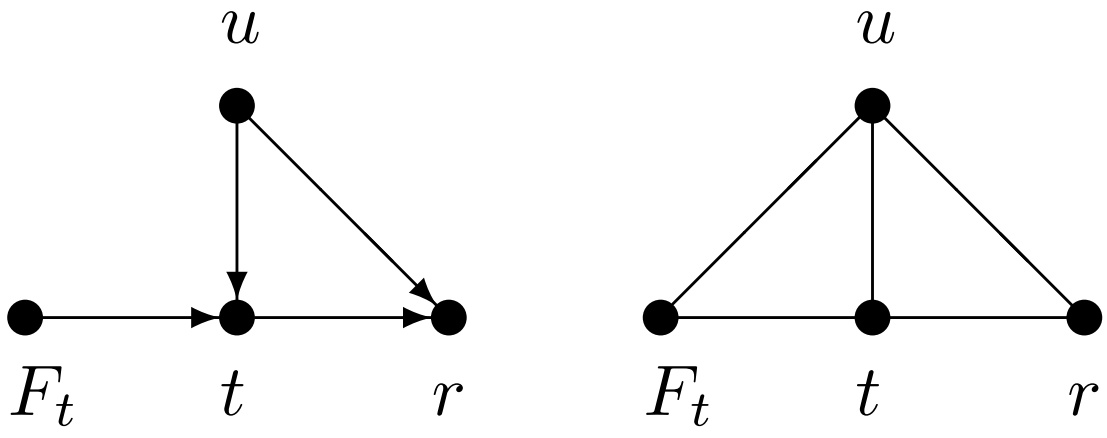
(BD1) Covariates in $C_0$ are unaffected by an intervention: $C_0 \perp_{\mathcal{D}'} F_t$;

(BD2) Intervention only affects response through the treatment it chooses: $R \perp_{\mathcal{D}'} F_t \,|\, C_0 \cup \{t\}$.

Then $C$ identifies the effect of the treatment $t$ on $R$ as

$$p(x_r \,||\, x_t^*) = \sum_{x_{C_0}} p(x_r \,|\, x_{C_0}, x_t^*) p(x_{C_0}).$$

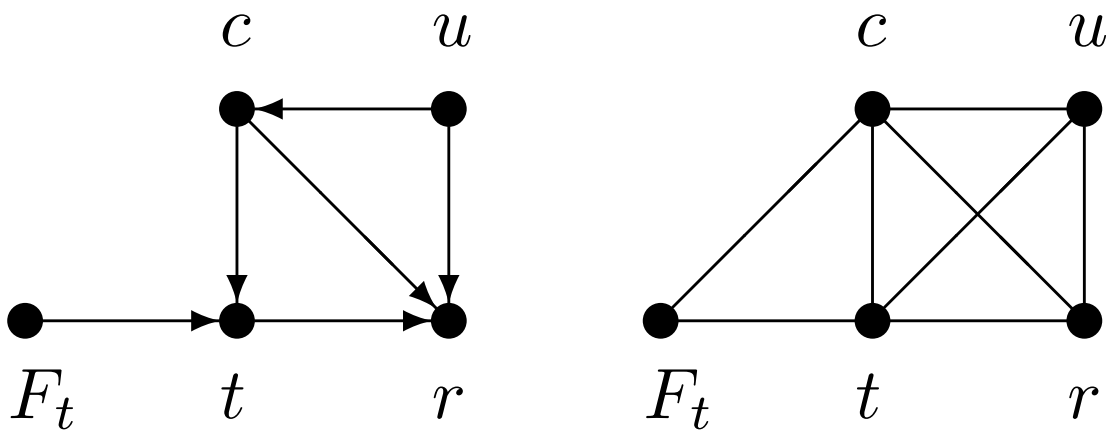
# Confounding




The unobserved *confounder* $X_u$ is affecting both treatment and response.

BD2 is violated; graph to the right reveals that $F_t$ is *not* $d$-separated from $r$ by $t$.

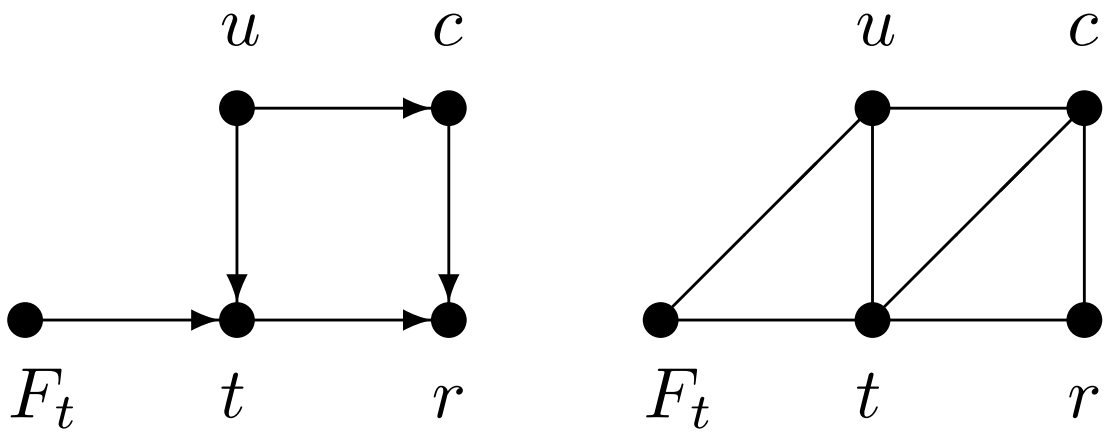
# Randomisation




When $X_t$ is randomised, possibly depending on observed covariate $c$, confounding is resolved.

Now $F_t \perp_{\mathcal{D}'} r \mid \{c, t\}$ and the treatment effect is identifiable.

# Sufficient covariate

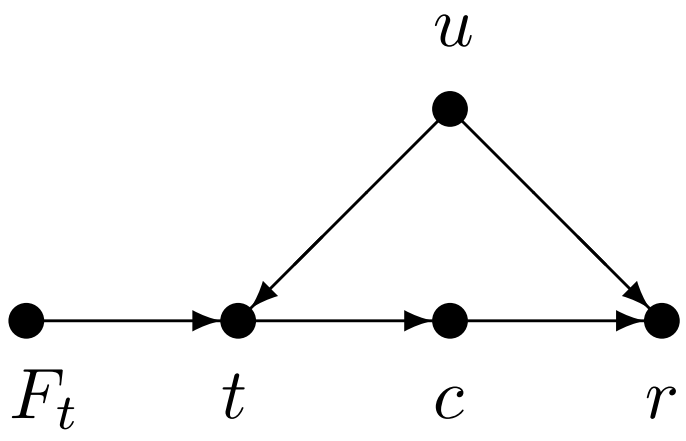



Alternatively, an observed covariate $c$ can ‘screen away’ the confounding effect on the treatment.

Also here, $F_t \perp_{\mathcal{D}'} r \mid \{c, t\}$ and the treatment effect is identifiable.

Assumption slightly more dubious.

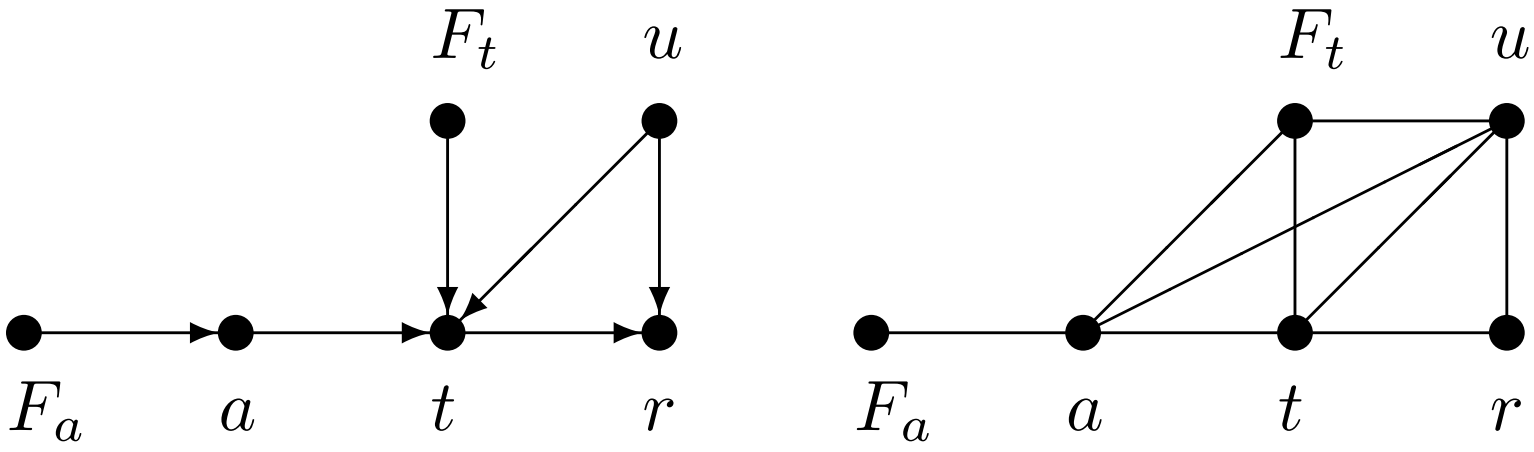
# Front-door formula




In this case $c$ is the *agent* through which the treatment effects the response. Then one can show

$$p(x_r \,||\, x_t^*) = \sum_{x_c} p(x_c \,|\, x_t^*) \sum_{x_t} p(x_r \,|\, x_c, x_t) p(x_t).$$

# Partial compliance




$a$ is treatment assigned, $t$ is treatment taken.

The graph to the right reveals that $r \perp_{\mathcal{D}'} F_a \mid \{a, t\}$ so the effect of the treatment assignment is identified.

However, $r$ is not $d$-separated from $F_t$ by $t$ so the effect of the treatment itself cannot be identified.

# Mapping variables

In a structural equation system

$$X_v \leftarrow g_v(x_{\mathrm{pa}(v)}, U_v),$$

each $(g_v, u_v)$ defines a map $\omega_v : \mathcal{X}_{\mathrm{pa}(v)} \to \mathcal{X}_v$ as

$$\omega_v(x_{\mathrm{pa}(v)}) = g_v(x_{\mathrm{pa}(v)}, u_v)$$

Different $u_v$ may lead to same map.

If some of $\mathrm{pa}(v)$ are unobserved, we may consider them as part of $U_v$, just losing the independence among $U_v$.

Conversely, from *mapping variables* $\omega_v$, we can define $g_v^*$

$$g_v^*(x_{\mathrm{pa}(v)}, \omega_v) = \omega_v(x_{\mathrm{pa}(v)}).$$

# Potential responses

Since now

$$g_v(x_{\mathrm{pa}(v)}, u_v) = g_v^*(x_{\mathrm{pa}(v)}, \omega_v) = \omega_v(x_{\mathrm{pa}(v)}),$$

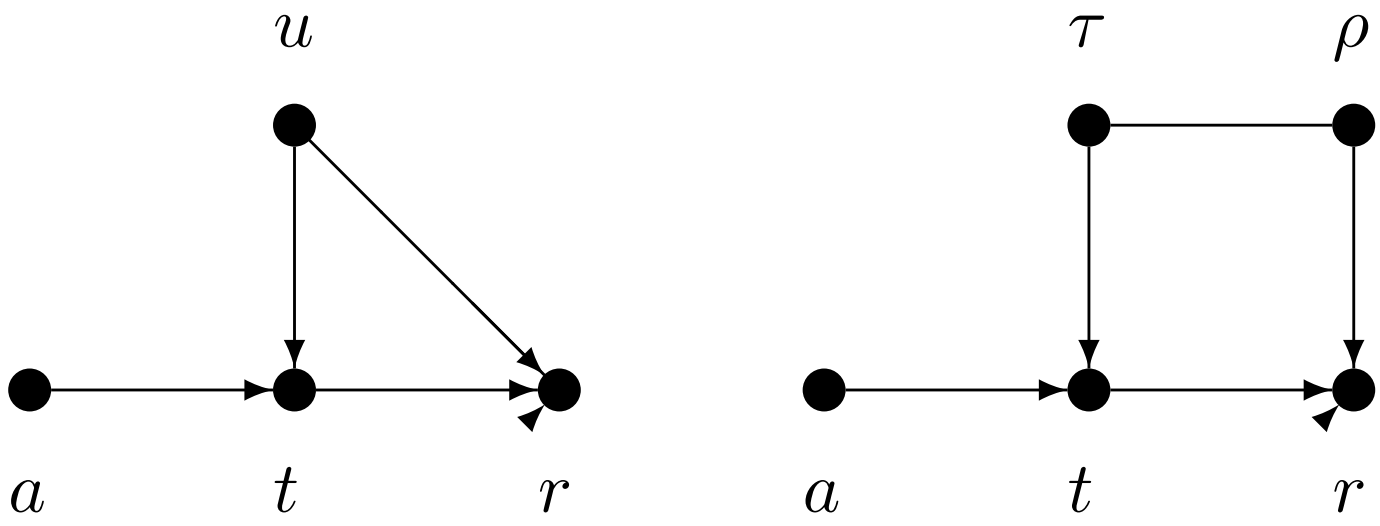
we obtain an observationally equivalent structural equation system

$$X_v \leftarrow g_v^*(X_{\mathrm{pa}(v)}, \Omega_v), v \in V,$$

for random maps $\Omega_v$, a system of *canonical functional form.*

Mapping variables $\omega_v(x_{\mathrm{pa}(v)})$ describe the *potential responses*, i.e. the values of $X_v$ that would have been observed, had the parent configuration been $x_{\mathrm{pa}(v)}$.

# Partial compliance and mapping variables




$$\omega_\tau : \mathcal{X}_a \to \mathcal{X}_t, \quad X_t(x_a, \omega_\tau) \leftarrow \omega_\tau(x_a) = g_t(x_a, x_u, U_t)$$

$$\omega_\rho : \mathcal{X}_t \to \mathcal{X}_r, \quad X_r(x_t, \omega_\rho) \leftarrow \omega_\rho(x_t) = g_r(x_t, x_u, U_r).$$

Undirected link between $\tau$ and $\rho$ indicates possible dependence.

# Possible maps

Four possible maps of each if all observed variables are binary:

The maps $\omega_\tau$ may well be called

$$\{\textit{always taker}, \textit{never taker}, \textit{complier}, \textit{defier}\},$$

so that

$$\textit{always taker}\,(x_a) = 1, \quad \textit{complier}\,(x_a) = x_a, \text{ etc.}$$

Similarly the four values of $\omega_\rho$ may be called

$$\{\textit{always cured}, \textit{never cured}, \textit{beneficial}, \textit{damaging}\}.$$

# Causal discovery and structural learning

$V$ variables. DAG $\mathcal{D}$ unknown and $P$ given.

Assume $P$ *faithful* to $\mathcal{D}$:

$$X_A \perp\!\!\!\perp X_B \mid X_S \iff A \perp_{\mathcal{D}} B \mid S$$

*Most distributions are faithful*

*Find $\mathcal{D}$ matching conditional independences of $P$.*

$\mathcal{D}$ and $\mathcal{D}'$ are *Markov equivalent* if the separation relations $\perp_{\mathcal{D}}$ and $\perp_{\mathcal{D}'}$ are identical.

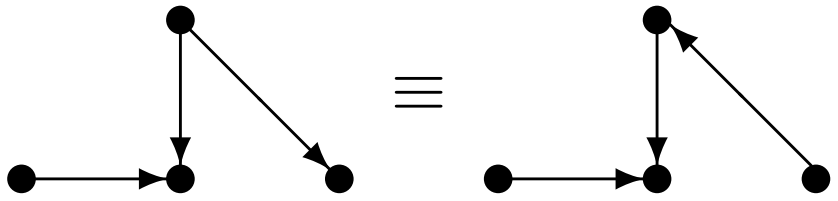
*$\mathcal{D}$ can only be determined up to Markov equivalence.*
Only “causal” aspect is causal motivation for looking for DAGs.

# Markov equivalence

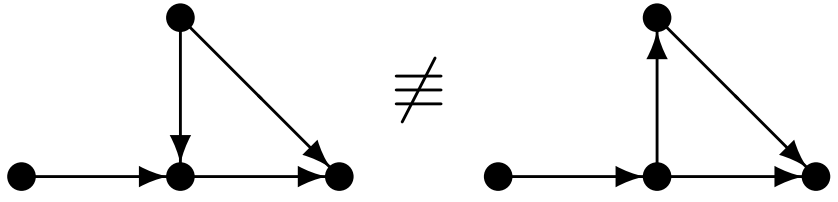
*$\mathcal{D}$ and $\mathcal{D}'$ are equivalent if and only if:*

1. $\mathcal{D}$ and $\mathcal{D}'$ have same *skeleton* (ignoring directions)
2. $\mathcal{D}$ and $\mathcal{D}'$ have same unmarried parents

so



but



# Constraint-based search

**Step 1:** Identify skeleton, using that, for a faithful distribution

$$u \not\sim v \iff \exists S \subseteq V \setminus \{u, v\} : X_u \perp\!\!\!\perp X_v \mid X_S.$$

Begin with complete graph and check first for $S = \emptyset$ and remove edges when independence holds. Then continue for increasing cardinality of $S$.

*PC-algorithm* exploits that only $S$ with $S \subseteq \mathrm{ne}(u)$ or $S \subseteq \mathrm{ne}(v)$ needs checking, where $\mathrm{ne}$ refers to current skeleton graph.

**Step 2:** Identify directions to be consistent with independence relations found in Step 1.

# Exact properties of PC-algorithm

*If $P$ is faithful to DAG $\mathcal{D}$, PC-algorithm finds $\mathcal{D}'$ equivalent to $\mathcal{D}$.*

It uses $N$ independence checks where $N$ is at most

$$N \leq 2\binom{|V|}{2}\sum_{i=0}^{d}\binom{|V|-1}{i} \leq \frac{|V|^{d+1}}{(d-1)!},$$

where $d$ is the maximal degree of any vertex in $\mathcal{D}$.

So worst case complexity is exponential, but algorithm fast for sparse graphs.

# Equivalence class searches

Searches directly in equivalence classes of DAGS.

Define *score function* $\sigma(P, \mathcal{D})$, measuring the adequacy of $\mathcal{D}$ for $P$ with the property that

$$\mathcal{D} \equiv \mathcal{D}' \implies \sigma(P, \mathcal{D}) = \sigma(P, \mathcal{D}').$$

Typically the score function will penalise $\mathcal{D}$ with unnecessary many links.

Equivalence class with maximal score is sought.

# Greedy equivalence class search

1. Initialize with empty DAG
2. Repeatedly search among equivalence classes with a single additional edge and go to class with highest score - until no improvement.
3. Repeatedly search among equivalence classes with a single edge less and move to one with highest score - until no improvement.

*For suitable score functions, this algorithm identifies correct equivalence class for* $P$. (Chickering 2002)

# Data uncertainty and structural learning

Situation less clear if $P$ is not known, but estimated:

**Constraint-based:** Independence checks may randomly give errors.

Algorithms more robust than PC exist.

Most checks are made with separation set $S$ small, so ‘power’ high.

Asymptotically correct if e.g. marginal BIC used in checks.

**Greedy equivalence search:** Asymptotically correct if using BIC or fully Bayesian approach.

# Latent variables and selection

More serious that *one would rarely expect all causally relevant variables to be measured.* Selection effects are also an issue.

More relevant to assume data obtained from $P$ by *marginalisation* to subset $V$ and *conditioning* with subset $C$ so $W = V \cup U \cup C$, data represents $P_V^C$, where $P$ is faithful to some DAG $\mathcal{D}$.

Graphs that describe independence relations in such cases are *Maximal Ancestral Graphs* (Richardson and Spirtes 2002)

Constraint-based methods for identifying MAGs exist.

Bayesian approach seems out of hand.

# References


Chickering, D. M. (2002). Optimal structure identification with greedy search. *Journal of Machine Learning Research*, **3**, 507–54.

Dawid, A. P. (2002). Influence diagrams for causal modelling and inference. *International Statistical Review*, **70**, 161–89.

Lauritzen, S. L. (2001). Causal inference from graphical models. In *Complex stochastic systems*, (ed. O. E. Barndorff-Nielsen, D. R. Cox, and C. Klüppelberg), pp. 63–107. Chapman and Hall/CRC Press, London/Boca Raton.

Lauritzen, S. L. and Nilsson, D. (2001). Representing and solving decision problems with limited information. *Management Science*, **47**, 1238–51.

Pearl, J. (2000). *Causality: Models, reasoning, and inference*. Cambridge University Press, Cambridge, UK.

Richardson, T. S. and Spirtes, P. (2002). Ancestral graph Markov models. *Annals of Statistics*, **30**, 963–1030.

Spirtes, P., Glymour, C., and Scheines, R. (1993). *Causation, prediction and search*. Springer-Verlag, New York. Reprinted by MIT Press.