

# Graphs for margins of Bayesian networks.

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# Correlation does not imply causation

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## How a short nap can raise blood pressure and cholesterol: Study finds people who take a siesta are more likely to have high blood pressure and high cholesterol

• **Napping for more than 30 minutes at a time**

598 shares

They were much favoured by Margaret Thatcher, Albert Einstein and even the Beatles. But while afternoon naps may revitalise tired brains, the same is not true for everyone, according to new research.



ELSEVIER

### Sleep Medicine

Volume 14, Issue 10, October 2013, Pages 950–954



Original Article

### Longer habitual afternoon napping is associated with a higher risk for impaired fasting plasma glucose and diabetes mellitus in older adults: results from the Dongfeng–Tongji cohort of retired workers

Weimin Fang<sup>a, b</sup>, Zhongliang Li<sup>a</sup>, Li Wu<sup>a</sup>, Zhongqiang Cao<sup>a</sup>, Yuan Liang<sup>a, c</sup>, Handong Yang<sup>a</sup>, Youjie Wang<sup>a, b</sup>, Tangchun Wu<sup>a</sup>

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“Dr Matthew Hobbs, head of research for Diabetes UK, said there was no proof that napping **actually caused** diabetes.”

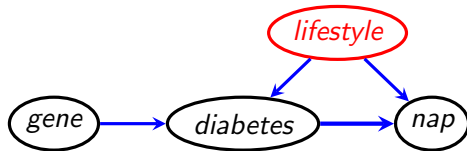
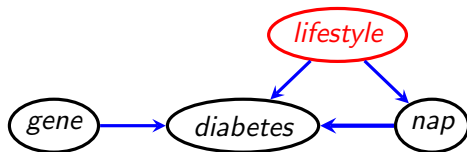
Abstract

Objectives

Afternoon napping is a common habit in China. We used data obtained from the Dongfeng–Tongji cohort to examine if duration of habitual afternoon napping was associated with risks for impaired fasting plasma glucose (IFG) and diabetes mellitus (DM) in a Chinese elderly population.

Methods

# Distinguishing Between Causal Models



In order to compare the models, we need to understand in what ways causal models will differ, both:

- observationally;
- under interventions.

# Outline

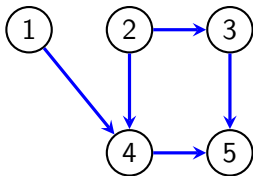
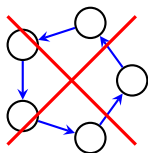
- 1 Introduction
- 2 DAGs
- 3 Margins of DAG Models
- 4 mDAGs
- 5 Markov Equivalence
- 6 Summary

# Directed Acyclic Graphs

vertices 

edges 

no directed cycles



directed acyclic graph (DAG),  $\mathcal{G}$

If  $w \rightarrow v$  then  $w$  is a **parent** of  $v$ :  $\text{pa}_{\mathcal{G}}(4) = \{1, 2\}$ .

If  $w \rightarrow \dots \rightarrow v$  then  $w$  is a **ancestor** of  $v$

# DAG Models

vertex

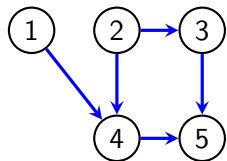


random variable

$X_a$



graph  $\mathcal{G}$



model  $\mathcal{M}(\mathcal{G})$

$P : X_i \perp\!\!\!\perp X_{\text{pre}(i)} \mid X_{\text{pa}(i)} [P]$   
ordered local Markov property



So in example above:

$$X_2 \perp\!\!\!\perp X_1$$

$$X_4 \perp\!\!\!\perp X_3 \mid X_1, X_2$$

$$X_3 \perp\!\!\!\perp X_1 \mid X_2$$

$$X_5 \perp\!\!\!\perp X_1, X_2 \mid X_3, X_4$$

# Global Markov Property

$P$  satisfies the **global Markov property** if for all sets  $A, B, C$ ,

$$A \text{ d-separated from } B \text{ by } C \implies X_A \perp\!\!\!\perp X_B \mid X_C [P].$$

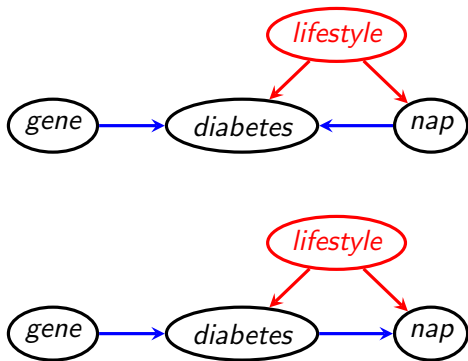
Theorem (Lauritzen et al, 1990)

$P$  satisfies the global Markov property if and only if it satisfies the ordered local Markov property.

**Point:** the model can be defined in terms of 'paths of information'.

# Causal Interventions

If we interpret the DAG as representing structural assumptions, then if we intervene on a node, the graph of the resulting model is just locally altered:



so if we force people to stop napping...



# Latent Projection

Can preserve conditional independences and causal coherence with latents using paths. DAG  $\mathcal{G}$  on vertices  $V = O \dot{\cup} U$ , define **latent projection**  $\mathcal{G}(O)$  as follows: (Verma and Pearl, 1992)

Whenever there is a path of the form



add



Whenever there is a path of the form

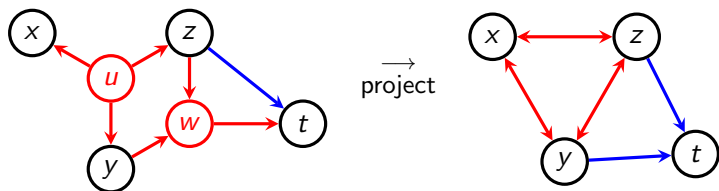


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Then remove the latent variables  $U$  from the graph.

# ADMGs



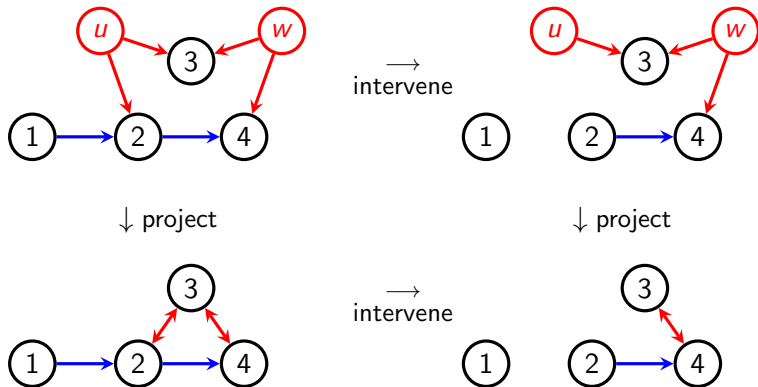
Latent projection leads to an **acyclic directed mixed graph** (ADMG) (equivalent to summary graph without undirected edges).

Can read off independences with d/m-separation. Like an ancestral graph, these are precisely observable independences from the original DAG. See Richardson (2003) for more details.

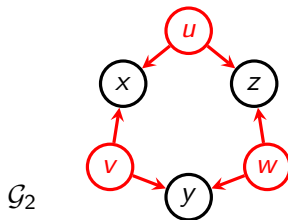
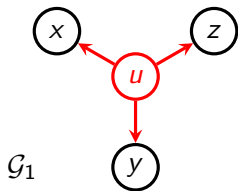
In addition, can see that projection preserves the causal structure; Verma and Pearl (1992).

# Causal Coherence

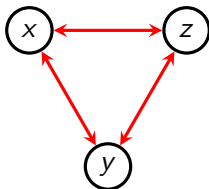
If we intervene on some observed variables, this 'breaks' their dependence upon their parents.



# ADMGs are not sufficient



These have the same latent projection:



But the model over  $(x, y, z)$  in  $\mathcal{G}_2$  is not saturated. Still true if we dichotomize.

# The Problem

- Verma and Pearl's latent projection only uses paths, which are inherently 'pairwise';
- the paths are objects suited to conditional independence, but not all constraints on margins are conditional independences;
- ADMGs are not a sufficiently rich class of graphs to capture the different models one can obtain.

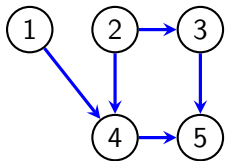
# Structural Equation Model View

There is another way to think about DAG models (e.g. Lauritzen et al, 1990).

$P \in \mathcal{M}(\mathcal{G})$  iff there exist functions  $f_i$  and independent variables  $E_i$  such that recursively setting

$$X_i = f_i(X_{\text{pa}(i)}, E_i)$$

gives  $X_V$  the distribution  $P$ .



$$X_1 = f_1(E_1)$$

$$X_2 = f_2(E_2)$$

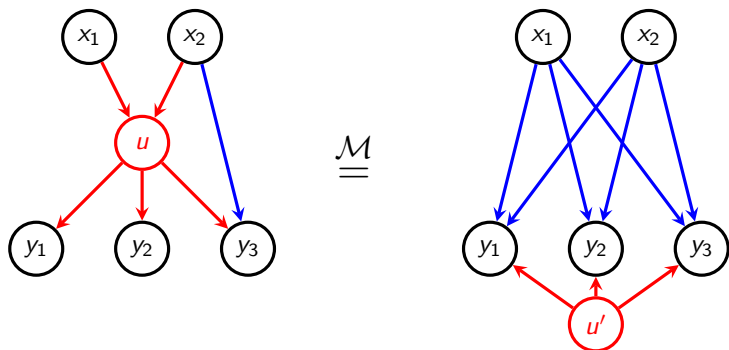
$$X_3 = f_3(X_2, E_3)$$

$$X_4 = f_4(X_1, X_2, E_4)$$

$$X_5 = f_5(X_3, X_4, E_5).$$

# Simplifications

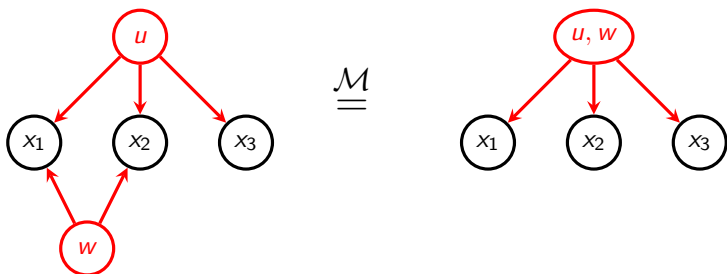
**Simplification 1.** WLOG latent vertices have no parents.



(Of course, this is not true if we assume a specific state-space: e.g. phylogenetic model)

# Simplifications

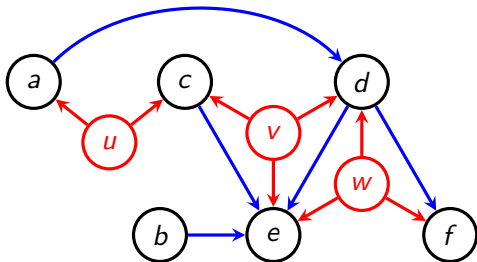
**Simplification 2.** If  $u, w$  are latent with  $\text{ch}_G(w) \subseteq \text{ch}_G(u)$ , then we don't need  $w$ .





# mDAGs

So we only need to consider models like this:



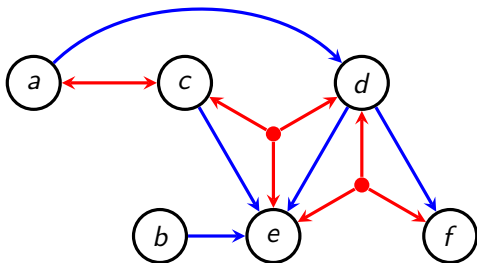
...which we represent with a hyper-graph called an **mDAG**.

Formally, an mDAG on  $V$  is a DAG (in blue), together with some inclusion maximal collection subsets of size at least 2 (red).

Going backwards and replacing bidirected edges with latents gives us the **canonical DAG**  $\bar{\mathcal{G}}$ .

# mDAGs

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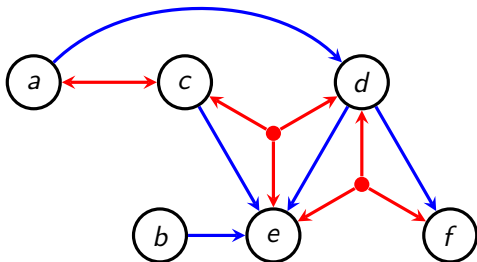


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Going backwards and replacing bidirected edges with latents gives us the **canonical DAG**  $\bar{\mathcal{G}}$ .

# Markov Properties



Given an mDAG  $\mathcal{G}$  and distribution  $P$ , say  $P$  is in the **complete model** for  $\mathcal{G}$ , or  $P \in \mathcal{M}(\mathcal{G})$  if it is the margin of some distribution in the model for the canonical DAG  $\mathcal{M}(\bar{\mathcal{G}})$ .

There are other (weaker) properties Shpitser et al. (2014).

# Latent Projection for mDAGs

For mDAG  $\mathcal{G}$  and subset of vertices  $O$ , form latent projection  $p(\mathcal{G}, O)$  by:

Whenever there is a path of the form

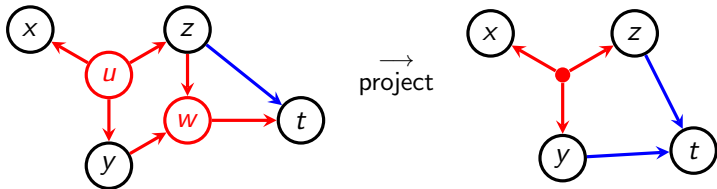


add



Whenever there is a maximal set  $B = \{x_1, x_2, \dots, x_k\}$  such that these variables share a hidden common cause, add hyper-edge  $B$ .

Then remove the latent variables  $U$  from the graph.



# Results

The mDAG latent projection preserves the distinction between models.

Theorem (Evans, 2014)

If  $p(\mathcal{G}, O) = p(\mathcal{G}', O)$  then the models induced by  $\mathcal{M}(\mathcal{G})$  and  $\mathcal{M}(\mathcal{G}')$  on the margin  $O$  are the same.

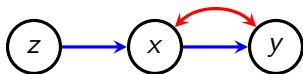
So the problem which arises with ADMGs never occurs for mDAGs.

Theorem (Evans, 2014)

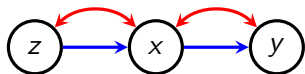
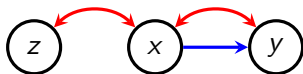
If  $C \subseteq O$  then  $p(\mathcal{G}_{\overline{C}}, O) = p(\mathcal{G}, O)_{\overline{C}}$ ; i.e. the projection respects causal interventions.

# Instrumental Variables

The Instrumental Variables model assumes causally exogenous variable  $z$  affects the treatment  $x$ .

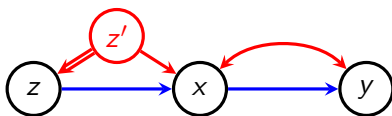


But it's well known that this is observationally indistinguishable from a hidden common cause for  $x$  and  $z$  (e.g. Didelez and Sheehan, 2007).



# Instrumental Variables

To see this, imagine  $z$  is an exact copy of  $z'$ .



Doesn't really matter whether  $x$  gets information from  $z$  or  $z'$ .

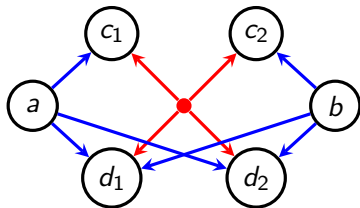
Very hard to see this equivalence with conditional independence.

# Instrument Generalisation

Let  $\mathcal{G}$  have bidirected edge  $B = C \dot{\cup} D$  with:

- Ⓐ every  $c \in C$  contained in no other bidirected edge;
- Ⓑ  $\text{pa}_{\mathcal{G}}(d) \supseteq \text{pa}_{\mathcal{G}}(C)$  for each  $d \in D$ .

Can 'split'  $B$  into  $C$  and  $D$  and add edges  $c \rightarrow d$  where necessary.



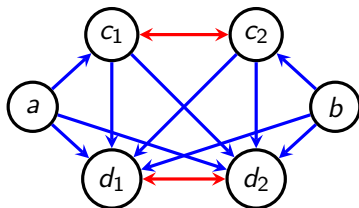


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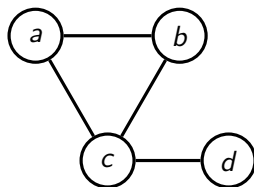
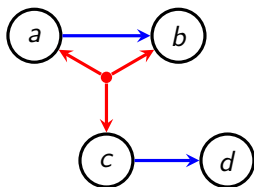
- Ⓐ every  $c \in C$  contained in no other bidirected edge;
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Can 'split'  $B$  into  $C$  and  $D$  and add edges  $c \rightarrow d$  where necessary.



# Skeletons

Define the **skeleton** of two mDAGs as the undirected graph with  $v - w$  whenever  $v$  and  $w$  are contained in some edge together.



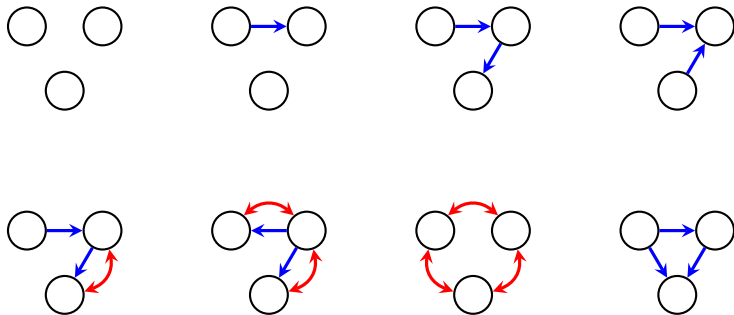
## Theorem

mDAGs with different skeletons induce different models in general.

(Consequence of Theorem 4.2 of Evans, 2012)

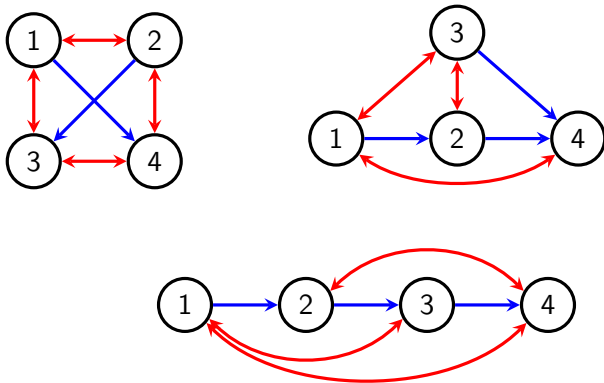
# Equivalence on Three Variables

Combining the previous results, there are 8 Markov equivalence classes on three variables.



## But Not on Four!

On four variables, it's still not clear whether or not the following models are saturated: (they are of full dimension in the discrete case)



# Summary

We have seen that:

- graphs with 'ordinary' edges can give a causally coherent representation of marginal models;
- **but**: ordinary mixed graphs are not rich enough to represent all models;
- mDAGs provide the most general necessary framework for representing causal DAGs under marginalization;
- general Markov equivalence in this class is hard, but we're getting there!

**Thank you!**

# Main References

Didelez and Sheehan. Mendelian Randomisation: Why Epidemiology needs a Formal Language for Causality, *SMMR*, 2007.

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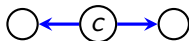
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# d-Separation

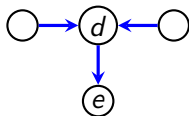
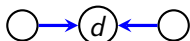
A **path** is a sequence of edges in the graph; vertices may not be repeated.

A path from  $a$  to  $b$  is **blocked** by  $C \subseteq V \setminus \{a, b\}$  if either

(i) any non-collider is in  $C$ :



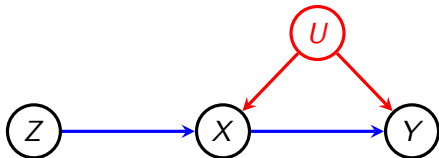
(ii) or any collider is not in  $C$ , nor has descendants in  $C$ :



Two vertices  $a$  and  $b$  are **d-separated** given  $C \subseteq V \setminus \{a, b\}$  if **all** paths are blocked.



# Inequality Results



$$p(x, y | z) = \int p(u) p(x | z, u) \cdot p(y | x, u) du$$

$$\text{Let } p^*(x, y | z) \equiv \int p(u) p(x | z, u) \cdot p(y | x = 0, u) du$$

Can't observe  $p^*$  but:

- **Compatibility:**  $p(0, y | z) = p^*(0, y | z)$  for each  $z, y$ ; and
- **Independence:**  $Y \perp\!\!\!\perp Z$  under  $p^*$ .

This 'compatibility' requirement turns out to place an inequality restriction on  $p$ :

$$\max_x \sum_y \max_z p(x, y | z) \leq 1.$$

# Inequality Results

Generalizing this argument, we find a rich theory of results on inequalities (Evans, 2012).

However these results are **not exhaustive!**

Finding **all** inequality constraints in marginal models is probably an NP hard problem.

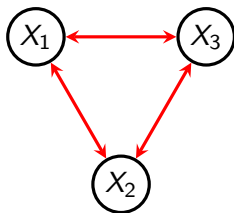
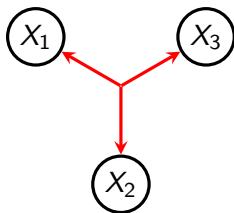
Additionally:

- fitting models with inequality constraints is not trivial;
- the usual asymptotic results do not necessarily apply.

Maybe the nested model is a good compromise!

# ADMGs are not sufficient

In general we need to distinguish between  $\{1, 2, 3\}$  and  $\{1, 2\}$ ,  $\{1, 3\}$ ,  $\{2, 3\}$ .



The model on the right is not saturated. Still true if we dichotomize.

# ADMGs are not sufficient

## Lemma

Let  $\mathcal{F}$ ,  $\mathcal{G}$ ,  $\mathcal{H}$  be mutually independent  $\sigma$ -algebras (so that  $\mathcal{F} \perp\!\!\!\perp \mathcal{G} \vee \mathcal{H}$  and so on), and let  $X$ ,  $Y$  and  $Z$  be random variables such that

- (i)  $X$  is  $\mathcal{F} \vee \mathcal{G}$ -measurable;
- (ii)  $Y$  is  $\mathcal{G} \vee \mathcal{H}$ -measurable;
- (iii)  $Z$  is  $\mathcal{F} \vee \mathcal{H}$ -measurable.

Then  $P(X = Y = Z) > 1 - \epsilon$  implies

$$\text{Var } X < 3\epsilon.$$

# Causal Equivalence

The two mDAGs below are Markov equivalent, and lead to the same graph under any ordinary causal intervention.

