Causal Inference for Health Data

(STATS C160/C260 — Winter 2026)

Lecture 2: Structural Causal Models and Causal Diagrams

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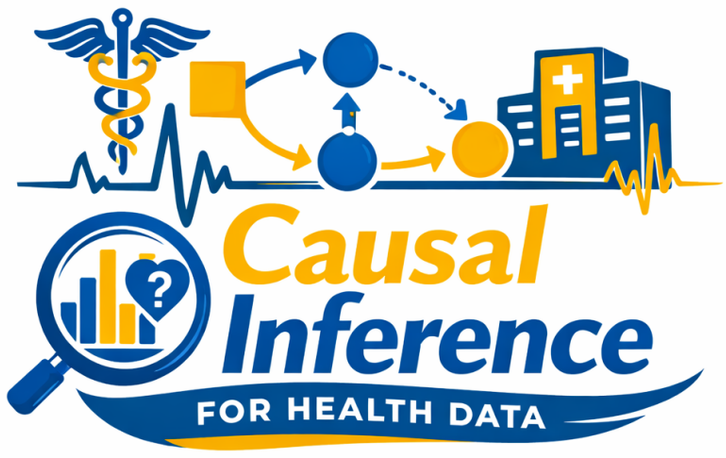


Announcements

- Class projects are individual (not collaborative),
- Reminder: get access to the MIMIC-IV dataset https://physionet.org/content/mimiciv/2.2/),
- Office Hours: 1.45pm-2.45pm today,
- Unofficial waitlist (jose.salas@stat.ucla.edu).

Recap: Simpson's Paradox

	HbA1c low (Y)	HbA1c high ($\neg Y$)		Sucess Rate
drug (X)	20	20	40	50%
no-drug ($\neg X$)	16	24	40	40%
	36	44		

$$P(Y \mid F, X) < P(Y \mid F, \neg X)$$

$$P(Y \mid \neg F, X) < P(Y \mid \neg F, \neg X)$$

but

$$P(Y \mid X) > P(Y \mid \neg X)\ !$$

($\neg F$)	HbA1c low (Y)	HbA1c high ($\neg Y$)		Sucess Rate
drug (X)	18	12	30	60%
no-drug ($\neg X$)	7	3	10	70%
	25	15	40	

(F)	HbA1c low (Y)	HbA1c high ($\neg Y$)		Sucess Rate
drug (X)	2	8	10	20%
no-drug ($\neg X$)	9	21	30	30%
	11	29	40	

Recap: Simpson's Paradox

$$P(Y \mid F, X) < P(Y \mid F, \neg X)$$

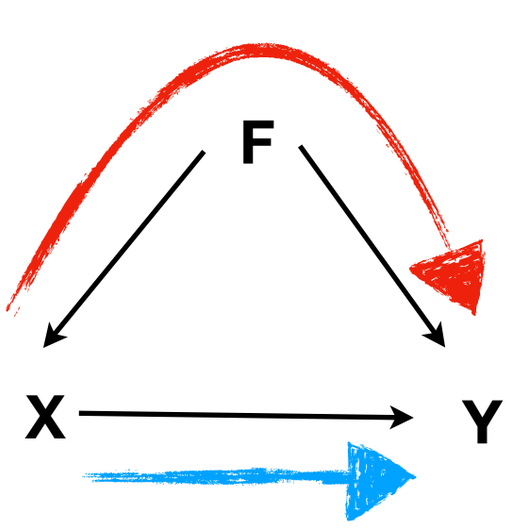
$$P(Y \mid \neg F, X) < P(Y \mid \neg F, \neg X)$$

	HbA1c low (Y)	HbA1c high ($\neg Y$)		Sucess Rate
drug (X)				
no-drug ($\neg X$)				

weight loss ($\neg F$)				cess ate
drug (X)				0%
no-drug ($\neg X$)	7	3	10	70%
	25	15	40	

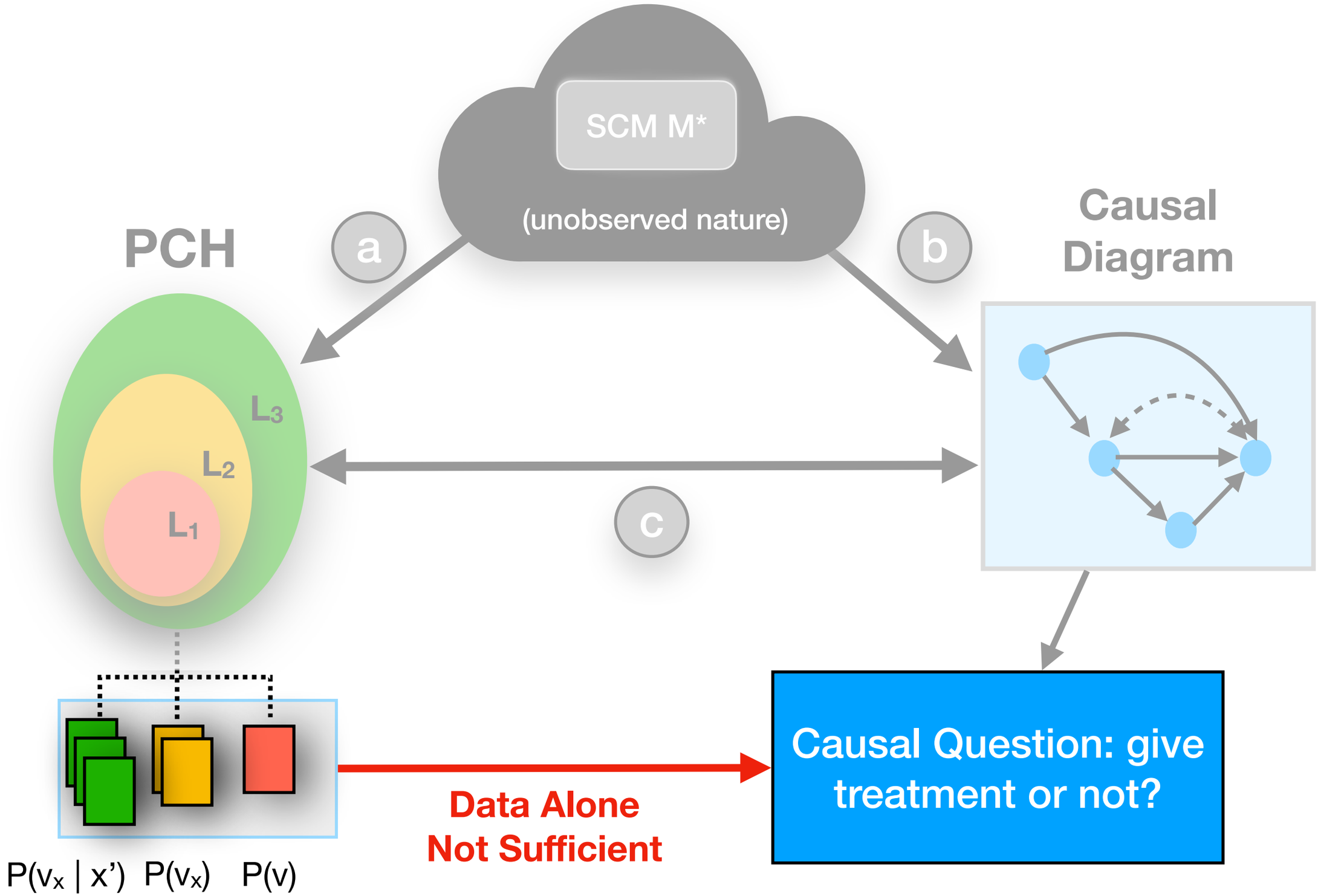
no-drug ($\neg X$)	9	21	30	30%
	11	29	40	

Sex-Stratified



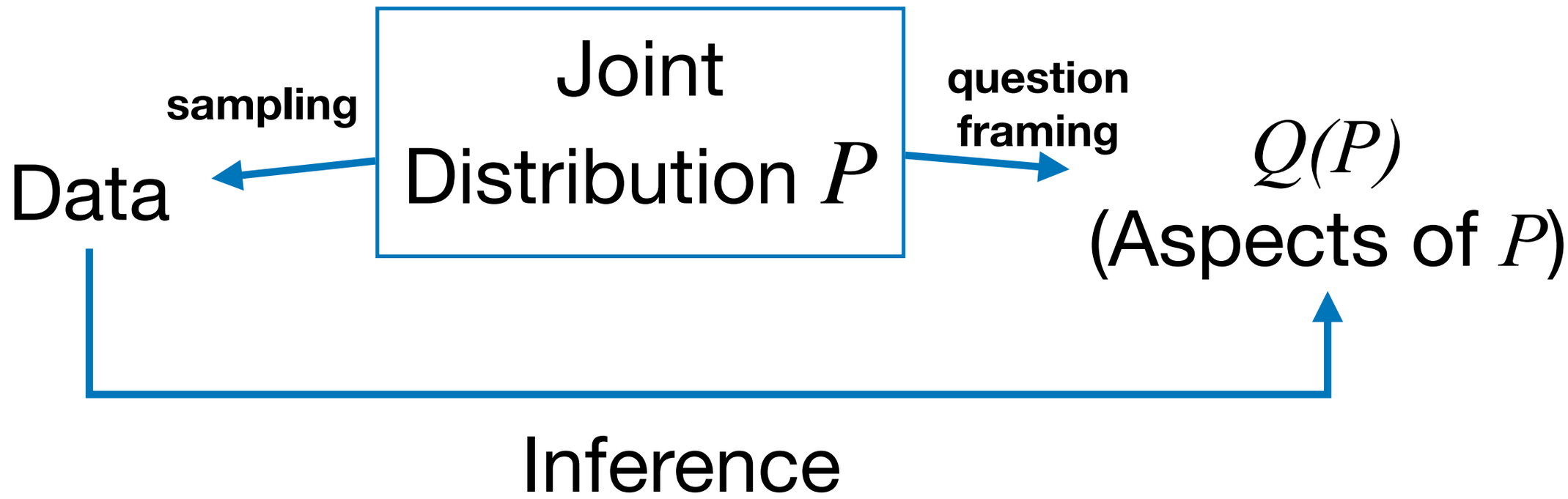
Weight Loss-Stratified

Recall: Data Alone was Not Enough — Why?



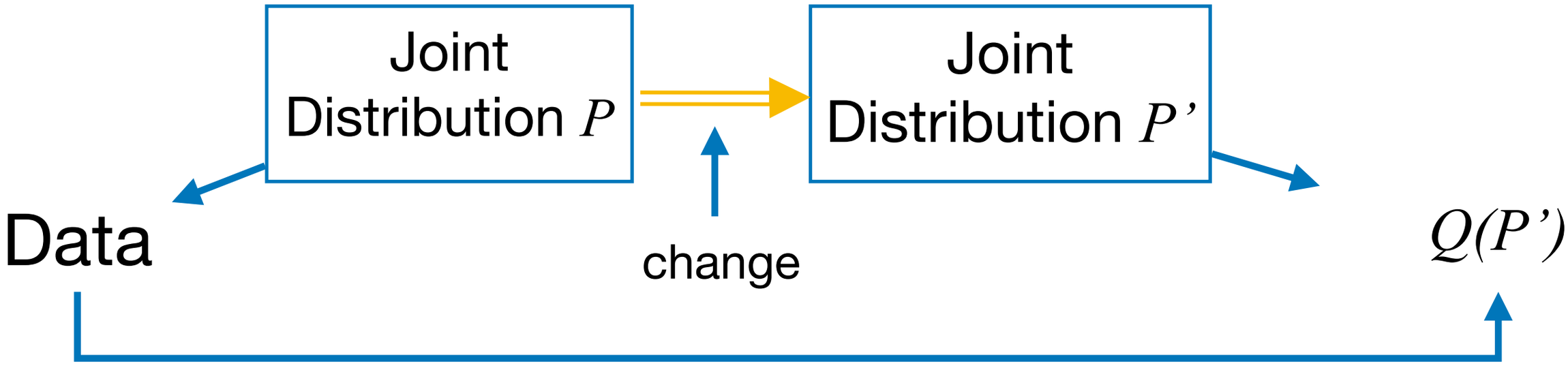
Statistics/ML Inferential Paradigm

- **Approach:** Perform inference based on data.



e.g., infer whether patients with characteristic X, F are likely to succeed in diabetes management Y — i.e., compute $Q = P(Y \mid X, F)$.

From Statistical to Causal Analysis



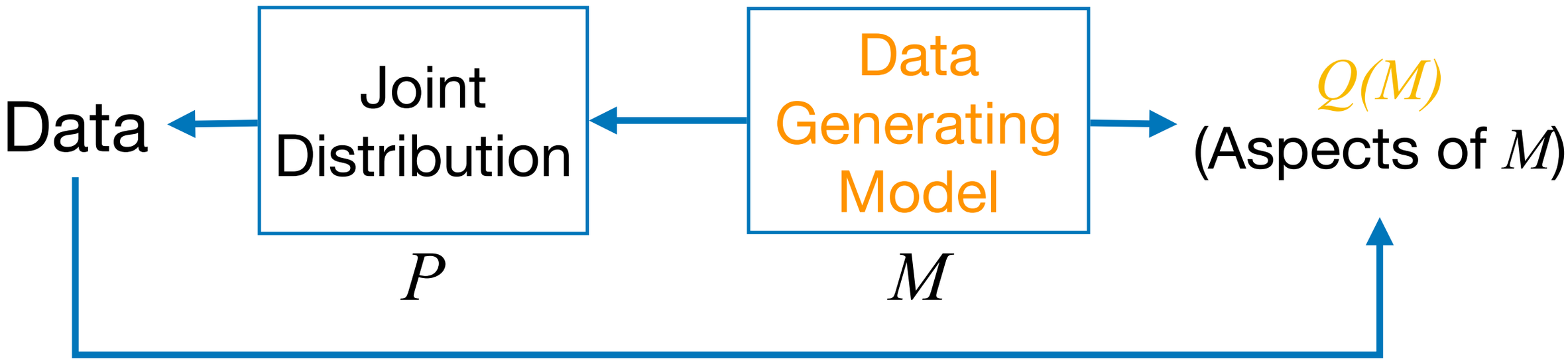
Inference

e.g., estimate $P'(diabetes\ controlled)$ if we *give semaglutide*.

Q: How to leverage *P* (factual) for *P'* (hypothetical)?

Needed: New formalism to represent both P & P'.
P is tied to the data; P' is never observed, no data.

New Oracle — The Structural Causal Model Paradigm



Inference

M – invariant mechanisms or protocols by which Nature assigns values to variables in the analysis.

P - model of data, M - model of reality

Modeling Reality (Example)

Variables we observe ($\mathbf{V}$):

X ($X=1$ for semaglutide, $=0$ for none)

F ($F=1$ for weight loss)

Y ($Y=1$ if patients lower HbA1c)

Modeling Reality (Example)

Variables we observe ($\mathbf{V}$):

X ($X=1$ for semaglutide, $=0$ for none)
F ($F=1$ for weight loss)
Y ($Y=1$ if patients lower HbA1c)

Variables that are unobserved ($\mathbf{U}$):

U_p ($U_p=1$ physician decides to treat)
U_h ($U_h=1$ for healthy enough diet)
U_i ($U_h=1$ for tight insulin control)

Modeling Reality (Example)

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F (F=*1* for weight loss)
Y (Y=*1* if patients lower HbA1c)

Variables that are unobserved ($\mathbf{U}$):

U_p (U_p =*1* physician decides to treat)
U_h (U_h =*1* for healthy enough diet)
U_i (U_h =*1* for tight insulin control)

How are the observed variables determined?

$$X \leftarrow U_p$$
$$F \leftarrow X \vee U_h$$
$$Y \leftarrow (X \wedge F) \vee U_i$$

To succeed, patient is either

- on treatment and loses weight,
- or controls their insulin tightly during observation.

Modeling Reality (Example)

Variables we observe ($\mathbf{V}$):

X ($X=1$ for semaglutide, $=0$ for none)

F ($F=1$ for weight loss)

Y ($Y=1$ if patients lower HbA1c)

How are the observed variables determined?

$X \leftarrow U_p$

$F \leftarrow X \vee U_h$

$Y \leftarrow (X \wedge F) \vee U_i$

Variables that are unobserved ($\mathbf{U}$):

U_p ($U_p=1$ physician decides to treat)

U_h ($U_h=1$ for healthy enough diet)

U_i ($U_h=1$ for tight insulin control)

What is the randomness over the unobserved vars:

$$P(U_p=1)=1/2,\ P(U_h=1)=1/8,\ P(U_i=1)=1/4$$

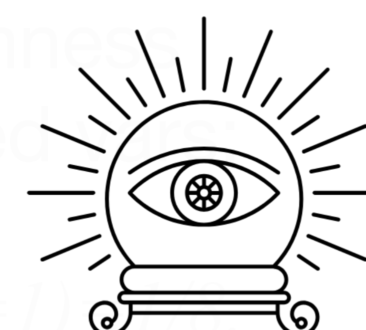
Modeling Reality (Example)

This is a fully specified Model of Reality!

It induces both P and P’ (more details soon).

This will be our Oracle,
which is known as **Structural Causal Model**.

(Now, let’s formalize this object)



Structural Causal Models — Definition

The New Oracle: Structural Causal Models

Definition 2.1.1: A structural causal model (SCM) M is a 4-tuple $<\boldsymbol{V}, \boldsymbol{U}, \mathcal{F}, P(\boldsymbol{u})>$, where

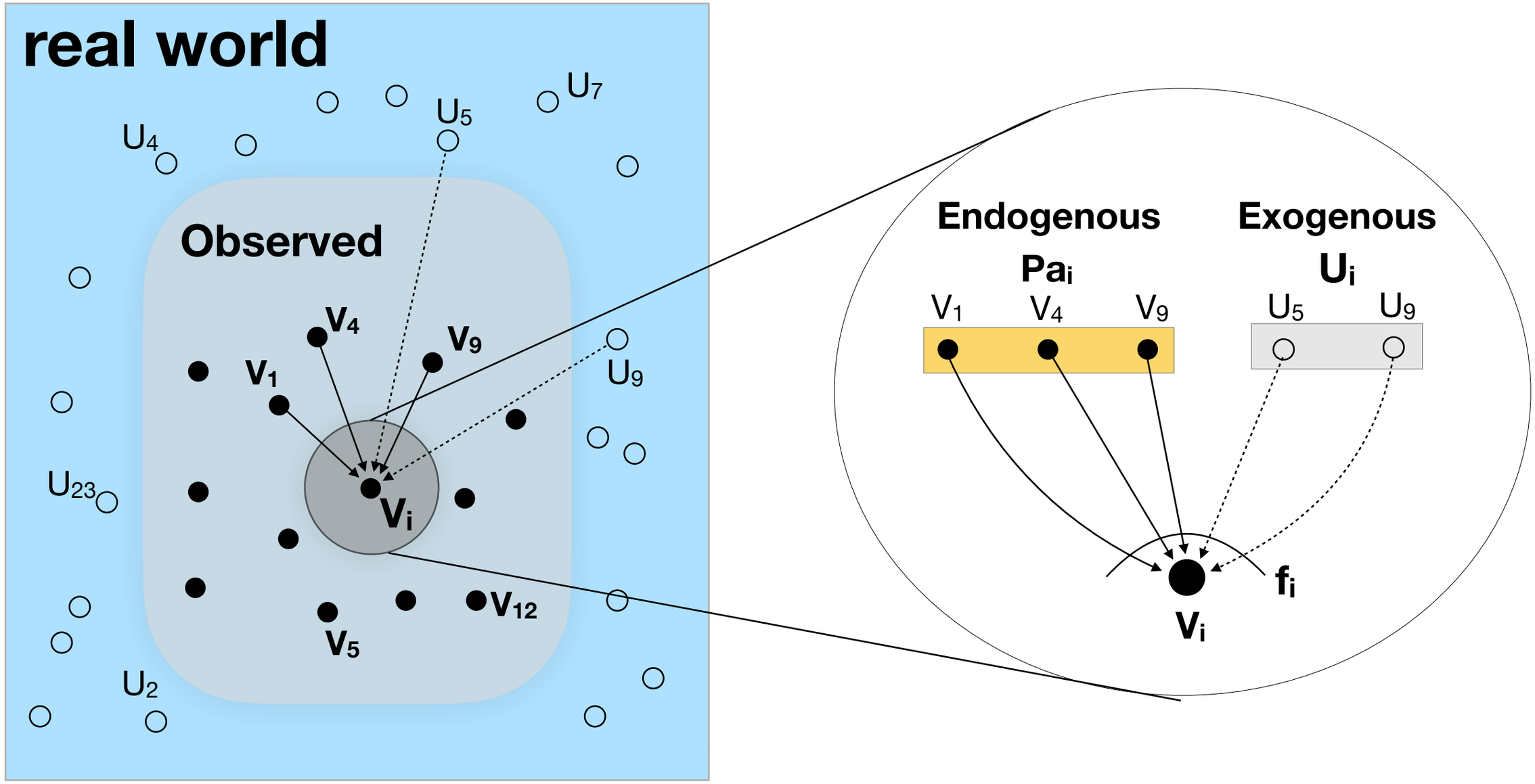
- $\boldsymbol{V} = \{V_1,\ldots,V_n\}$ are endogenous variables;
- $\boldsymbol{U} = \{U_1,\ldots,U_m\}$ are exogenous variables;
- $\mathcal{F} = \{f_1,\ldots, f_n\}$ are functions determining $\boldsymbol{V}$, $v_i \leftarrow f_i(pa_i, u_i),\ Pa_i \subset V_i, U_i \subset U;$
- $P(\boldsymbol{u})$ is a distribution over $\boldsymbol{U}$

Not regression!

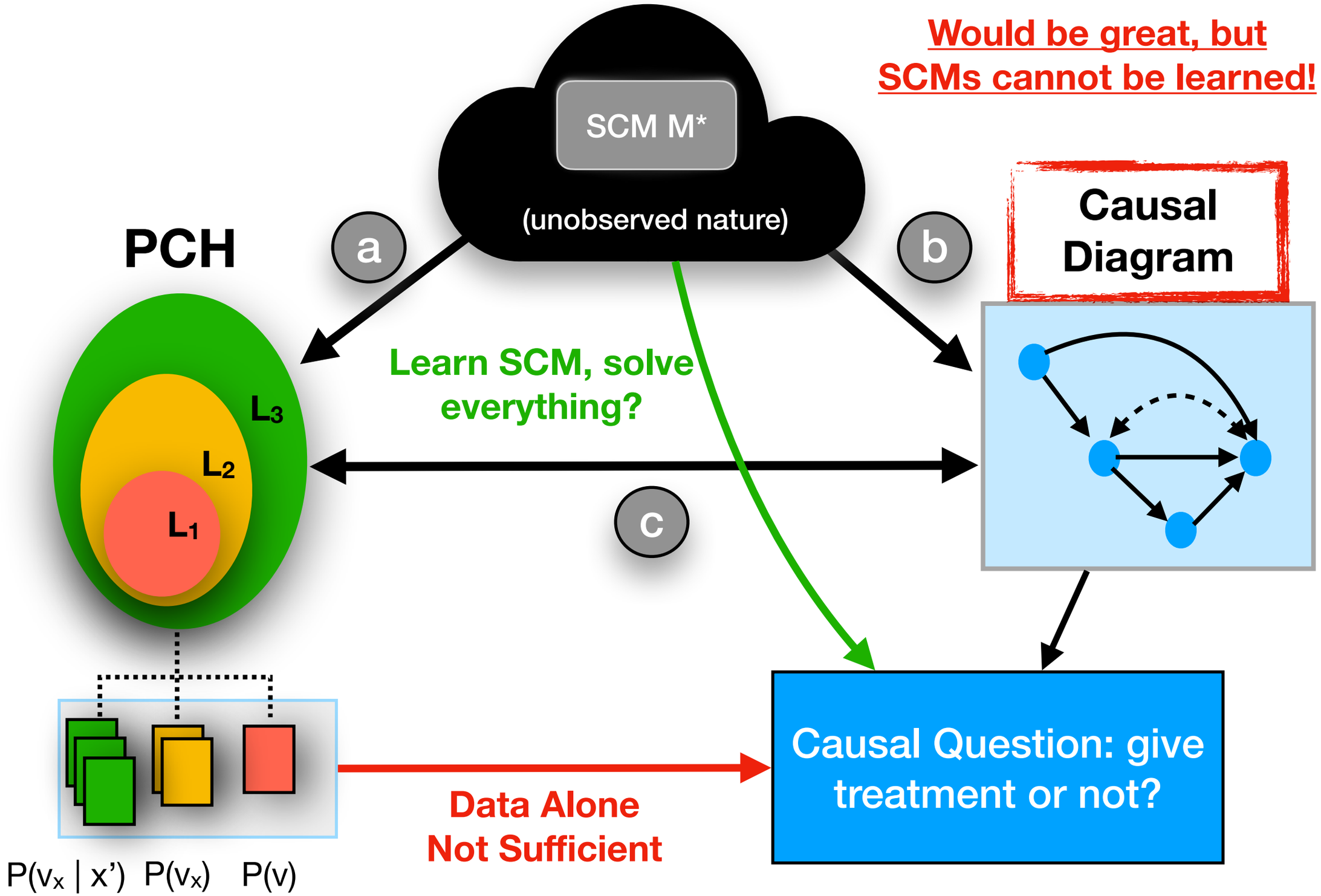
e.g. $y = \alpha + \beta X + U_Y$

Axiomatic Characterization: (Galles-Pearl, 1998; Halpern, 1998).

Structural Causal Models



Are we aiming to learn the SCM?



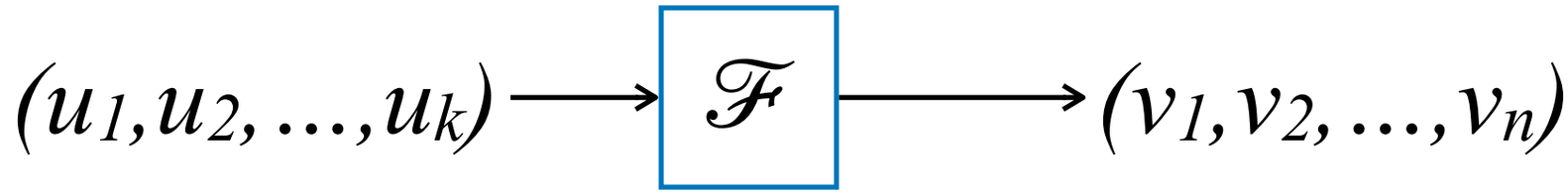
Structural Properties

1. SCM induces an L_1-distribution.
2. SCM defines a causal diagram.
3. SCM spawns the Pearl Causal Hierarchy.

Property 1: SCM induces L_1-distribution.

1. SCM induces distribution $P(\boldsymbol{v})$

- $\mathcal{F}$ can be seen as a mapping from $\boldsymbol{U} \longrightarrow \boldsymbol{V}$



- When the input $\boldsymbol{U}$ is a set of random vars, then the output $\boldsymbol{V}$ also becomes a set of r.v's.
- P(v) is the layer 1 of the PCH, known as the observational (or passive) prob. distribution.
- Each event, person, observation, etc… corresponds to an instantiation of $\boldsymbol{U}=\boldsymbol{u}$.

1. SCM induces distribution $P(\boldsymbol{v})$

- in our example, each patient follows one of eight groups according to the unobservables in the model:

$$\mathscr{F} = \begin{cases} f_X : U_p \\ f_F : X \vee U_h \\ f_Y : (X \wedge F) \vee U_i \end{cases}$$

$\mathscr{F}$

$(U_p{=}1,\ U_h{=}1,\ U_i{=}1) \longrightarrow (X{=}1,\ F{=}1,\ Y{=}1)$

$(U_p{=}0,\ U_h{=}1,\ U_i{=}1) \longrightarrow (X{=}0,\ F{=}1,\ Y{=}1)$

$(U_p{=}1,\ U_h{=}0,\ U_i{=}1) \longrightarrow (X{=}1,\ F{=}1,\ Y{=}1)$

$(U_p{=}1,\ U_h{=}1,\ U_i{=}0) \longrightarrow (X{=}1,\ F{=}1,\ Y{=}1)$

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1. SCM induces distribution $P(v)$

- in our example, each patient follows one of eight groups according to the unobservables in the model:

$$\mathcal{F} = \begin{cases} f_X : U_p \\ f_F : X \vee U_h \\ f_Y : (X \wedge F) \vee U_i \end{cases}$$

$P(u)$

1/64	$(U_p=1,\ U_h=1,\ U_i=1) \longrightarrow (X=1,\ F=1,\ Y=1)$
1/64	$(U_p=0,\ U_h=1,\ U_i=1) \longrightarrow (X=0,\ F=1,\ Y=1)$
7/64	$(U_p=1,\ U_h=0,\ U_i=1) \longrightarrow (X=1,\ F=1,\ Y=1)$
3/64	$(U_p=1,\ U_h=1,\ U_i=0) \longrightarrow (X=1,\ F=1,\ Y=1)$
21/64	$(U_p=1,\ U_h=0,\ U_i=0) \longrightarrow (X=1,\ F=1,\ Y=1)$
3/64	$(U_p=0,\ U_h=1,\ U_i=0) \longrightarrow (X=0,\ F=1,\ Y=0)$
3/64	$(U_p=0,\ U_h=0,\ U_i=1) \longrightarrow (X=0,\ F=0,\ Y=1)$
21/64	$(U_p=0,\ U_h=0,\ U_i=0) \longrightarrow (X=0,\ F=0,\ Y=0)$

X	F	Y	P(x,f,y)
0	0	0	21/64
0	0	1	3/64
0	1	0	3/64
0	1	1	1/64
1	0	0	0
1	0	1	0
1	1	0	0
1	1	1	32/64

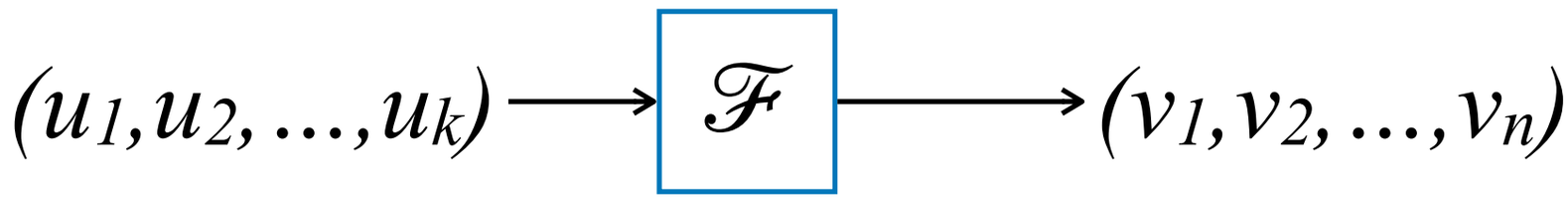
$P(v)$

1. SCM induces distribution $P(\boldsymbol{v})$

- **Observational Distribution** (Def. 2.2.1). An SCM $M = \langle \boldsymbol{V}, \boldsymbol{U}, \mathcal{F}, P(\boldsymbol{u}) \rangle$ defines a joint probability distribution $P^M(V)$ *s.t. for every* $Y \subseteq V$,

$$P^M(y) = \sum_{u|Y(u)=y} P(u)$$

- $\mathcal{F}$ can be seen as a mapping from $\boldsymbol{U} \longrightarrow \boldsymbol{V}$

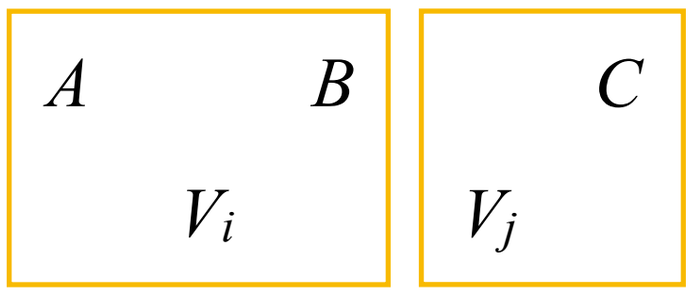


Property 2: SCM defines a causal diagram.

2. SCM → Causal Diagram

- Every SCM M induces a graphical model called causal diagram.
- Represented as a DAG where:
  - Each $V_i \in \boldsymbol{V}$ is a node,

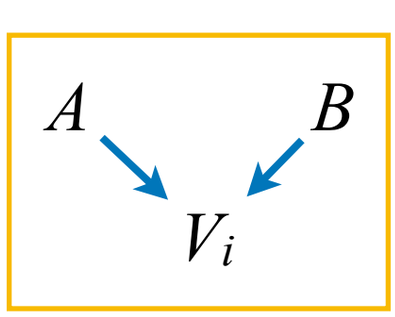
$$V_i \leftarrow f_i(A, B, U)$$
$$V_j \leftarrow f_j(C, U)$$

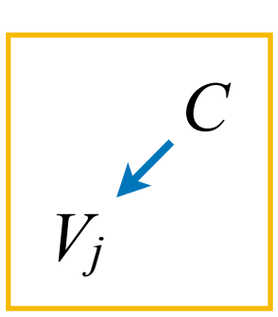


2. SCM → Causal Diagram

- Every SCM M induces a graphical model called causal diagram.
- Represented as a DAG where:
  - Each $V_i \in \boldsymbol{V}$ is a node,
  - There is $W \longrightarrow V_i$ if for $W \in Pa_i$,

$$V_i \leftarrow f_i(A,B,U)$$
$$V_j \leftarrow f_j(C,U)$$





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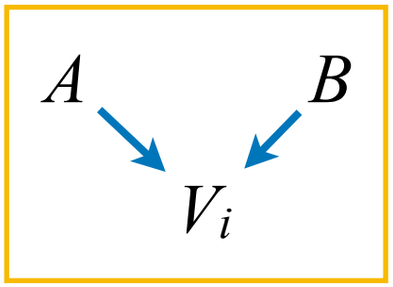
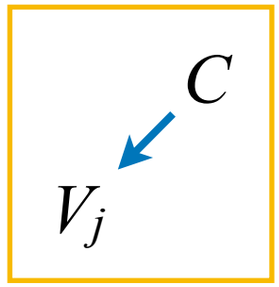
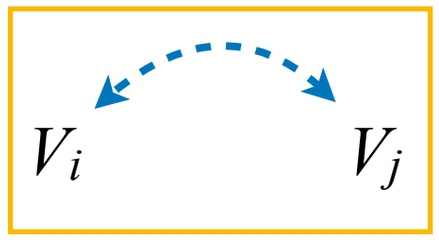
- Each $V_i \in \boldsymbol{V}$ is a node,
- There is $W \longrightarrow V_i$ if $W \in Pa_i$,
- There is $V_i \dashleftarrow\dashrightarrow V_j$ whenever $U_i \cap U_j \neq \varnothing$.

$$V_i \leftarrow f_i(A,B,U)$$
$$V_j \leftarrow f_j(C,U)$$







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- Each $V_i \in \boldsymbol{V}$ is a node,
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$$V_i \leftarrow f_i(A,B,U)$$
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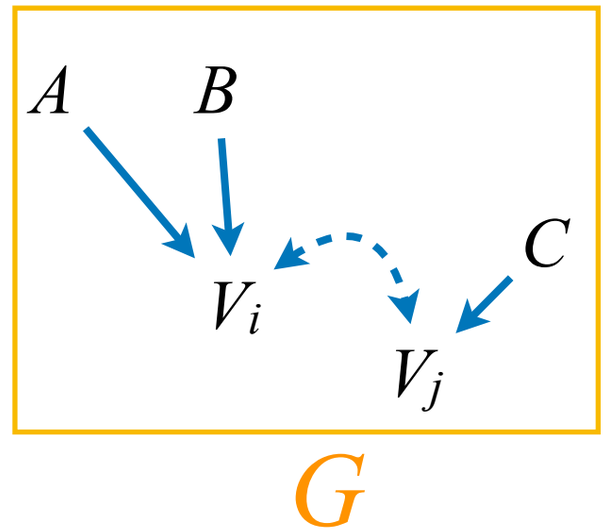


Causal Diagram — Definition

- ***Causal Diagram*** (Def. 2.4.1) — Consider an SCM $M = <\boldsymbol{V}, \boldsymbol{U}, \mathcal{F}, P(\boldsymbol{u})>$. Then G is said to be a causal diagram (of M) if constructed as follows:

1. add vertex for every endogenous variable $\boldsymbol{V_i} \in \boldsymbol{V}$.

2. add edge ($\boldsymbol{V_j} \rightarrow \boldsymbol{V_i}$) for every $\boldsymbol{V_i}, \boldsymbol{V_j} \subset$ V if $\boldsymbol{V_j}$ appears as argument of $\boldsymbol{f_i} \in \mathcal{F}$.

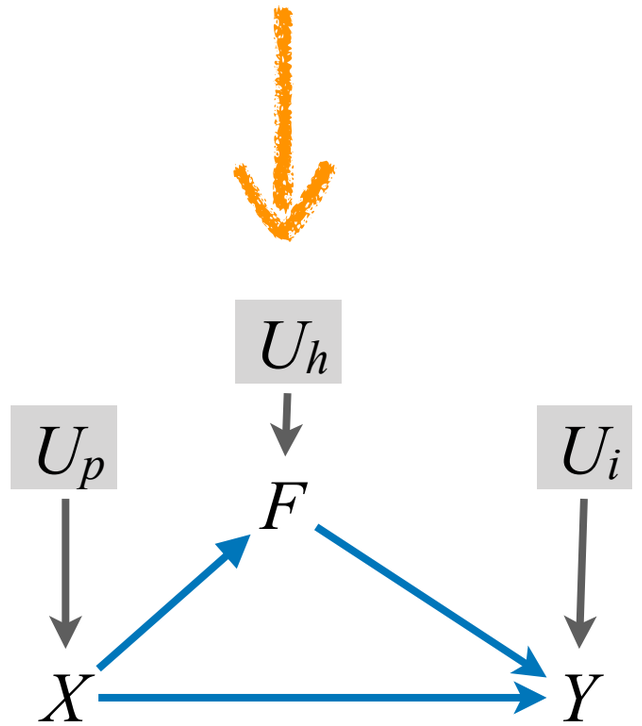
3. add a bidirected edge ($\boldsymbol{V_j} \dashleftarrow\dashrightarrow \boldsymbol{V_i}$) for every $\boldsymbol{V_i}, \boldsymbol{V_j} \subset$ V if $\boldsymbol{U_i}, \boldsymbol{U_j} \subset \boldsymbol{U}$ are correlated or the corresponding functions $\boldsymbol{f_i}, \boldsymbol{f_j}$ share some $U \in$ **U** as argument.

2. SCM → Causal Diagram

Recall the semaglutide example:

- Endogenous (observed) variables $\boldsymbol{V}$:
  - X ($X=1$ for treated, $=0$ for untreated)
  - F ($F=1$ for weight loss, $D=0$ o/w)
  - Y ($Y=1$ if reduced HbA1c, $=0$ o/w)
- Exogenous (unobserved) Variables $\boldsymbol{U}$:
  - U_p (physician decides treatment)
  - U_h ($=1$ healthy diet, $=0$ o/w)
  - U_i for tight insulin control

$$\mathcal{F} = \begin{cases} X \leftarrow U_p \\ F \leftarrow X \vee U_h \\ Y \leftarrow (X \wedge F) \vee U_i \end{cases}$$



2. SCM → Causal Diagram

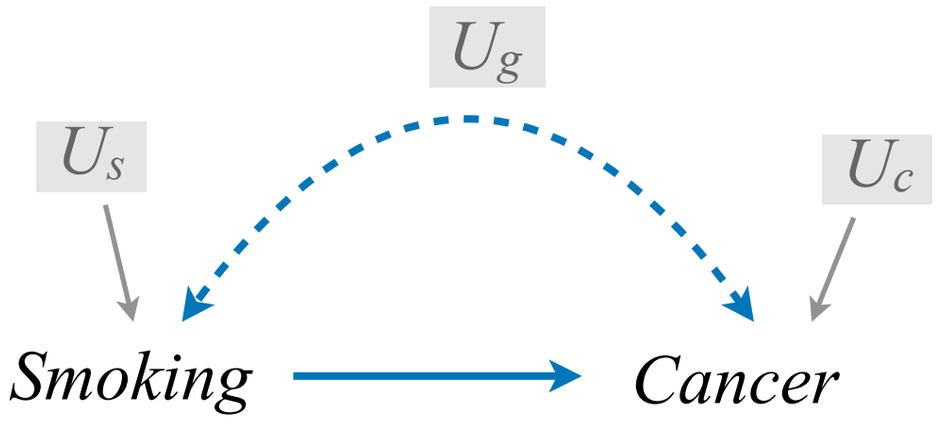
Another example:

- $\boldsymbol{V} = \{ Smoking, Cancer \}$
- $\boldsymbol{U} = \{ U_s, U_c, U_g \}$
- $\mathcal{F}$:

  $Smoking \leftarrow \mathrm{f}_{\mathrm{Smoking}}(U_s, U_g)$

  $Cancer \leftarrow \mathrm{f}_{\mathrm{Cancer}}(Smoking, U_c, U_g)$

unobserved genotype



Remark 1. The mapping from the SCM to a causal graph is just 1-way (i.e., non-injective) since the graph itself is compatible with infinitely many SCMs with the same scope (the same functional signatures and exogenous distributions).

Remark 2. This observation will be central to causal inference since, in most practical settings, researchers may know the scope of the functions, for example, but not the details about the underlying mechanisms.

Causal Diagrams

- Convention. The unobserved variables are left implicit in the graph.

Food for thought

Does the causal diagram give us any clues about the (in)dependence relations in the obs. distribution P(V)?

- Is T independent of W?
- Is W independent of T?
- Is Z independent of T?
- Is Z independent of X?
- Is Y independent of W?
- Is Y independent of W if we know the value of X?

$$M = \begin{cases} Z \leftarrow f_Z(u_z) \\ X \leftarrow f_x(u_x) \\ W \leftarrow f_W(z, x, u_w) \\ Y \leftarrow f_Y(x, u_y) \\ T \leftarrow f_T(w, u_t) \end{cases}$$

Property 1

Prop. 2