

A Crash Course in Good and Bad Controls

Carlos Cinelli (UW), Andrew Forney (LMU), and Judea Pearl (UCLA)

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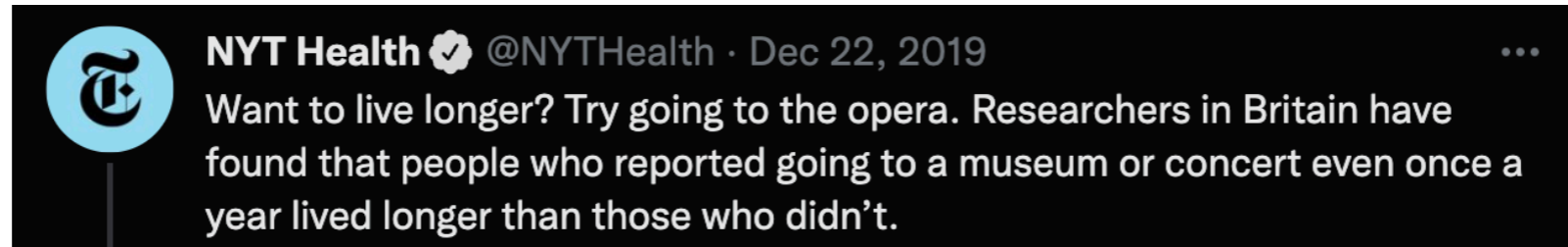
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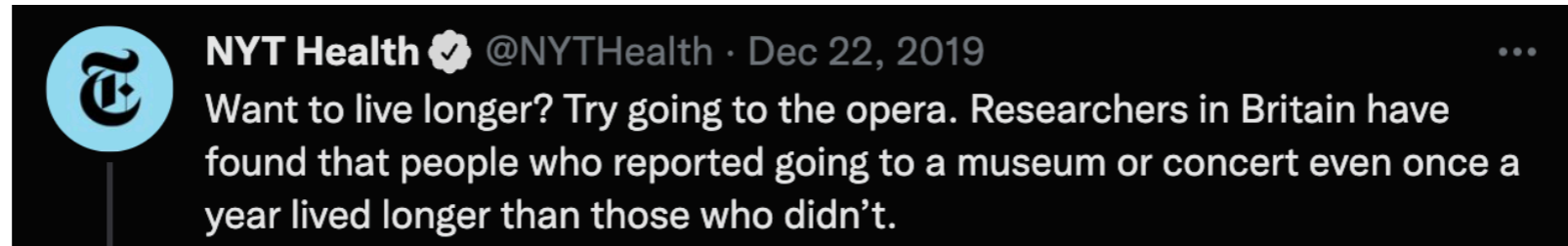
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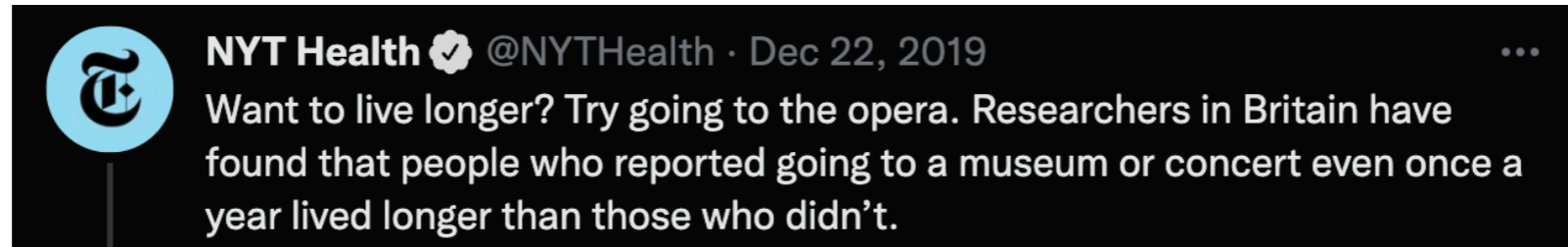
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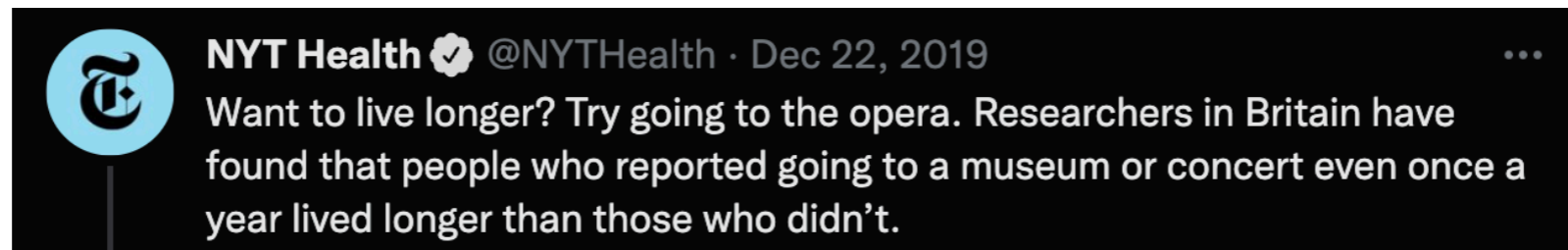


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The study **controlled for** socioeconomic factors like a participant’s income, education level and mobility, said Andrew Steptoe, a co-author of the study and the head of University College London’s research department of behavioral science and health.

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What now?

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We will see how causal diagrams can make otherwise difficult problems very easy to solve, *by – literally – simple inspection of a diagram.*

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Birth-weight paradox: Infants born to smokers were found to have higher risks of mortality than infants born to non-smokers. However, among infants with low birth-weight (LBW), this relationship was reversed. This reversal of effects has created many controversies in epidemiology—does it mean that maternal smoking is beneficial for LBW infants?

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These are all related to “bad controls.”

Preliminaries – Causal Diagrams

Causal Diagrams

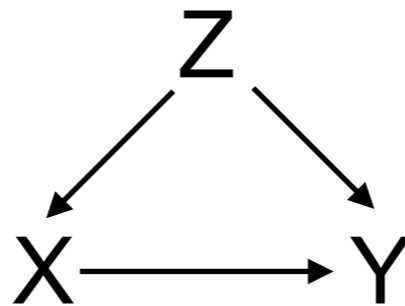
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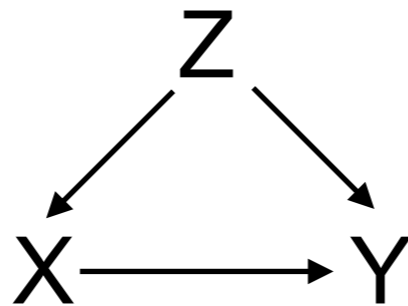
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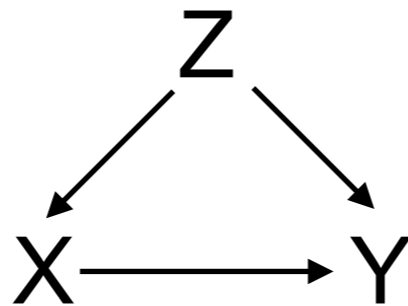


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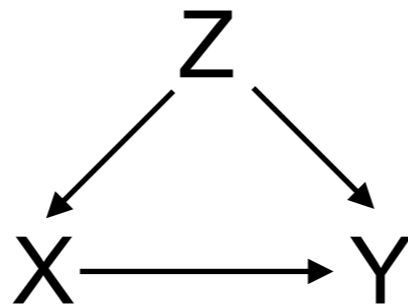


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2. Arrows denote a (possible) direct causal effect between one variable on another. For instance, the arrows $X \rightarrow Y$ and $Z \rightarrow Y$ state that both the drug and income could possibly affect health.

Note that no parametric assumptions need to be made regarding the functional form of the causal relationships, nor the distribution of variables.

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Let us understand better each of these forms of association, and when they are closed or opened.

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1. Mediators: *X causally* affects *Y* through *Z*. This is a causal path from *X* to *Y*.
If left untouched, the path is open.

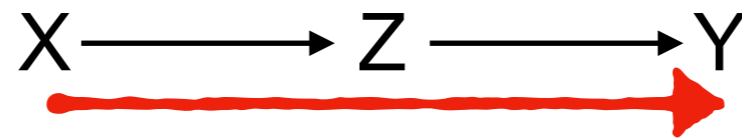
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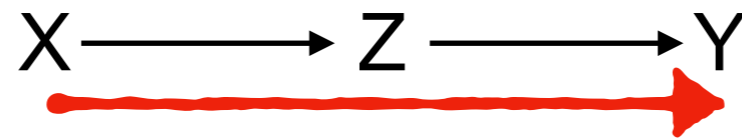
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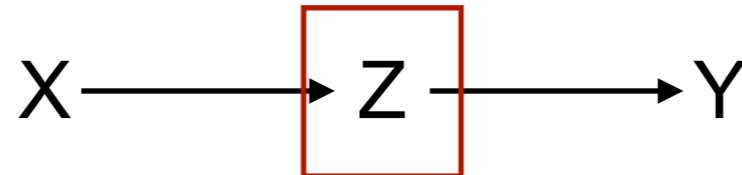


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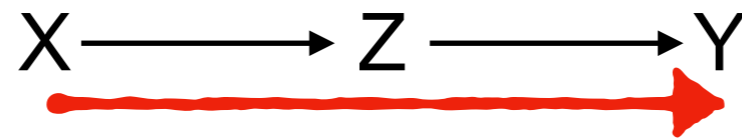


Conditioning on the mediator Z blocks the flow of association.

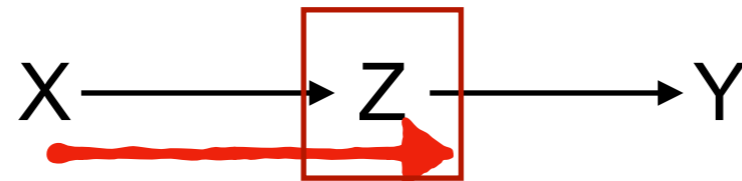


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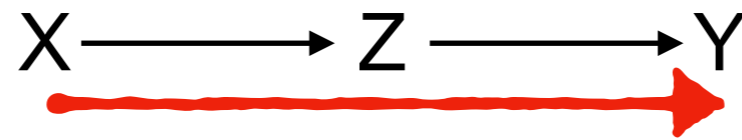


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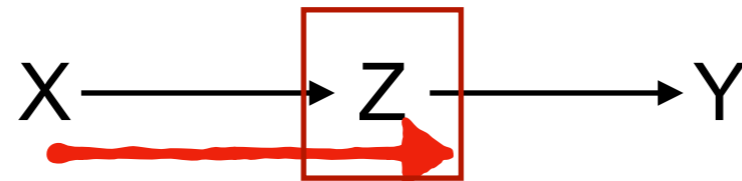


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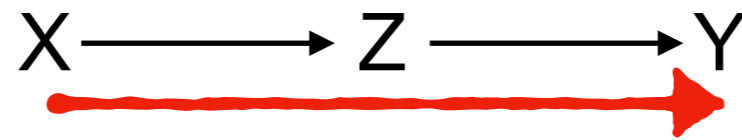
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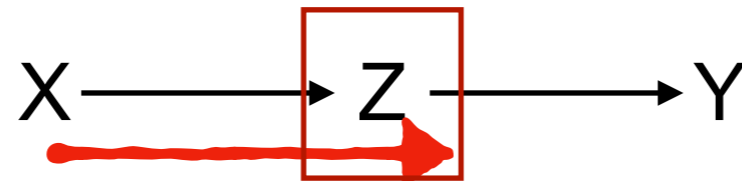
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Example: Consider a drug (X) that affects a health outcome (Y) by lowering blood pressure (Z).

Conditioning on blood pressure blocks the mechanism through which the drug affects health. Thus you will not see any association between drug use and health status among those with the same level of blood pressure.

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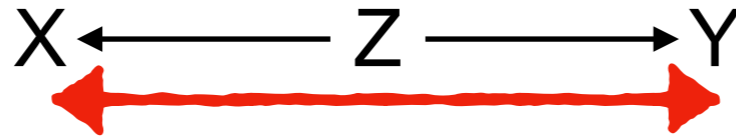
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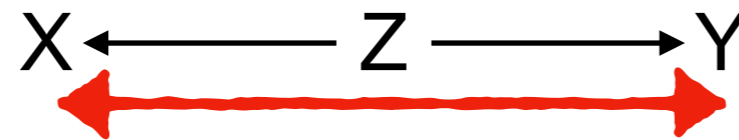
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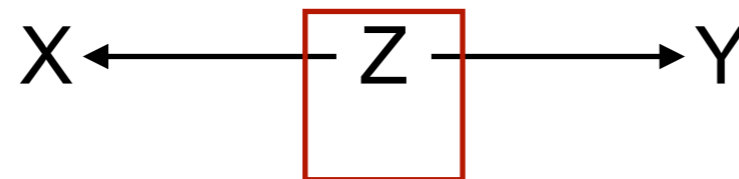


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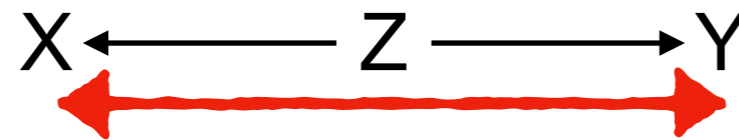


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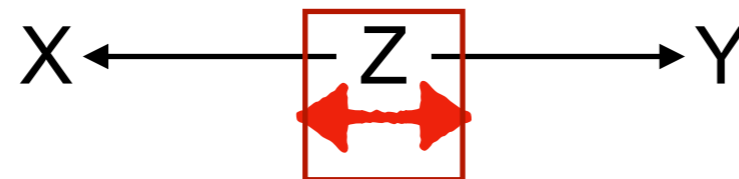


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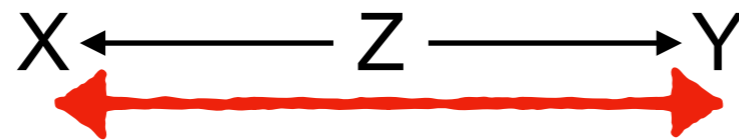


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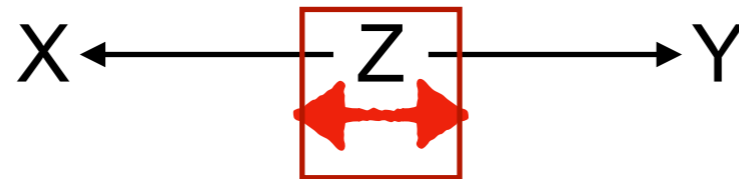


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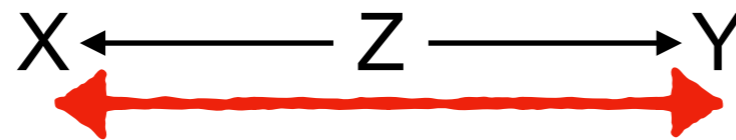
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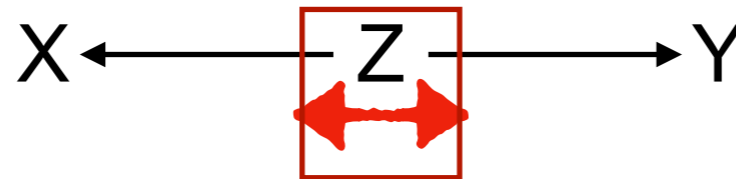
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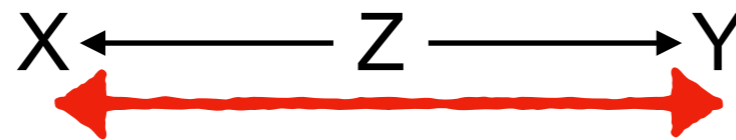


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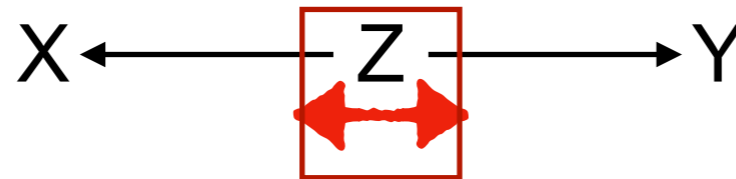
However, rich people (*Z* = income) are both more likely to go to the opera and also more likely to be healthy. This will induce a spurious association between *X* and *Y*.

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Conditioning on income (Z) blocks this spurious association.

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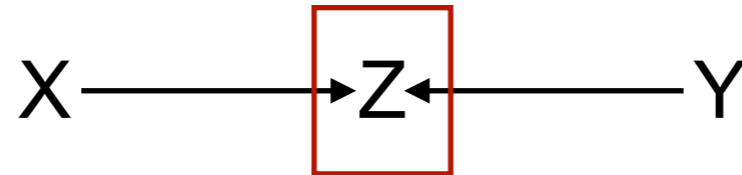


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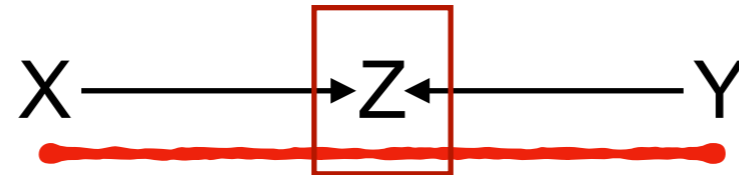


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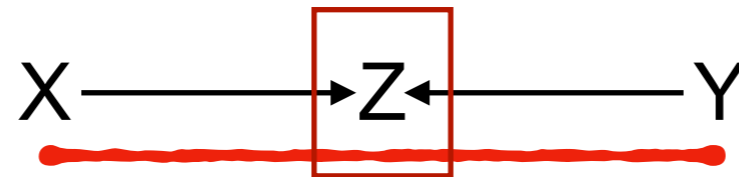


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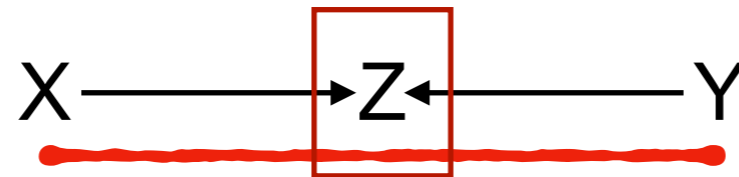
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Example: Imagine beauty (X) and talent (Y) are independent in the general population. However, suppose that movie agencies only hire ($Z=1$) actors whose beauty + talent exceed a certain threshold.

Even though there's no causal relationship between beauty and talent in the general population, you will see a *negative* association between beauty and talent *both among hired actors ($Z=1$) and not hired actors ($Z=0$).*

Building blocks of a causal diagram

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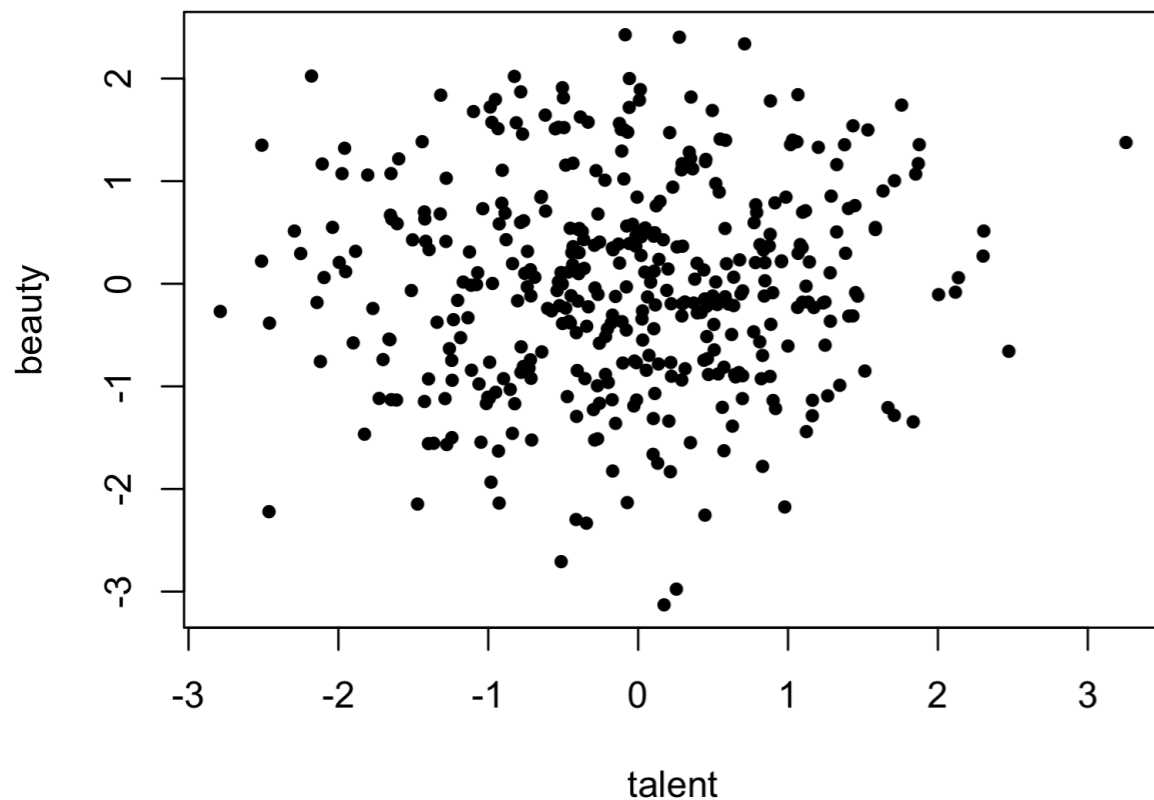
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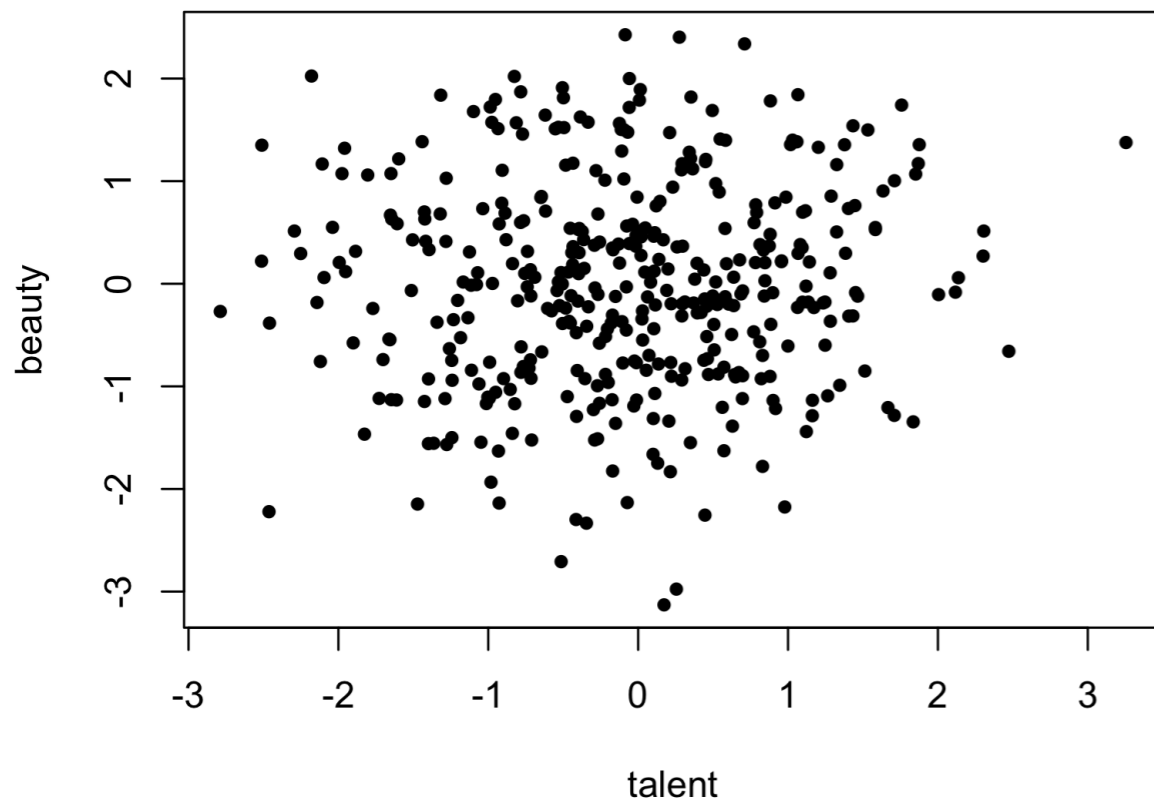
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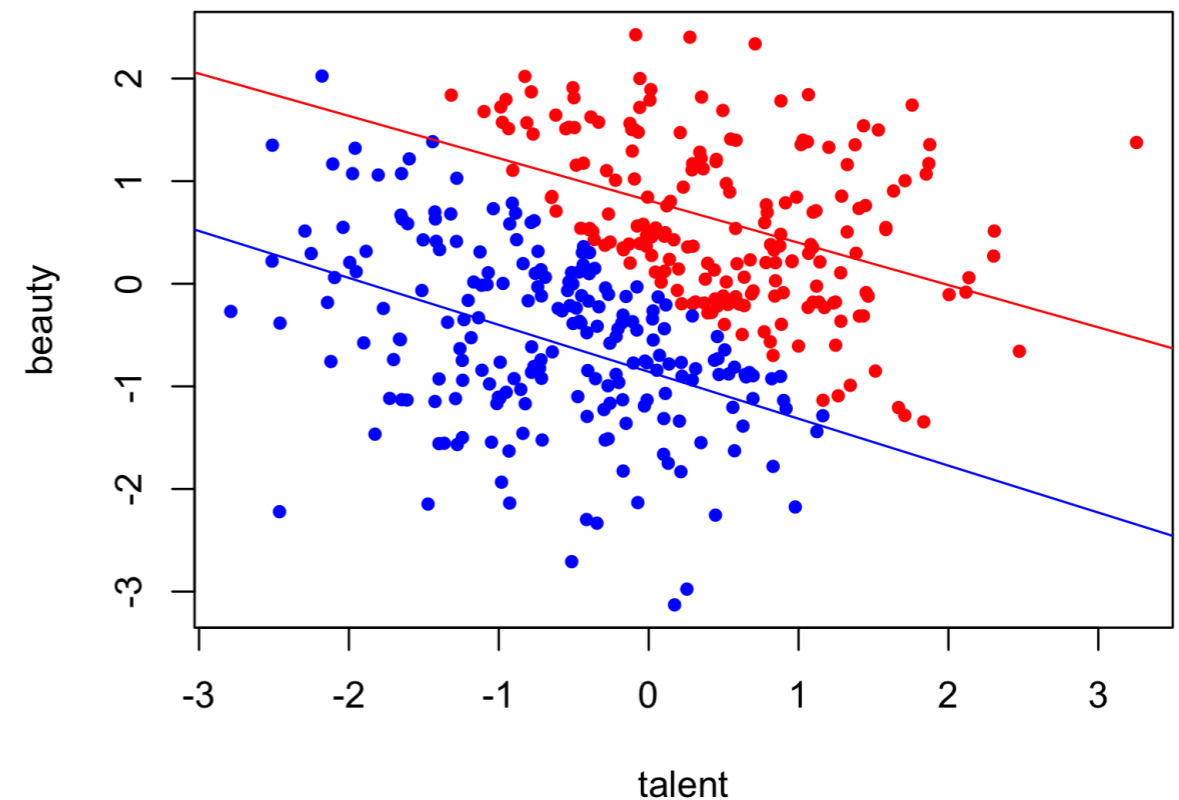
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Within Z=1 or Z=0
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Building blocks of a causal diagram

To summarize:

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- (i) conditioning on the effects of a mediator partially closes a causal path;
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Opening and closing paths.

We can now judge whether any path on a causal diagram, no matter how complicated, is opened or closed.

Opening and closing paths.

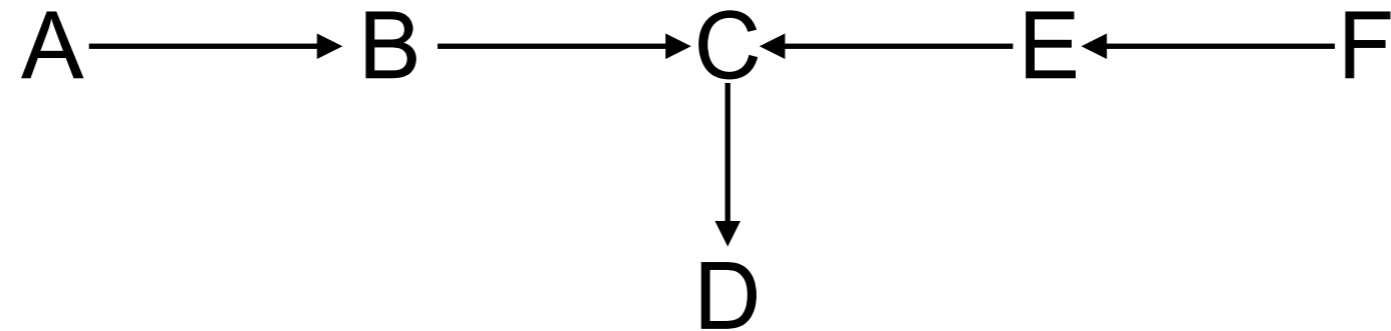
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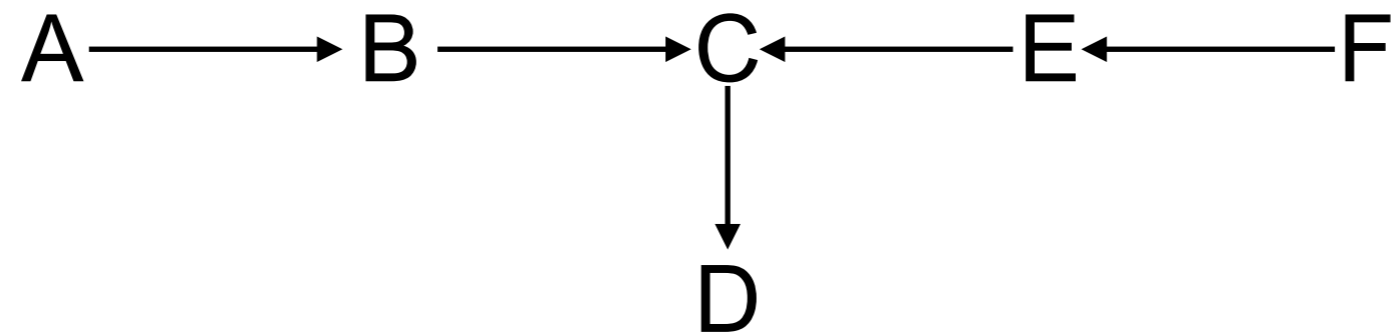
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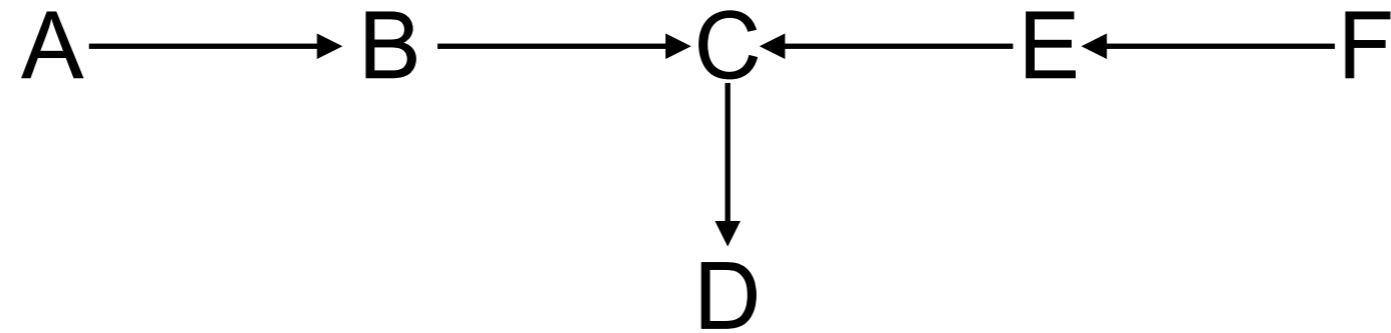


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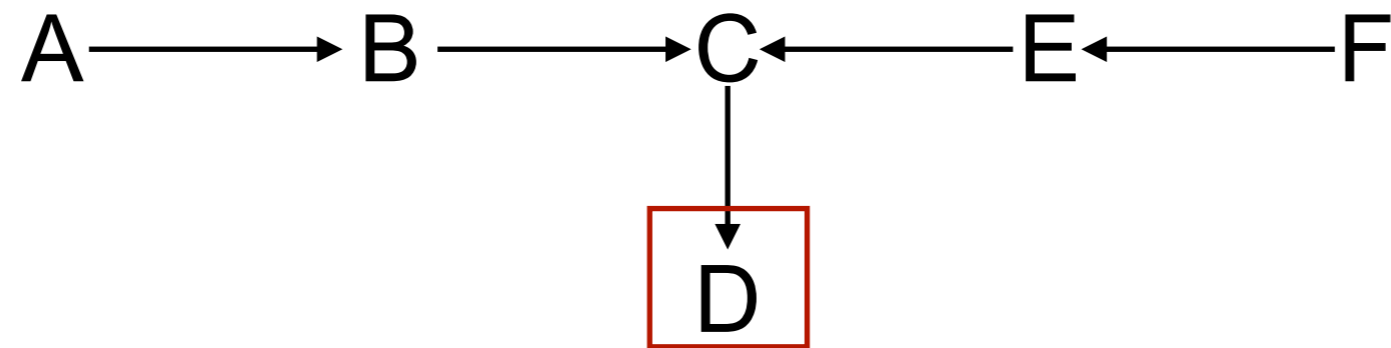
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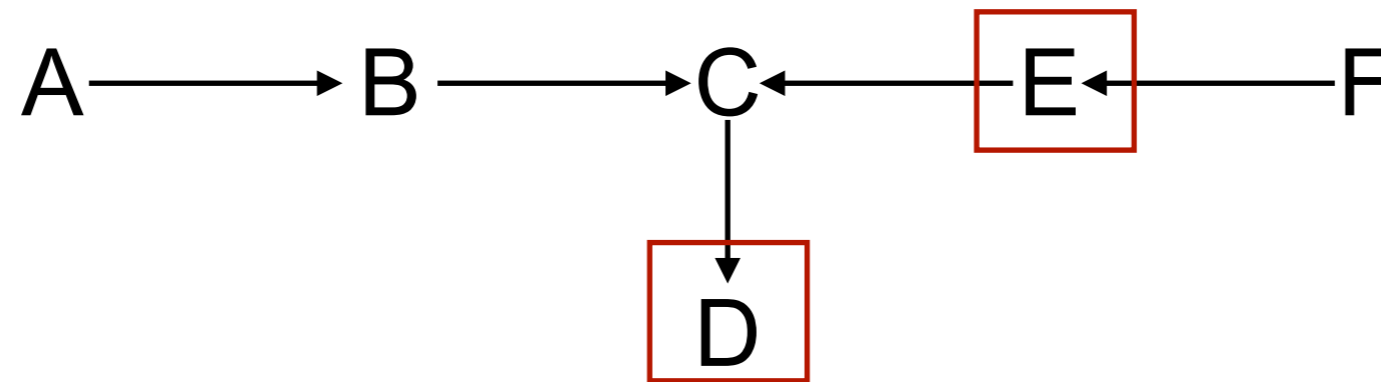
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Although simple, mastering this does require some practice. So let's apply those principles in very simple examples.

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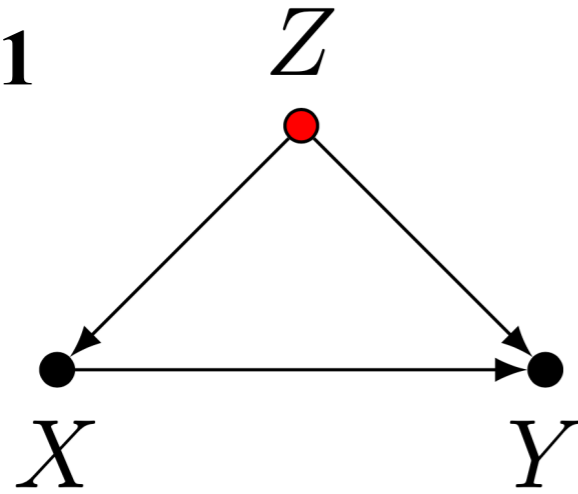
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Here will focus on *practicing our graphical skills*. Later we will see how these very simple models can help you make sense of real world scenarios.

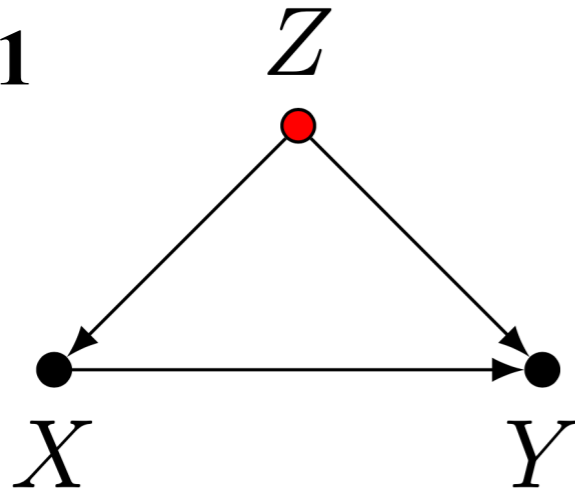
“Good” Controls – Blocking backdoor paths

Model 1



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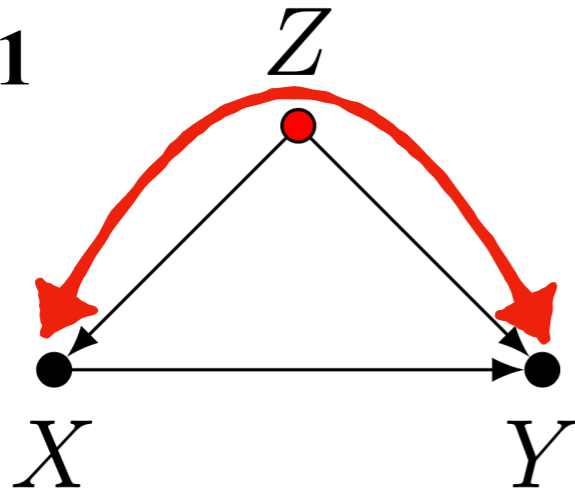
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Z is a confounder, and creates a spurious association between X and Y.

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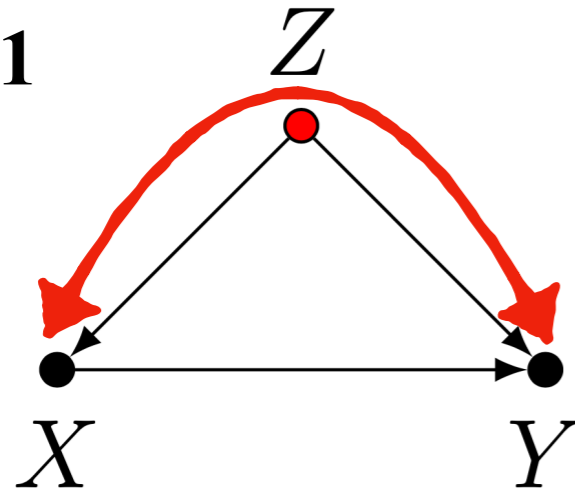
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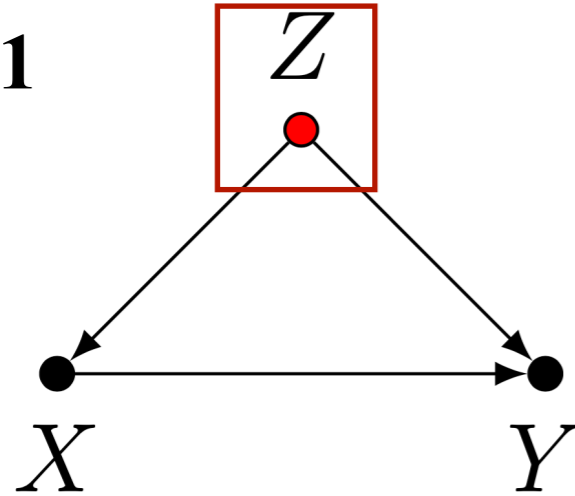


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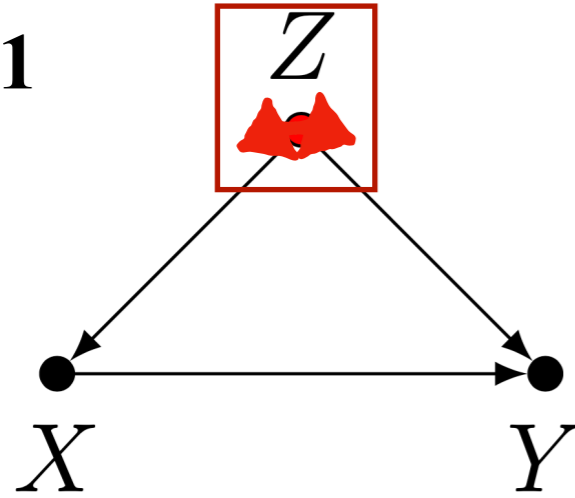


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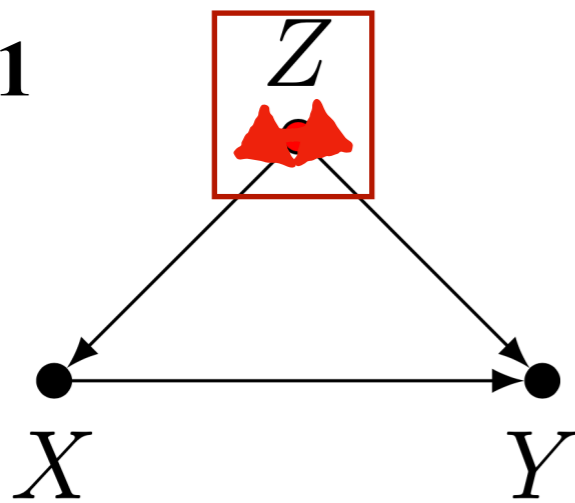


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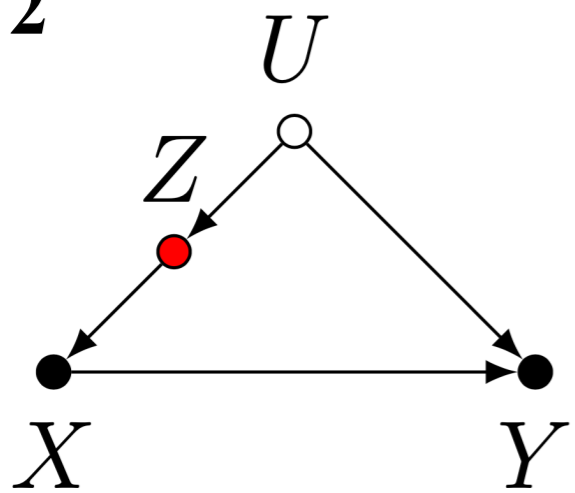
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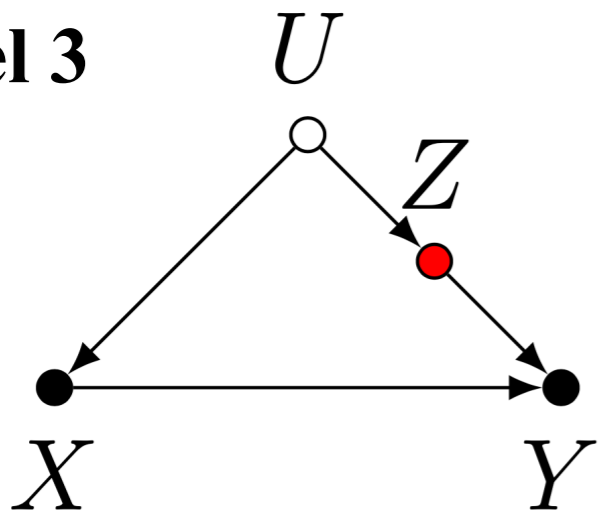
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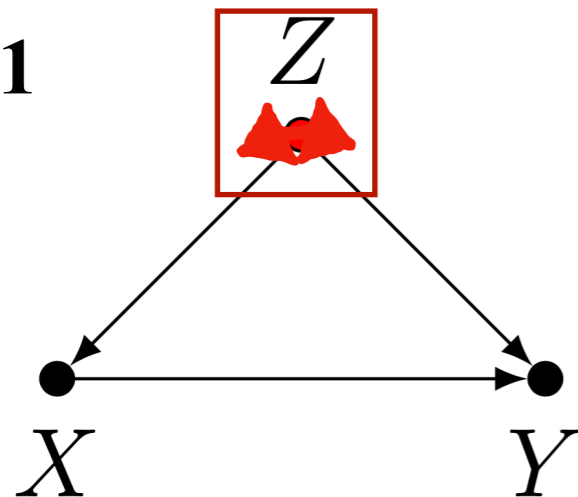


Model 3



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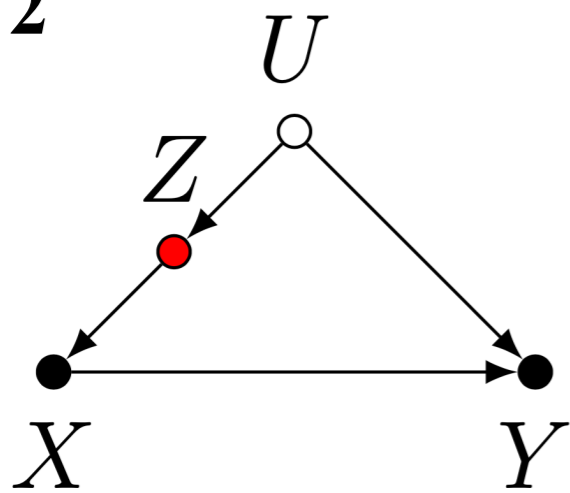
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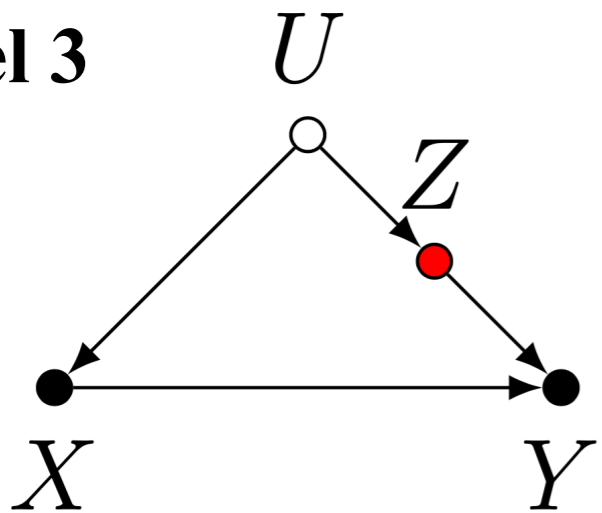
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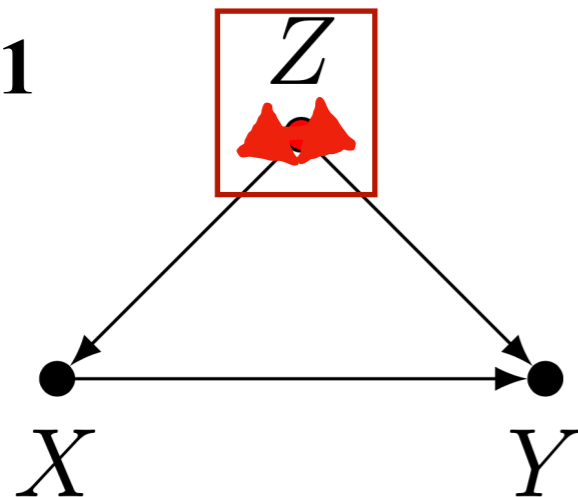
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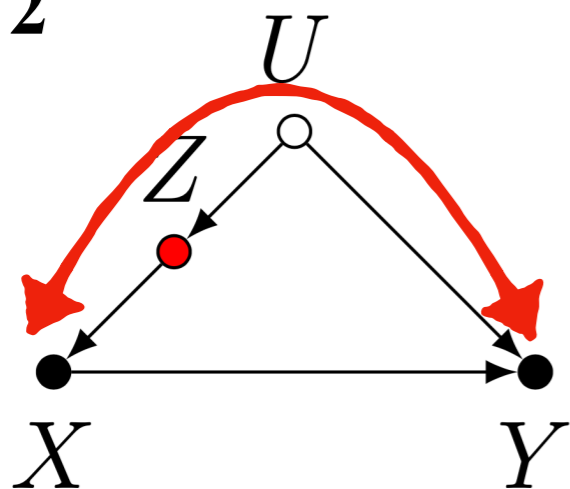
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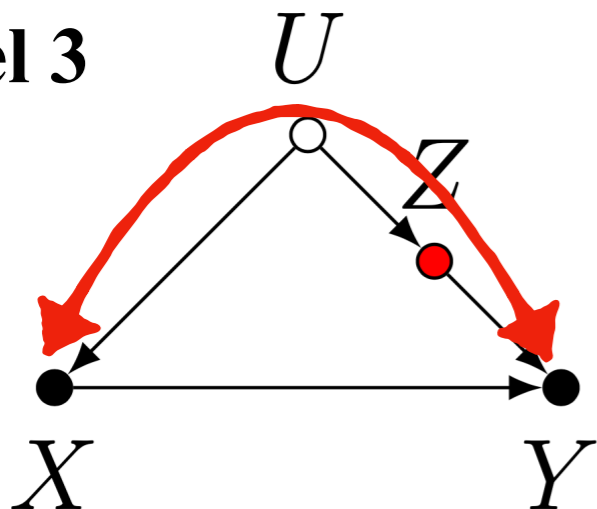
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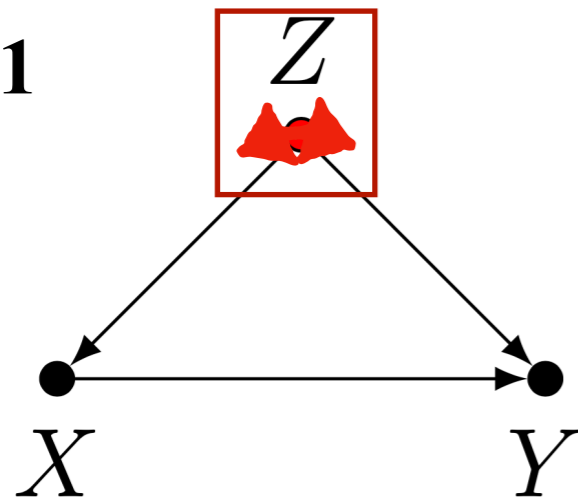
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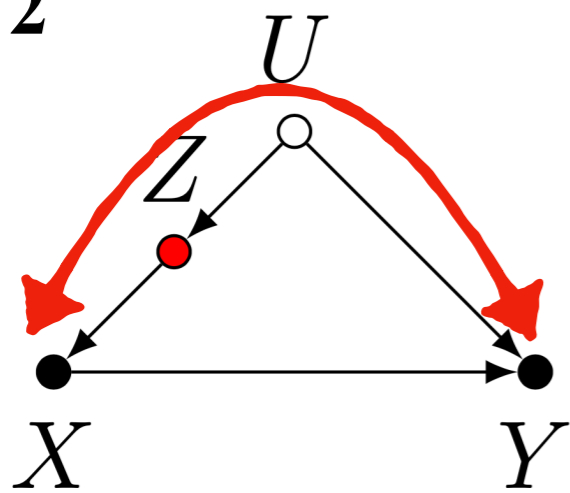
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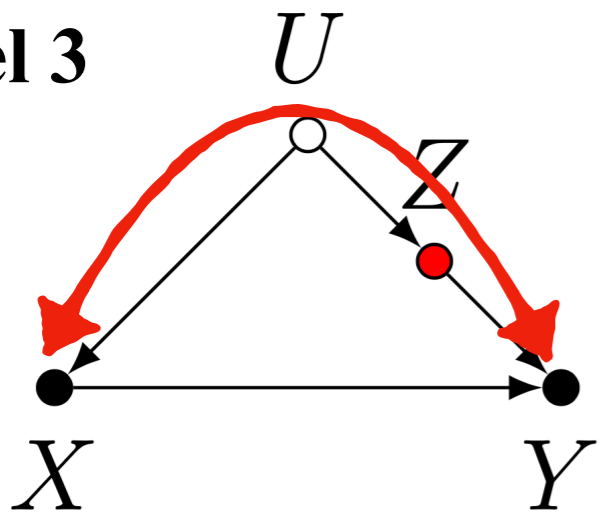
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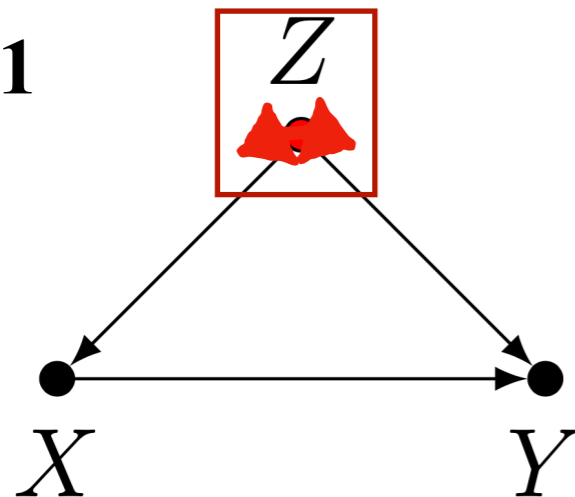
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Note that Z is not a common cause of X and Y. Thus Z is not a traditional “confounder” as before.

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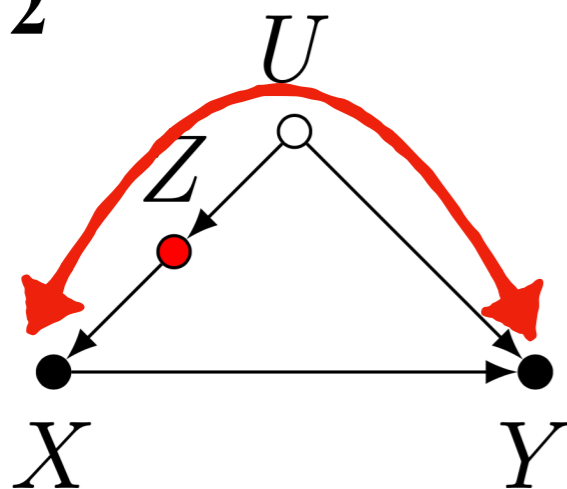
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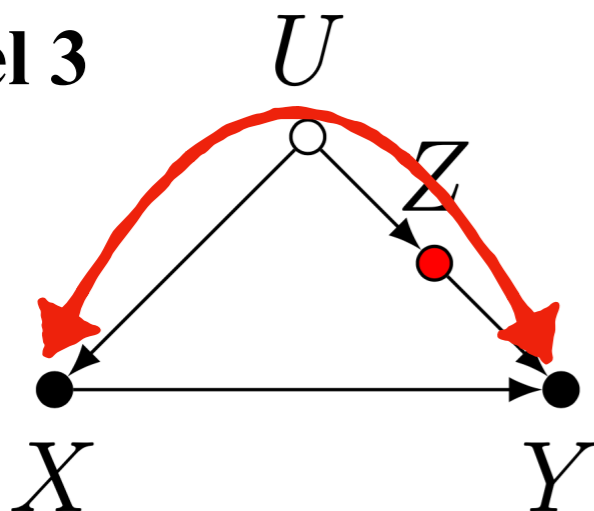
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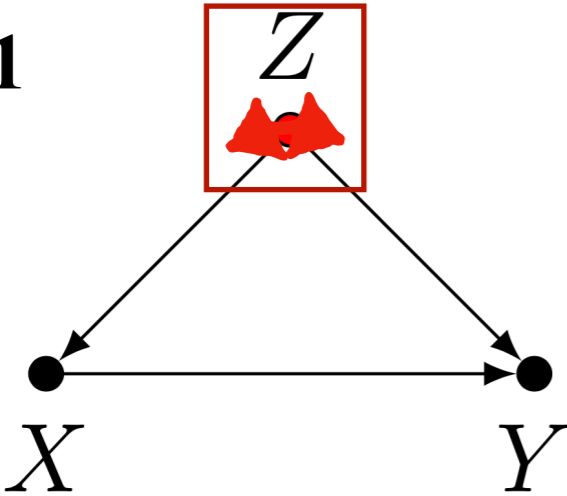


Note that Z is not a common cause of X and Y. Thus Z is not a traditional “confounder” as before.

However controlling for Z does block the backdoor path due to U, and again, we obtain an unbiased estimate of the ATE.

“Good” Controls – Blocking backdoor paths

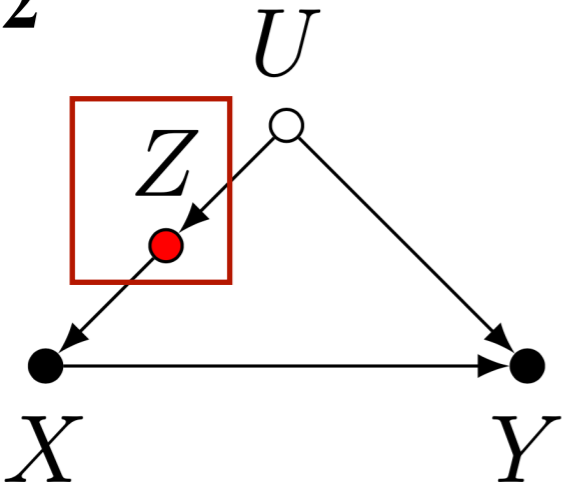
Model 1



Z is a confounder, and creates a spurious association between X and Y.

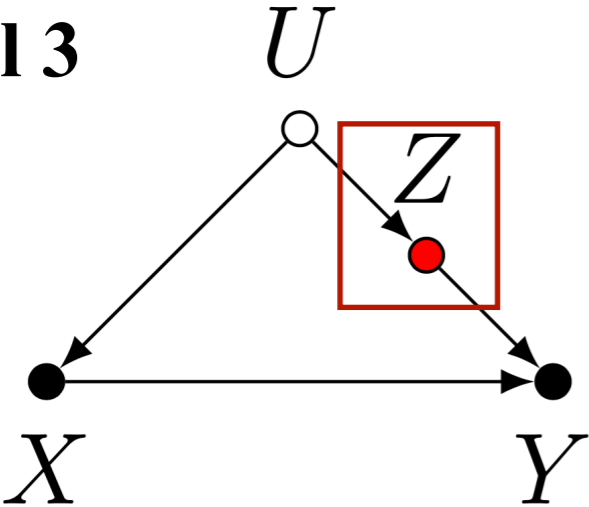
Once we control for Z we block the backdoor path, producing an unbiased estimate of the ATE.

Model 2



In Models 2 and 3 we have a backdoor path due to the unobserved confounder U.

Model 3

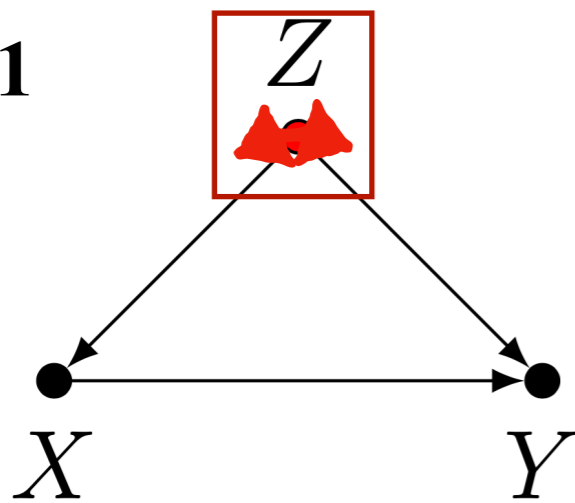


Note that Z is not a common cause of X and Y. Thus Z is not a traditional “confounder” as before.

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“Good” Controls – Blocking backdoor paths

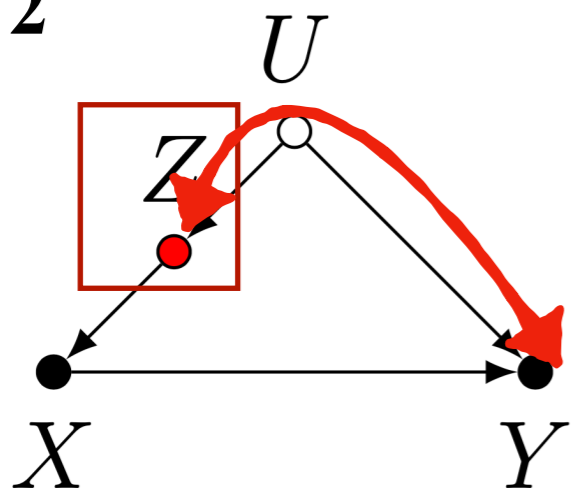
Model 1



Z is a confounder, and creates a spurious association between X and Y.

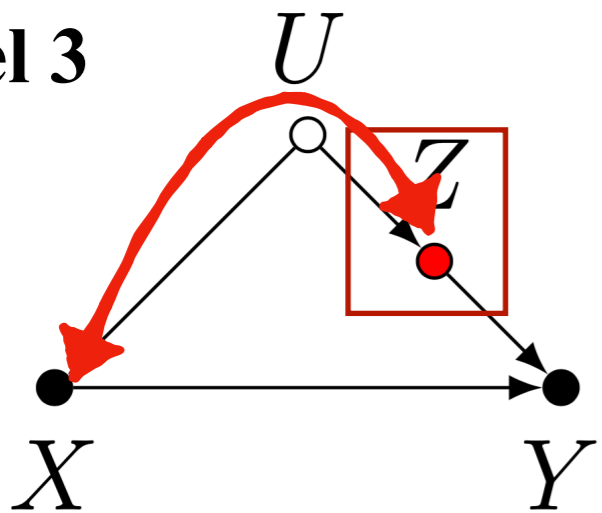
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Model 2



In Models 2 and 3 we have a backdoor path due to the unobserved confounder U.

Model 3



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“Good” Controls – Blocking backdoor paths

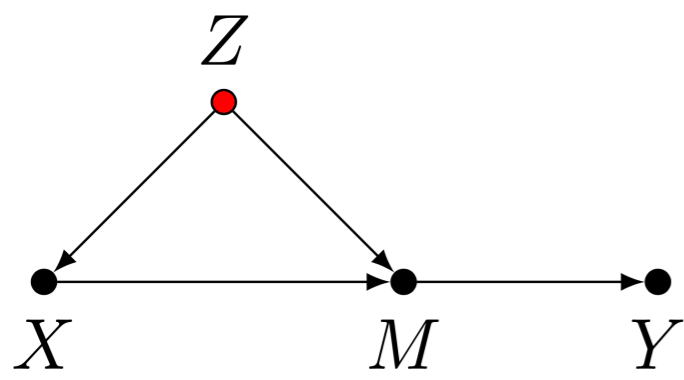
Modelers need to keep in mind that common causes of X and any mediator (between X and Y) also confound the effect of X and Y .

For instance consider models 4, 5 and 6 below:

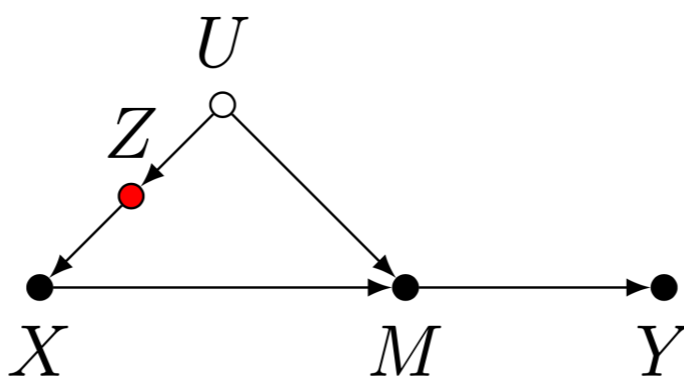
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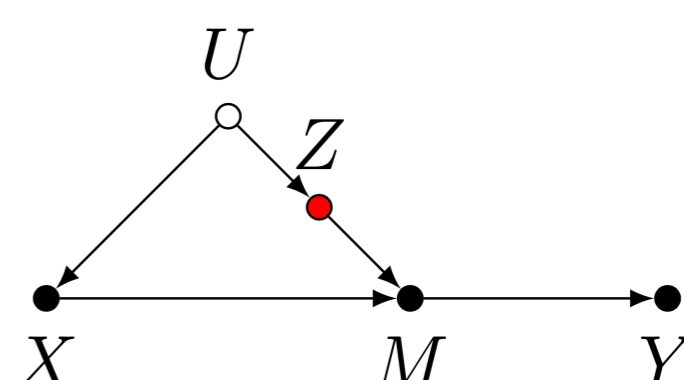
For instance consider models 4, 5 and 6 below:



Model 4



Model 5

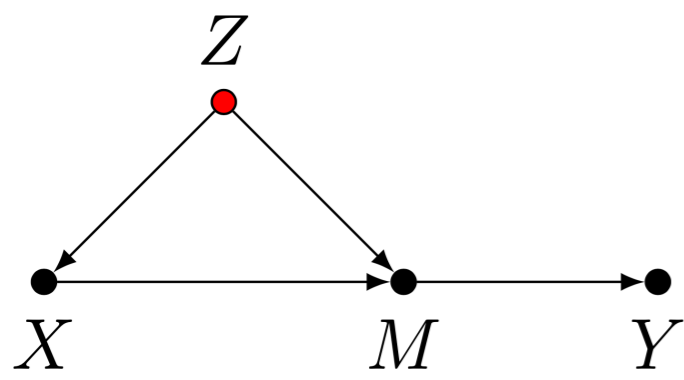


Model 6

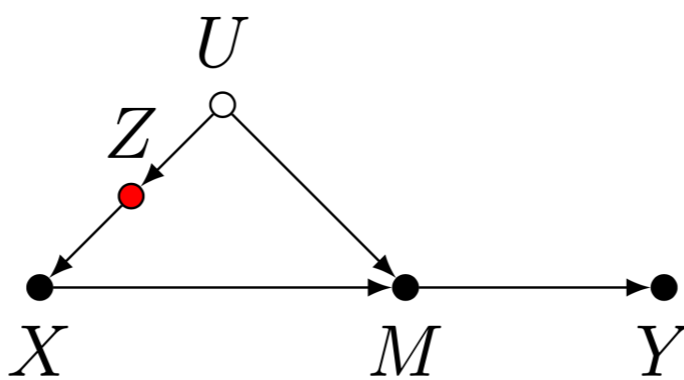
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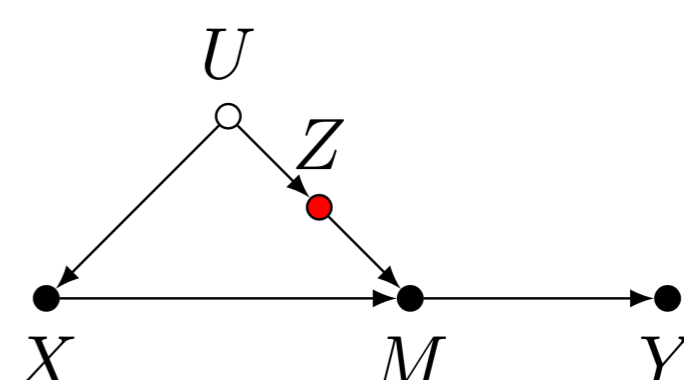
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Model 4



Model 5



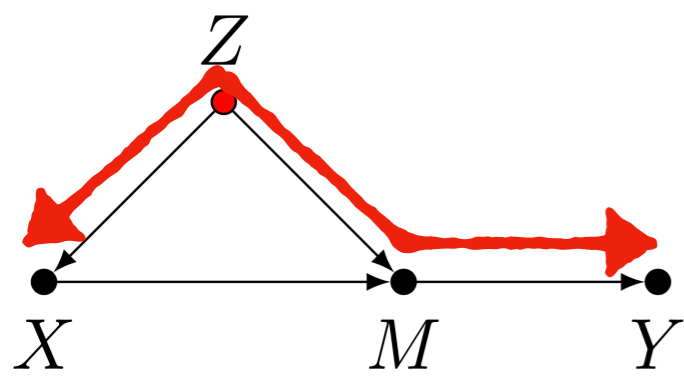
Model 6

Note these are analogous to Models 1, 2 and 3, and there is an open backdoor path from X to Y.

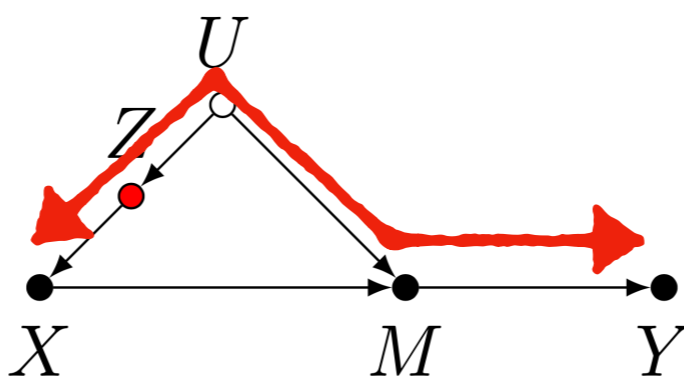
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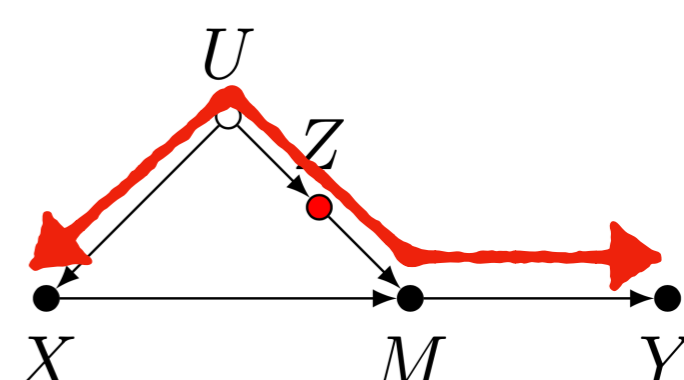
For instance consider models 4, 5 and 6 below:



Model 4



Model 5



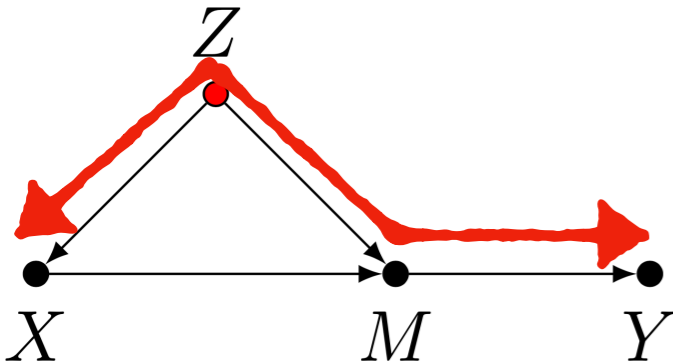
Model 6

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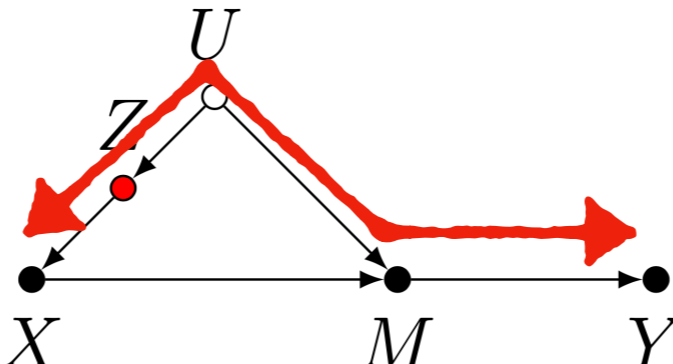
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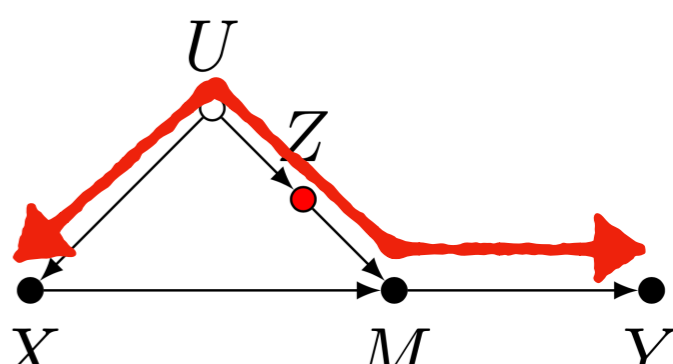
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Model 4



Model 5



Model 6

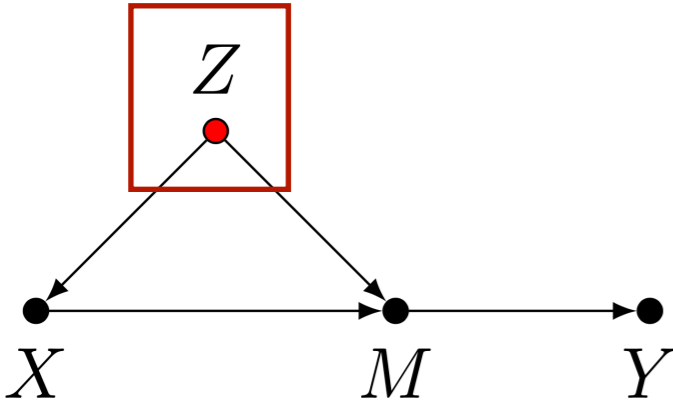
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Here, as before, controlling for Z blocks the backdoor path from X to Y, and produces an unbiased estimate of the ATE.

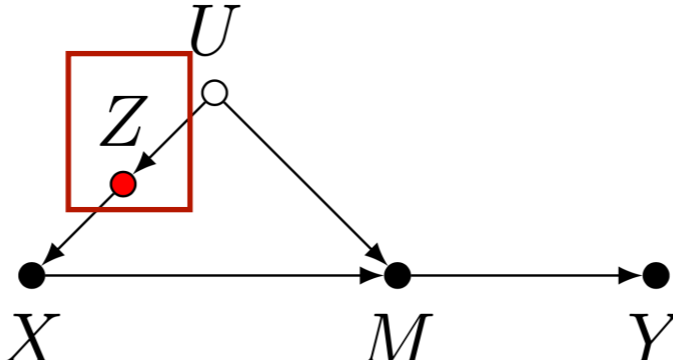
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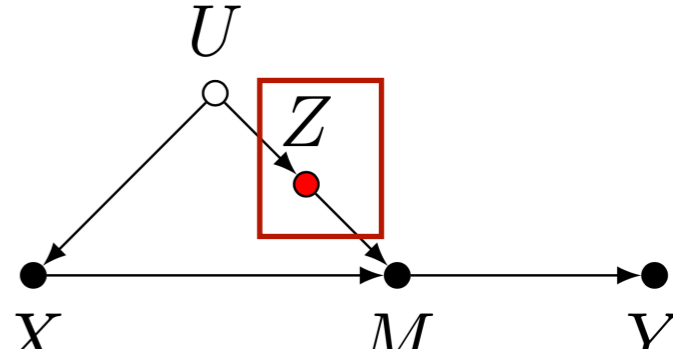
For instance consider models 4, 5 and 6 below:



Model 4



Model 5



Model 6

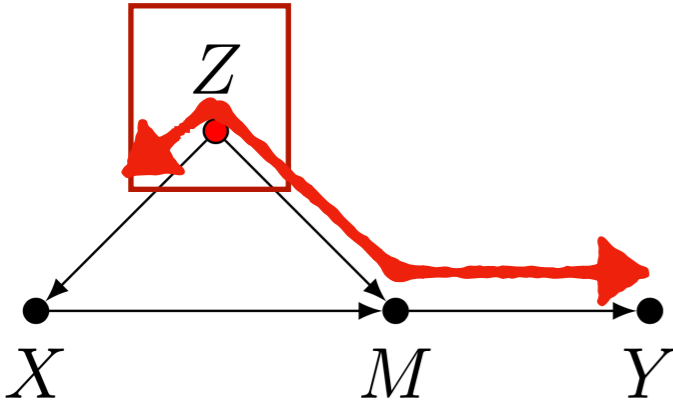
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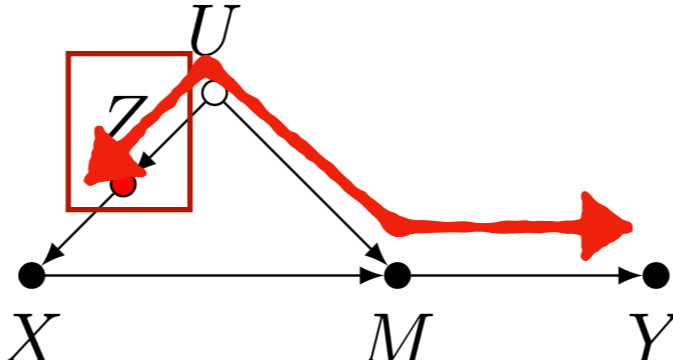
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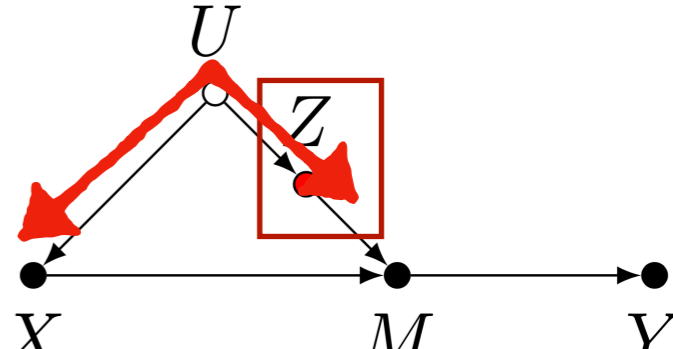
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Model 4



Model 5



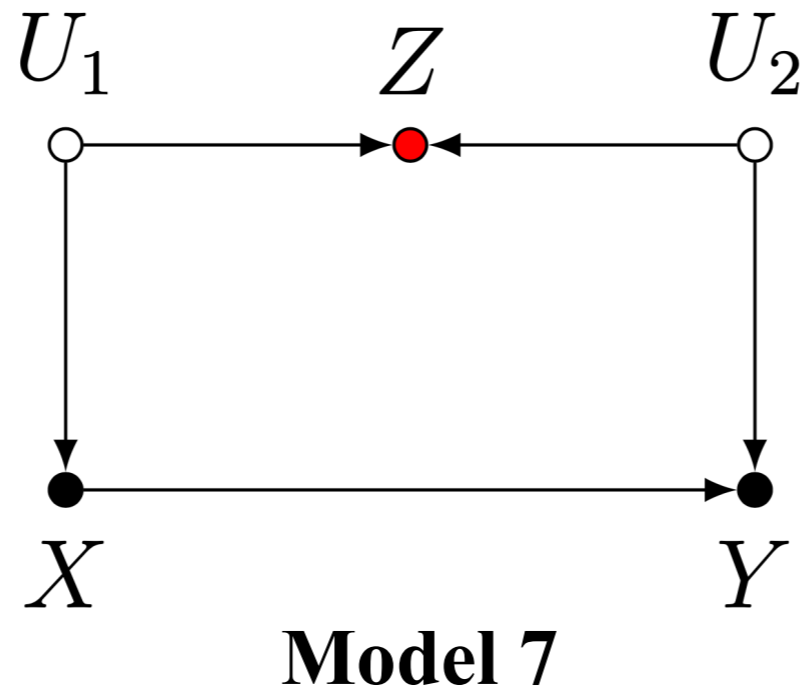
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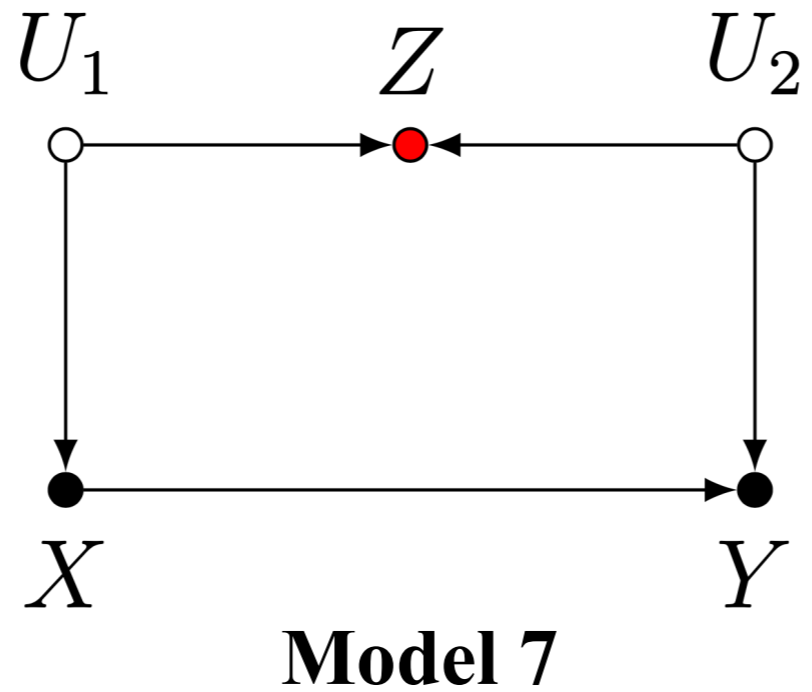
“Bad” Control – M-Bias

We now encounter our first bad control.



“Bad” Control – M-Bias

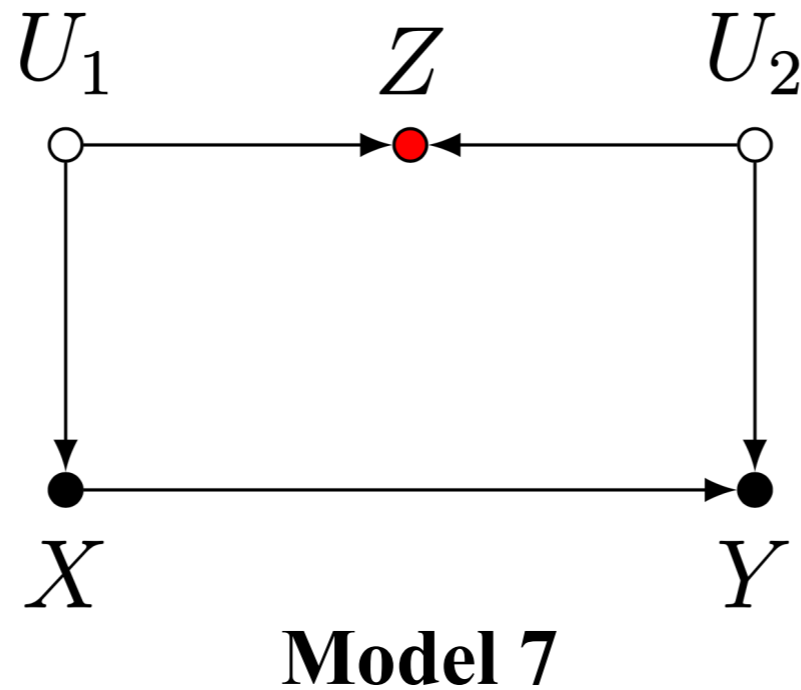
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Note that: (i) Z is correlated with the treatment X ; (ii) Z is correlated with the outcome Y ; (iii) Z is “pre-treatment.” Traditional analysis would thus consider Z a confounder, and perhaps deem Z a good control.

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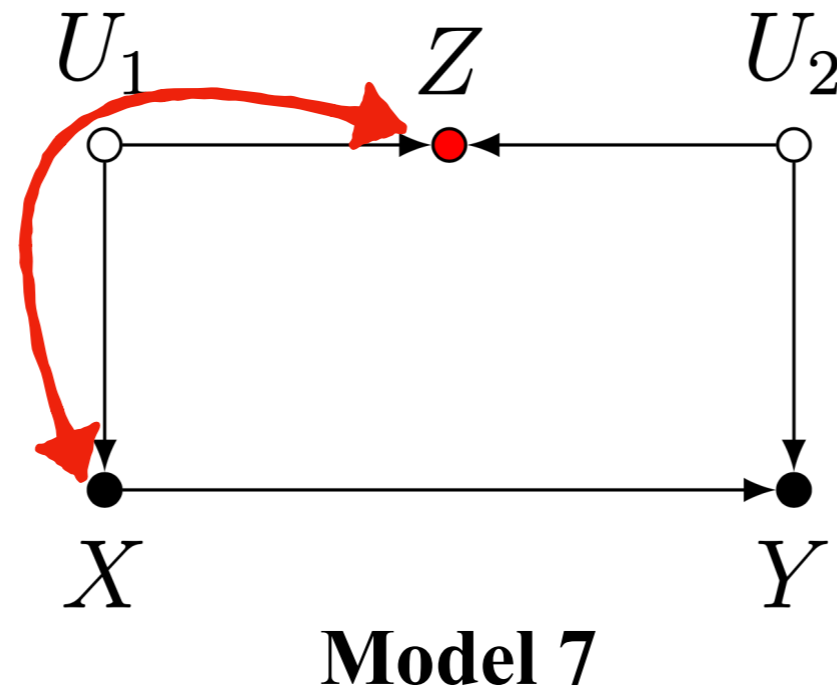


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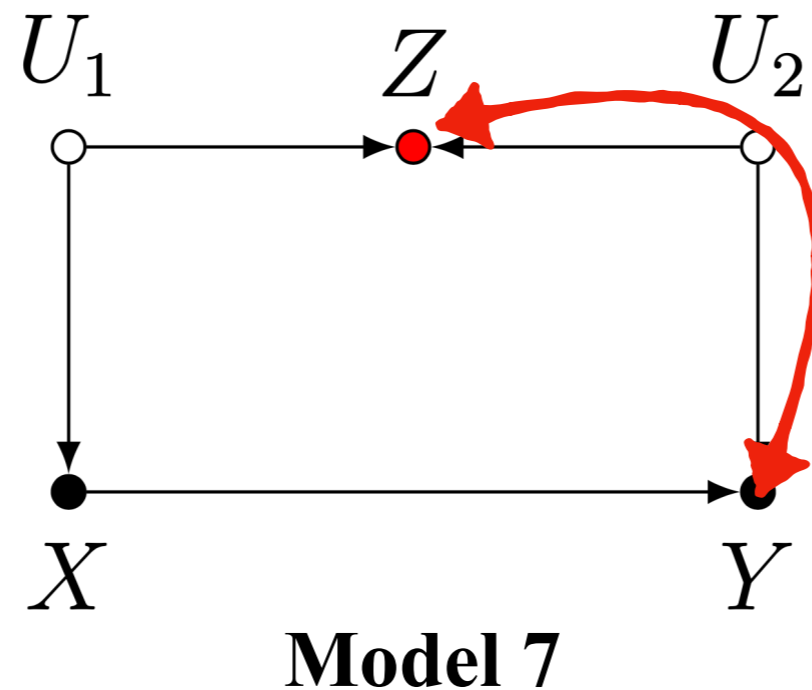


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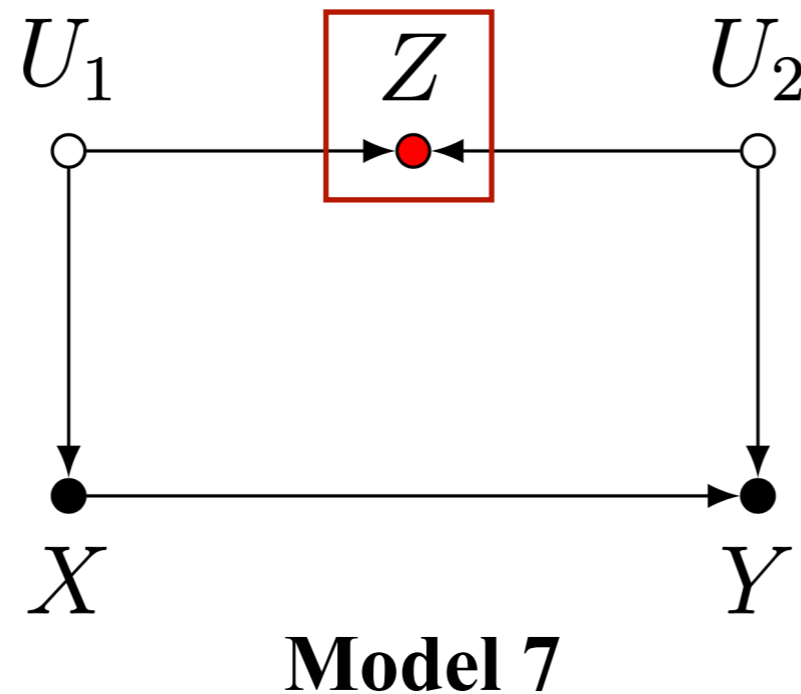


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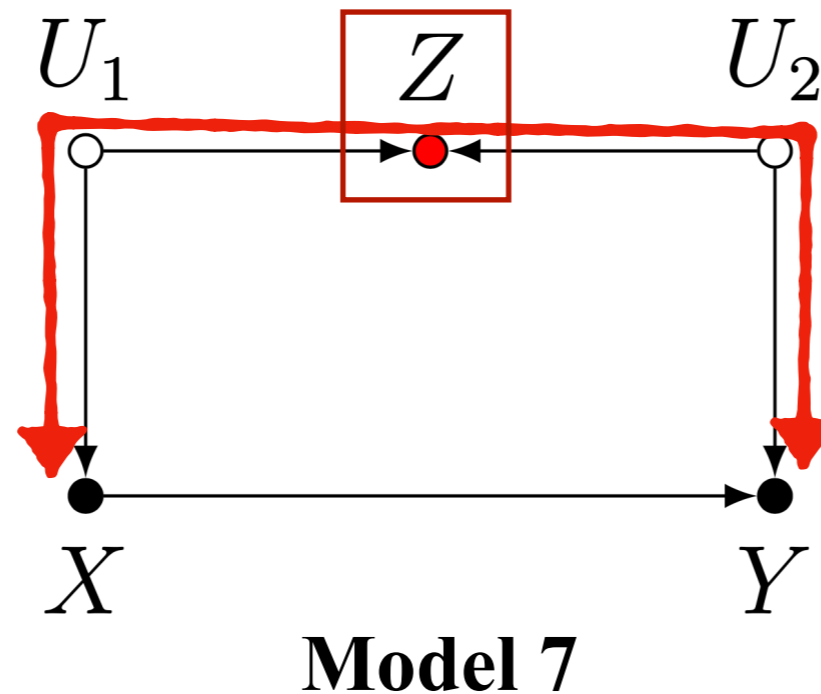
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Conditioning on Z , however, *opens* the path $X \leftarrow U_1 \rightarrow Z \leftarrow U_2 \rightarrow Y$, and thus spoils a previously unbiased estimate.

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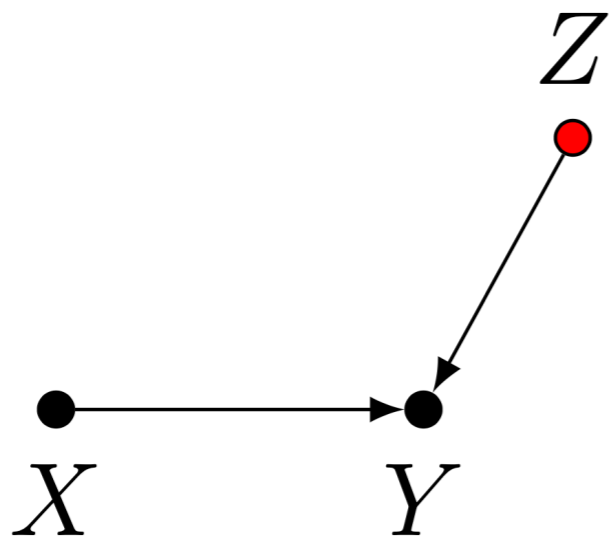


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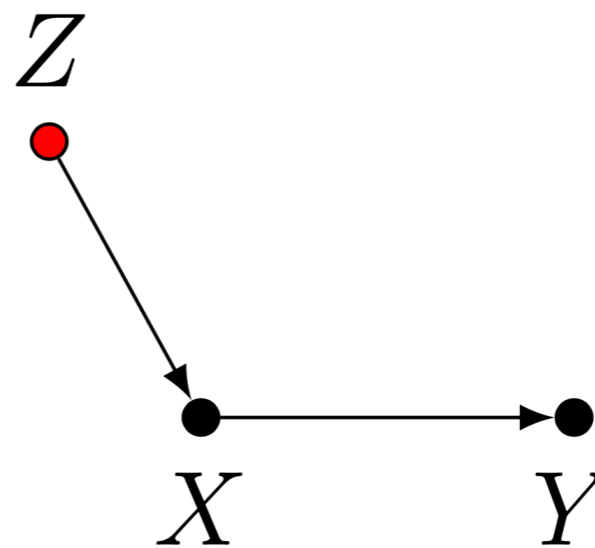
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“Neutral” Controls (but different impacts on precision)

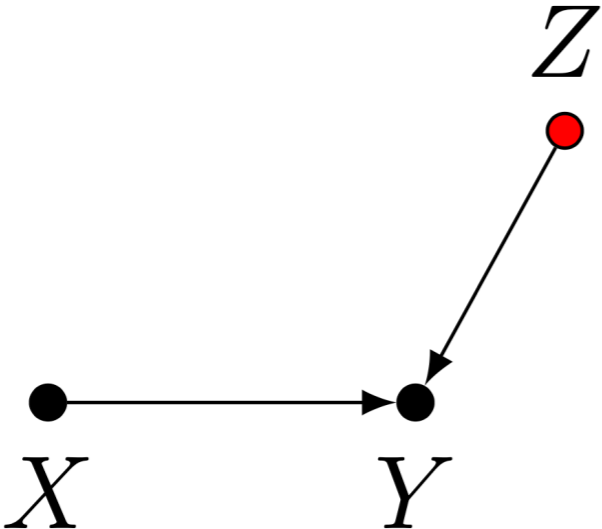


Model 8

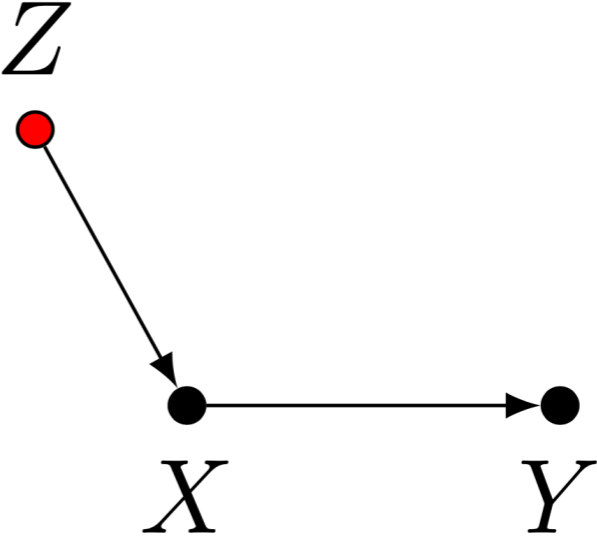


Model 9

“Neutral” Controls (but different impacts on precision)



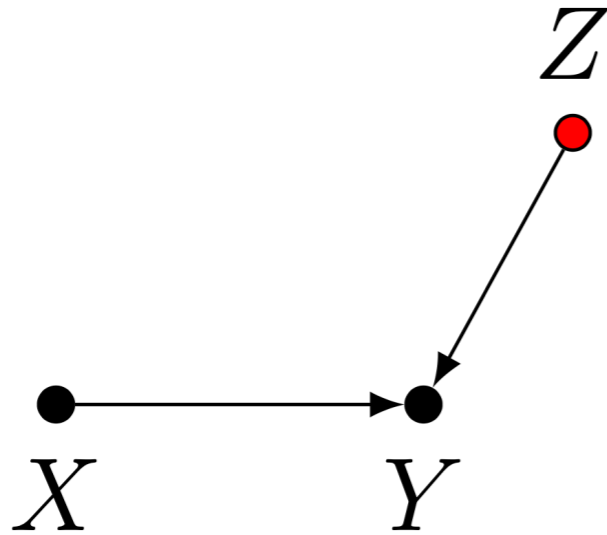
Model 8



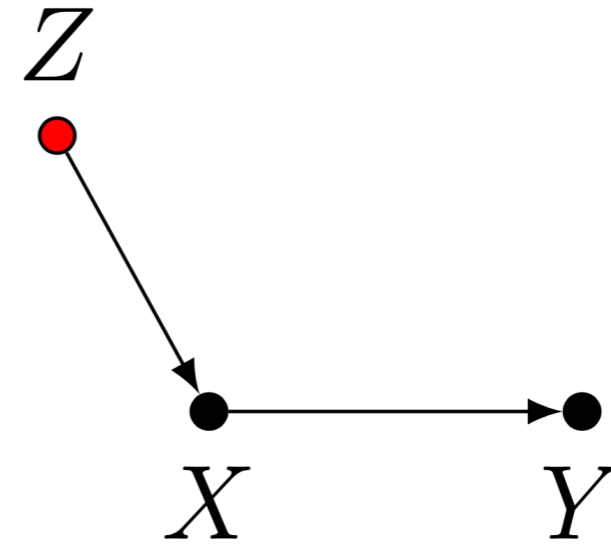
Model 9

In Models 8 and 9, Z is not a confounder, nor does Z block any backdoor paths. Likewise, controlling for Z does not open any spurious paths from X to Y.

“Neutral” Controls (but different impacts on precision)



Model 8

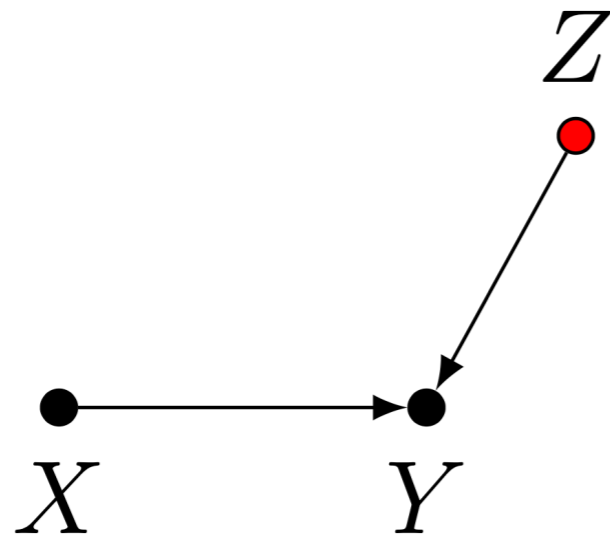


Model 9

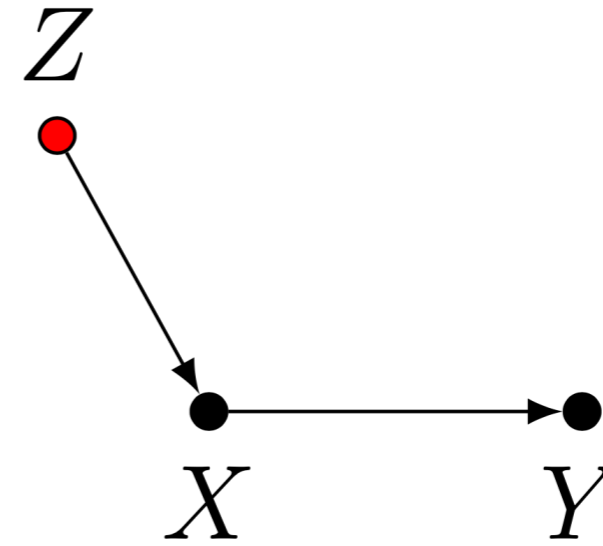
In Models 8 and 9, Z is not a confounder, nor does Z block any backdoor paths. Likewise, controlling for Z does not open any spurious paths from X to Y .

Thus, in terms of asymptotic bias, Z is thus a “neutral control.”

“Neutral” Controls (but different impacts on precision)



Model 8



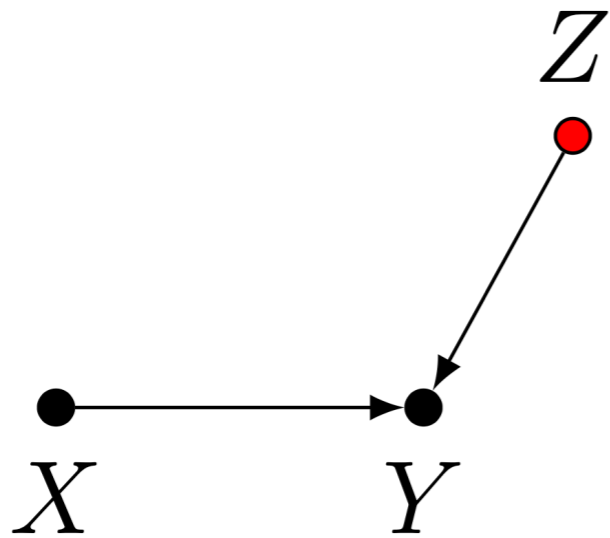
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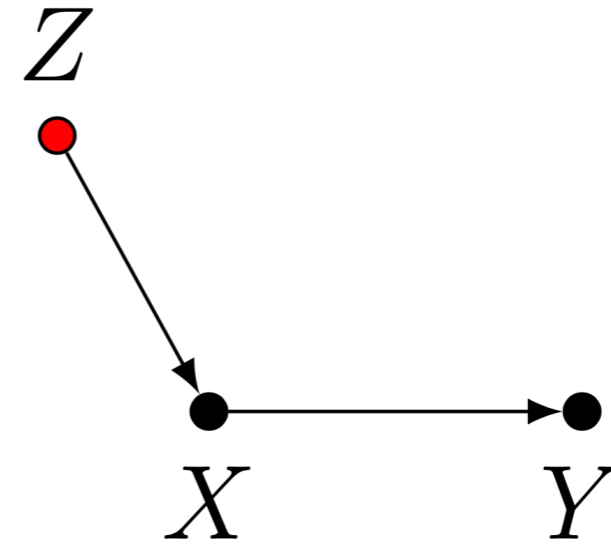
Thus, in terms of asymptotic bias, Z is thus a “neutral control.”

As a general rule-of-thumb, however, in order to obtain more precise estimates of the ATE, we want to reduce the variation of the outcome (due to sources other than the treatment), and not reduce the variation of the treatment itself.

“Neutral” Controls (but different impacts on precision)



Model 8



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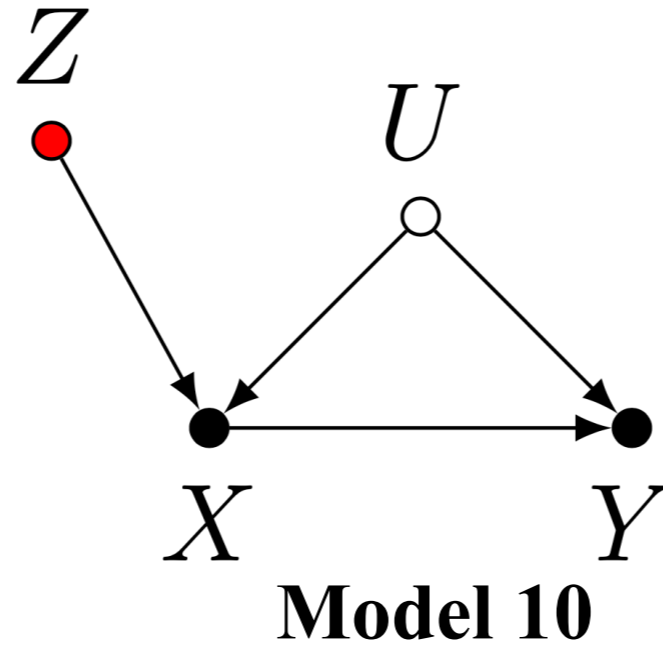
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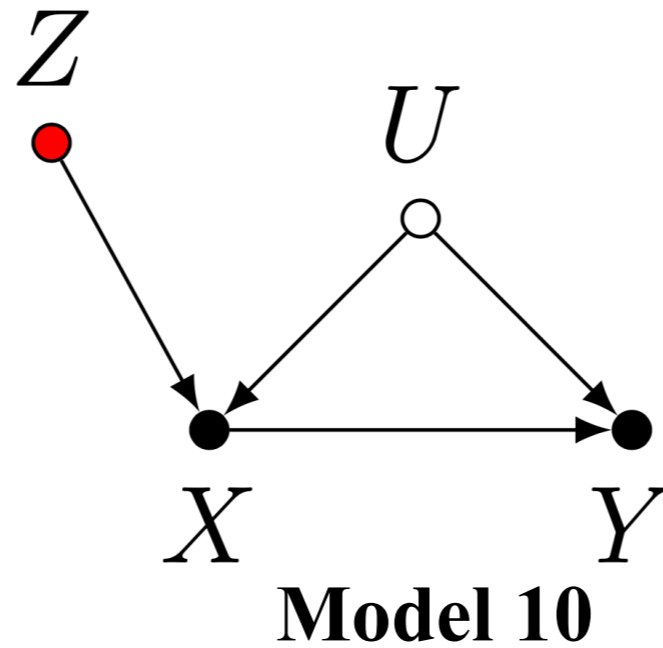
Thus, in Model 8, Z improves the precision of the ATE estimate;

Whereas in Model 9 Z hurts the precision of the ATE estimate.

“Bad” Control (bias amplification)

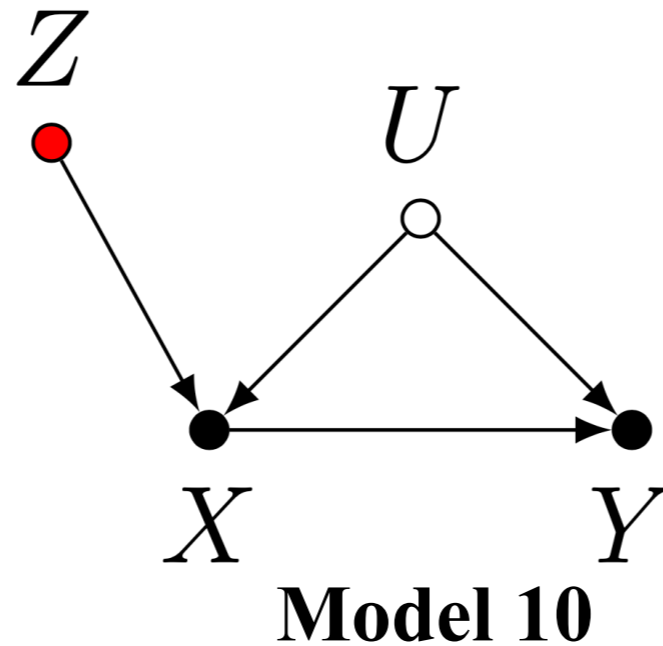


“Bad” Control (bias amplification)



We now encounter our second “pre-treatment” bad control.

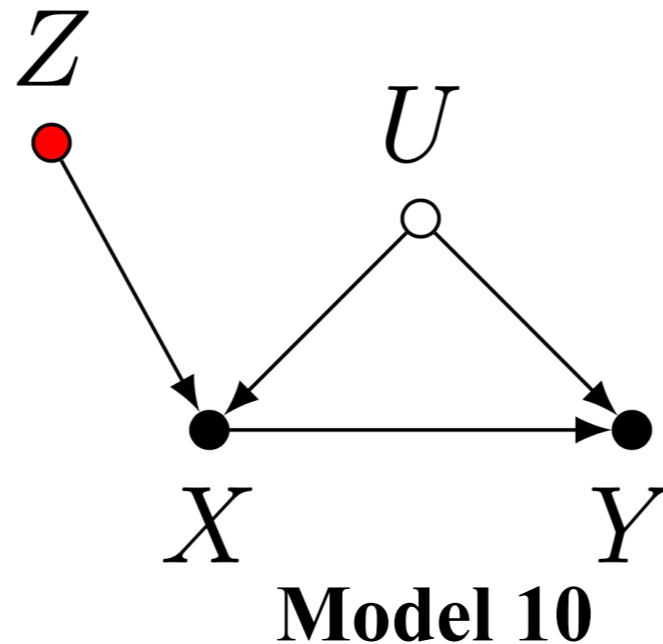
“Bad” Control (bias amplification)



We now encounter our second “pre-treatment” bad control.

Note here that: (i) Z is “pre-treatment;”; (ii) Z is associated with X (causally); (ii) Z is associated with Y; and (iii) Z is associated with Y conditional on X.

“Bad” Control (bias amplification)

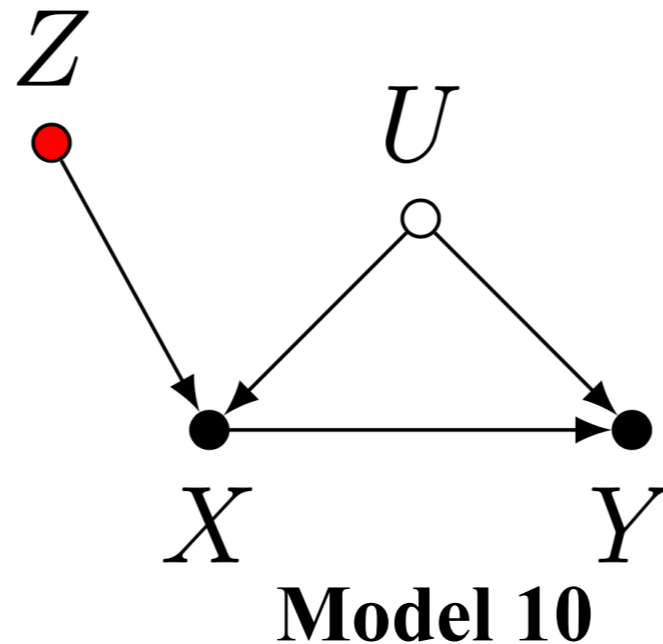


We now encounter our second “pre-treatment” bad control.

Note here that: (i) Z is “pre-treatment;”; (ii) Z is associated with X (causally); (ii) Z is associated with Y; and (iii) Z is associated with Y conditional on X.

Thus, Z seems like an ordinary confounder begging to be controlled.

“Bad” Control (bias amplification)



We now encounter our second “pre-treatment” bad control.

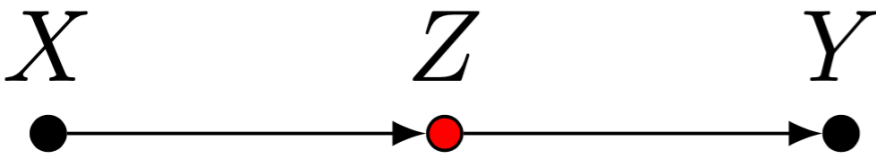
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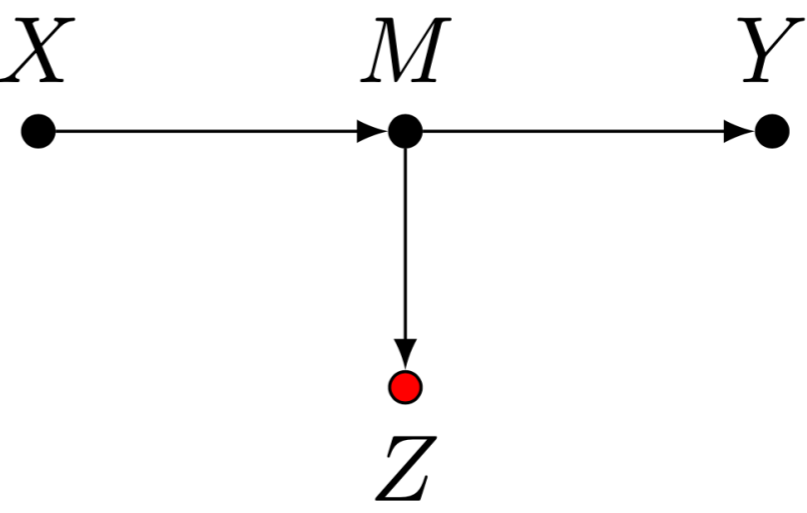
However, analysis shows that adjusting for Z will not only fail to deconfound the effect of X on Y , but, in linear models, it will *amplify* any existing bias.

“Bad” Controls (overcontrol bias)

Model 11

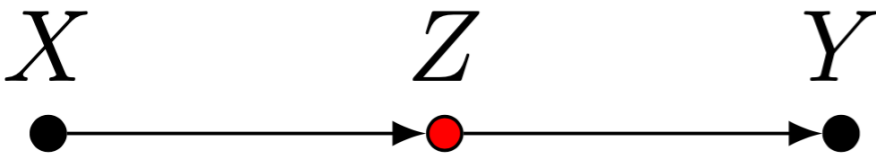


Model 12

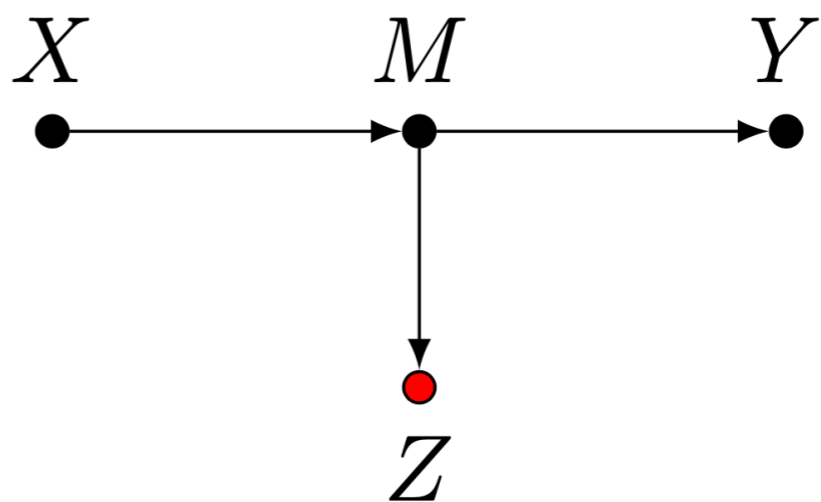


“Bad” Controls (overcontrol bias)

Model 11



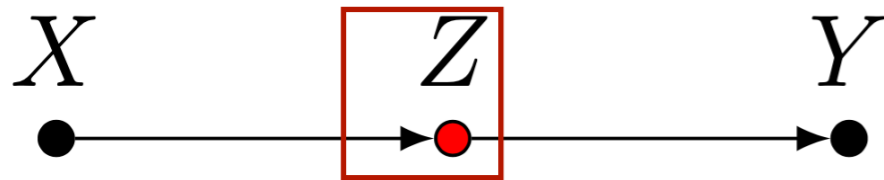
Model 12



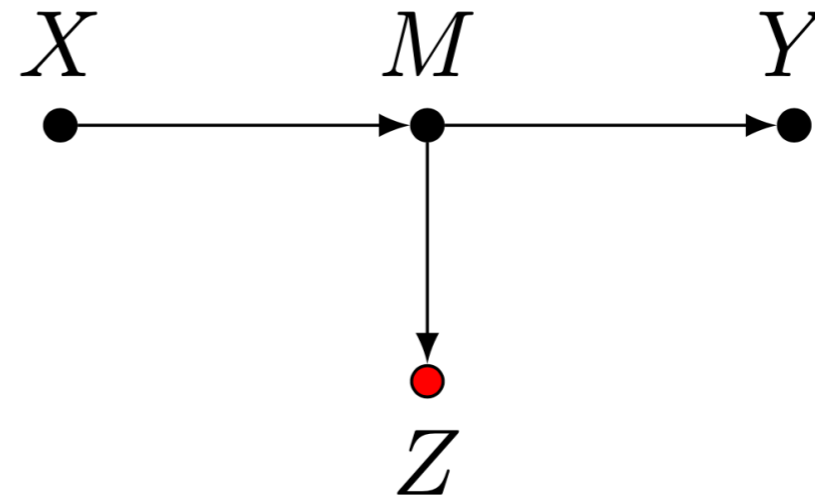
If our target quantity is the ATE, we want to leave all channels through which the causal effect flows “untouched.”

“Bad” Controls (overcontrol bias)

Model 11



Model 12

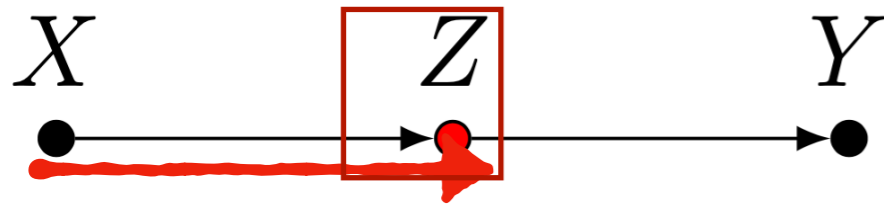


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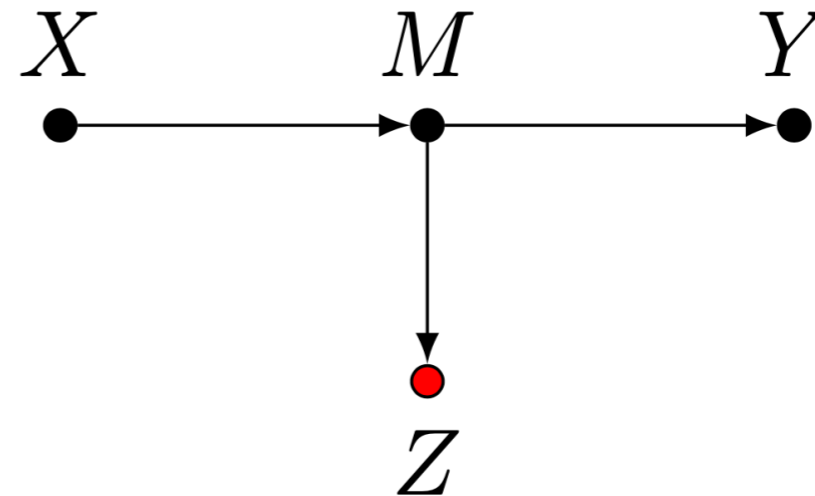
In Model 11, Z is a *mediator* of the causal effect of X on Y . Controlling for Z will block the very effect we want to estimate (the total effect of X on Y), thus biasing our estimates (this is usually known as “overcontrol bias”).

“Bad” Controls (overcontrol bias)

Model 11



Model 12

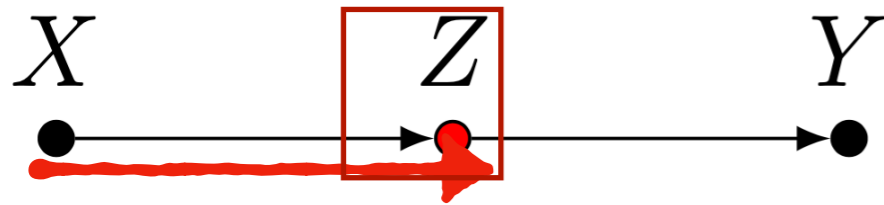


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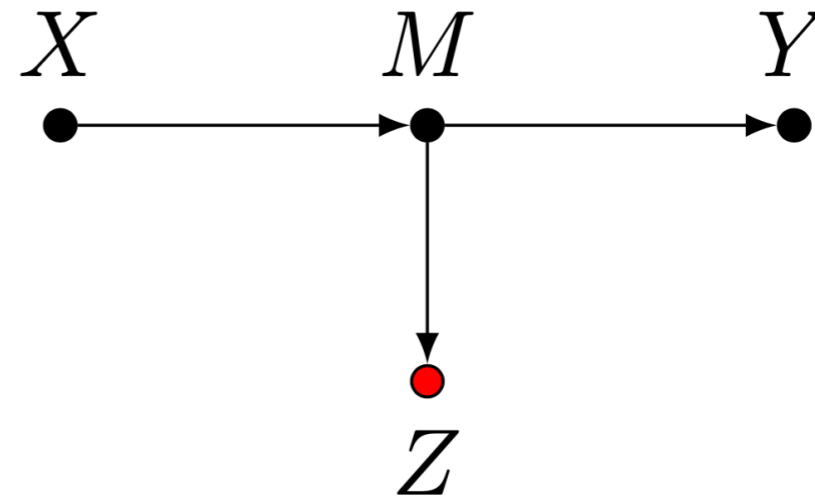
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“Bad” Controls (overcontrol bias)

Model 11



Model 12

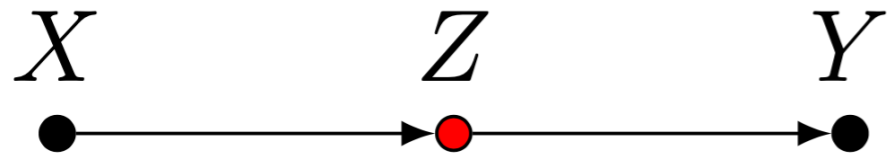


If our target quantity is the ATE, we want to leave all channels through which the causal effect flows “untouched.”

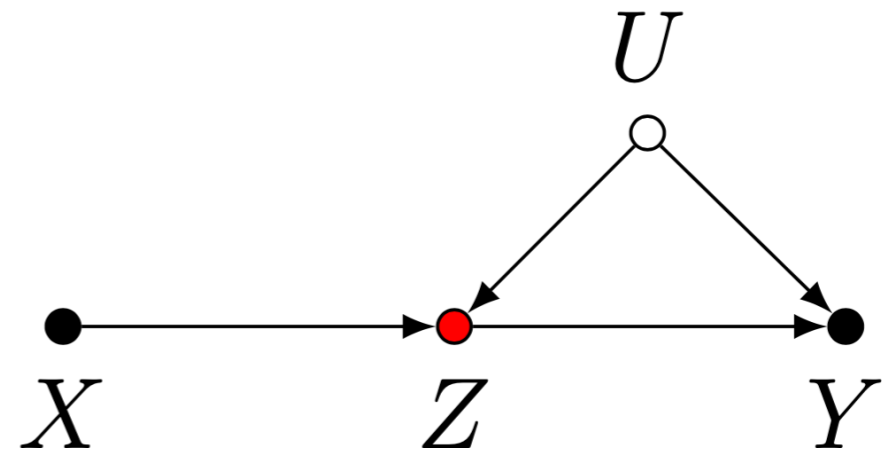
In Model 11, Z is a *mediator* of the causal effect of X on Y . Controlling for Z will block the very effect we want to estimate (the total effect of X on Y), thus biasing our estimates (this is usually known as “overcontrol bias”).

In Model 12, although Z is not itself a mediator of the causal effect of X on Y , *controlling for Z is equivalent to partially controlling for the mediator M* , and will thus bias our estimates.

Total versus direct effect

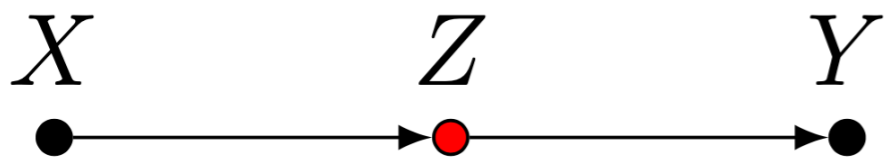


Model 11

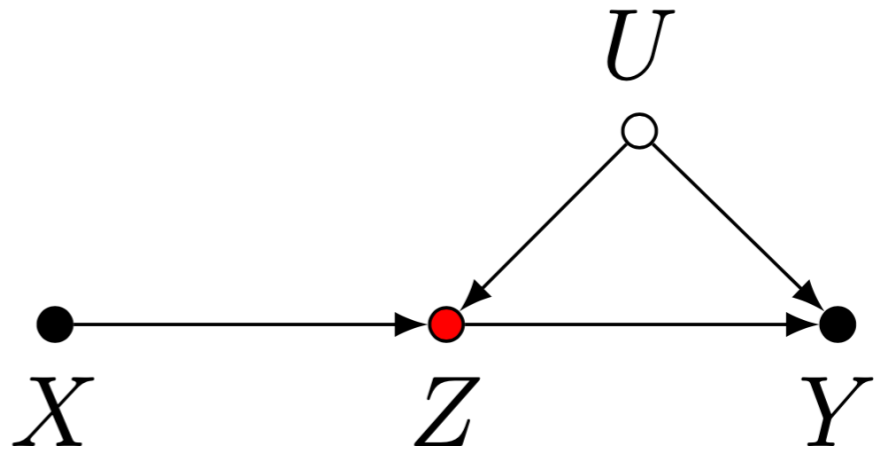


Variation of Model 11

Total versus direct effect



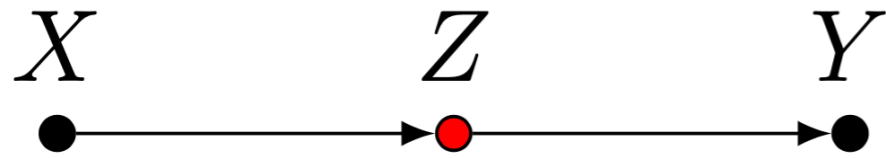
Model 11



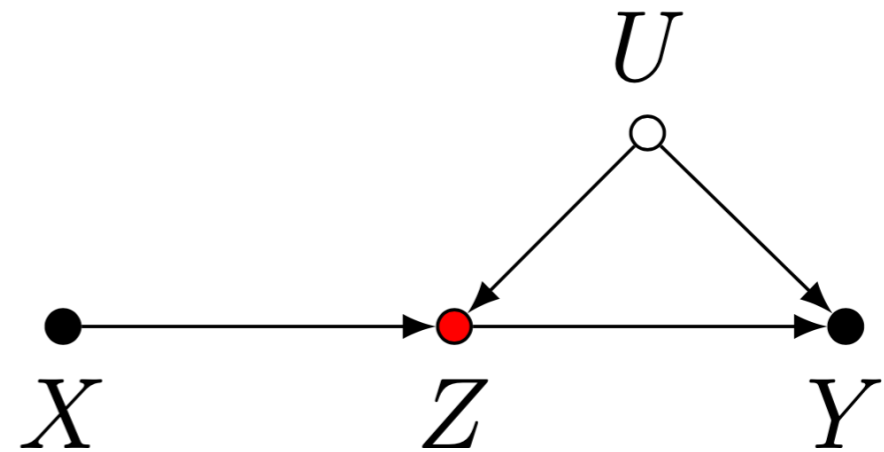
Variation of Model 11

The previous considerations assumed we were interested in the *total* effect of X on Y. If we were interested in the *controlled direct effect (CDE)* of X on Y, then adjusting for Z in Model 11 would *indeed be appropriate*.

Total versus direct effect



Model 11

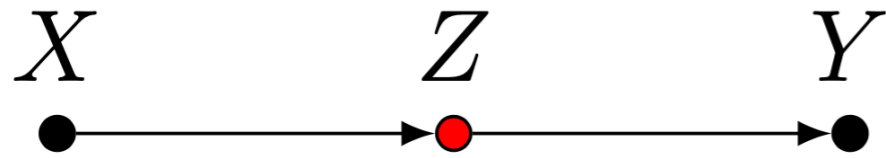


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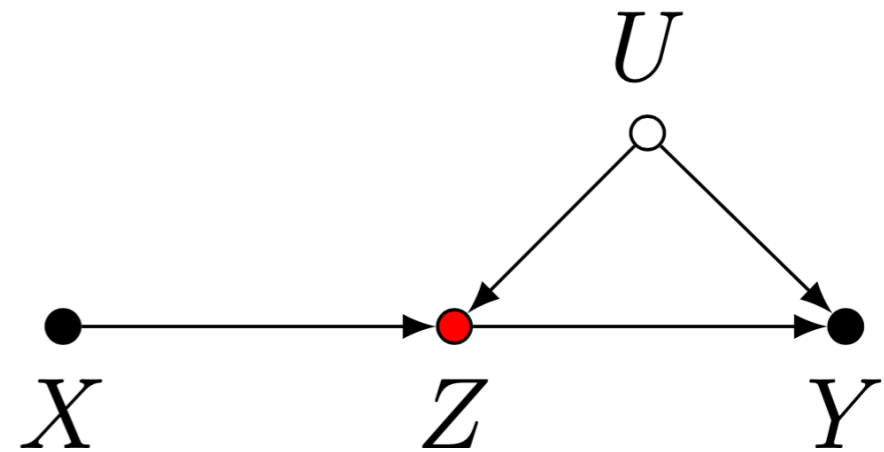
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Total versus direct effect



Model 11



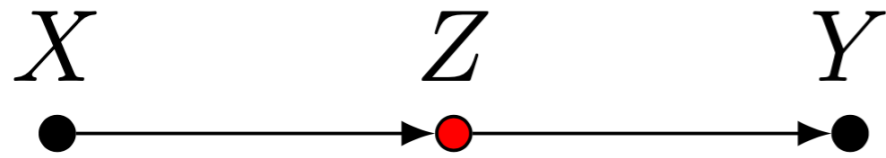
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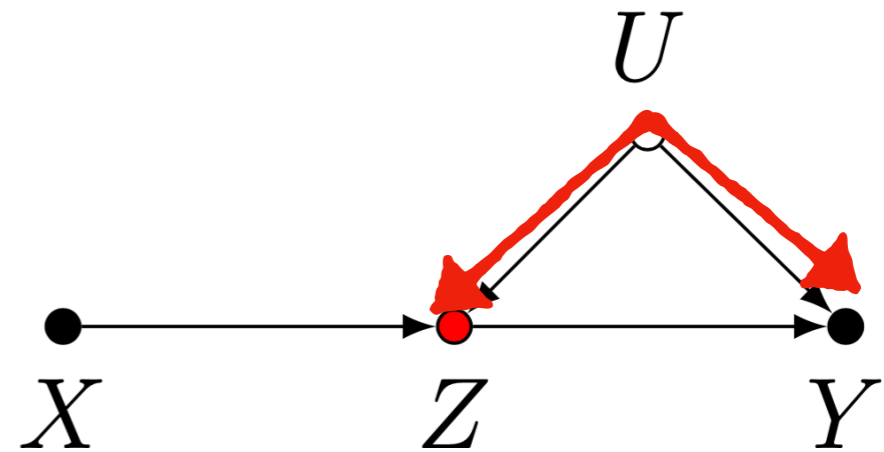
However, consider a variation of Model 11 with an unobserved confounder of Z and Y , denoted by U .

First notice that U does not confound the effect of X on Y . Thus the total effect remains unbiased, as it were in Model 11, *so long as we do not adjust for Z* .

Total versus direct effect



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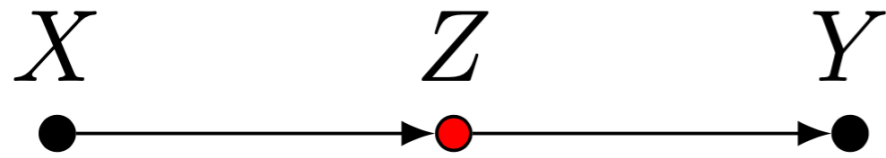
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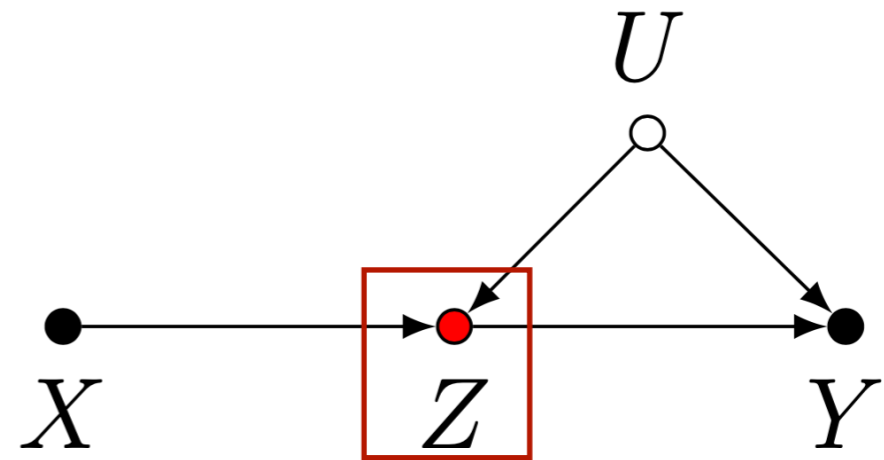
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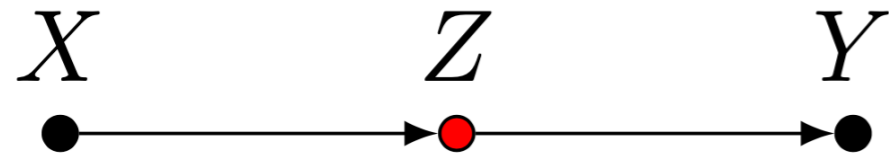
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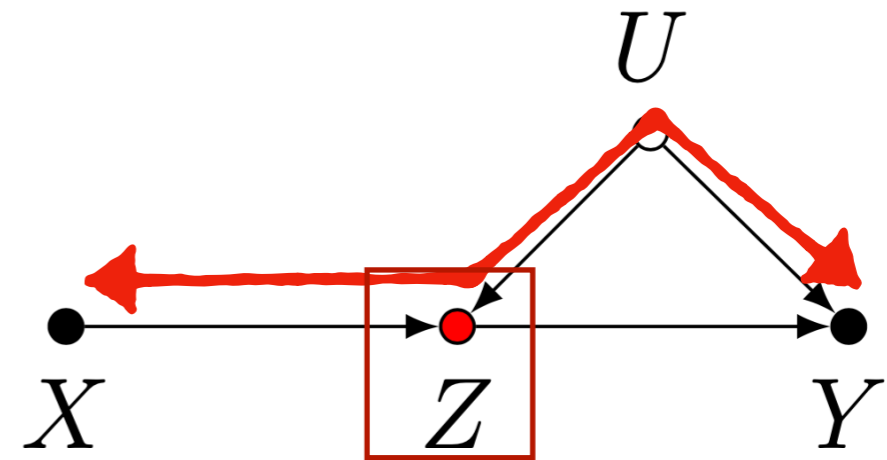
First notice that U does not confound the effect of X on Y . Thus the total effect remains unbiased, as it were in Model 11, *so long as we do not adjust for Z* .

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Total versus direct effect



Model 11



Variation of Model 11

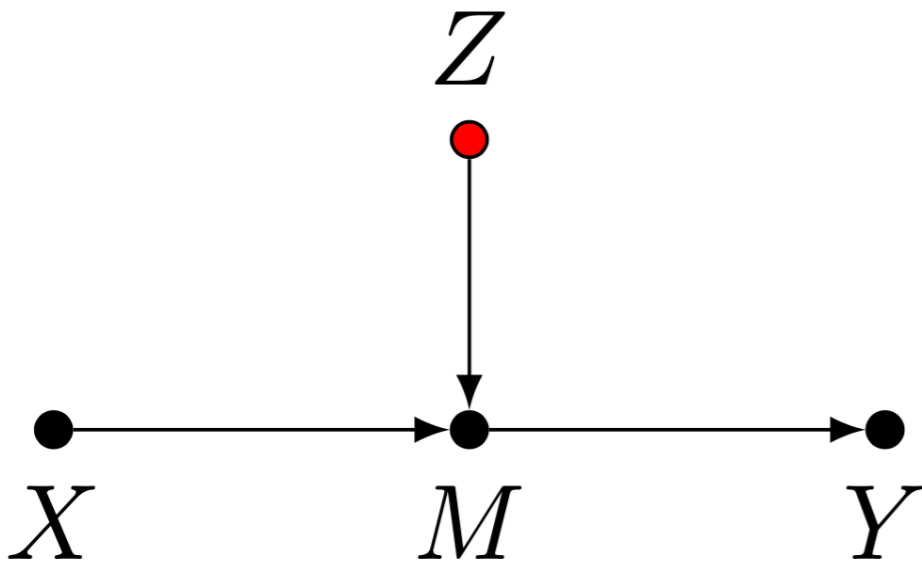
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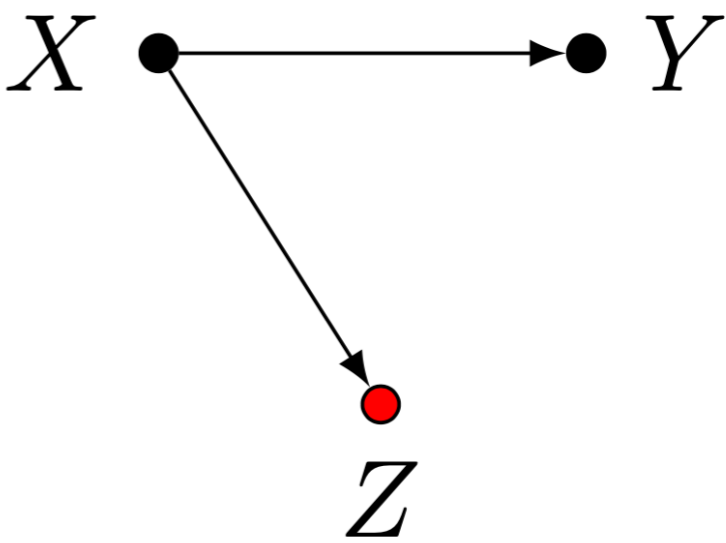
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“Neutral” Controls (but different impacts on precision)

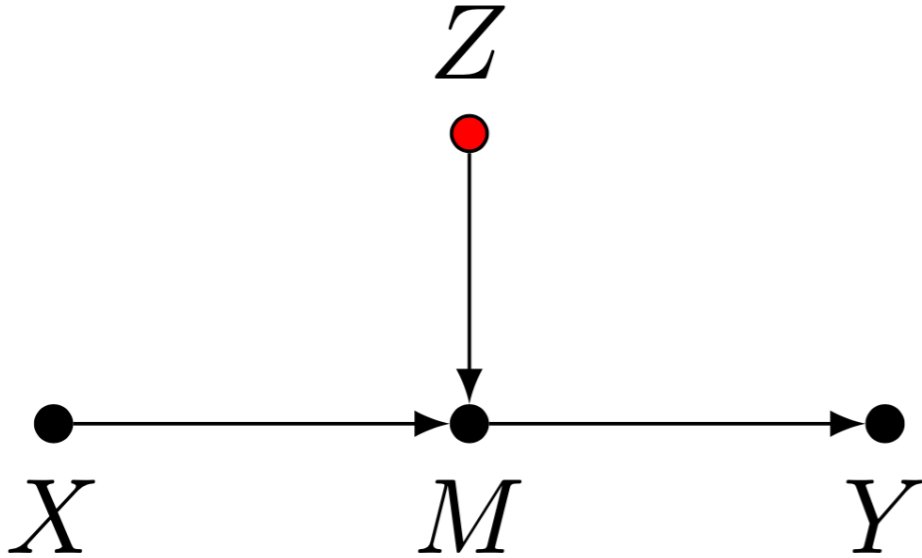


Model 13

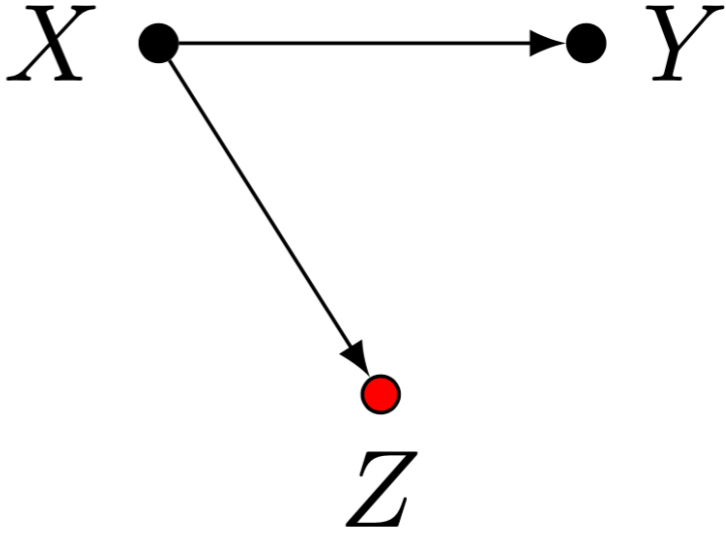


Model 14

“Neutral” Controls (but different impacts on precision)



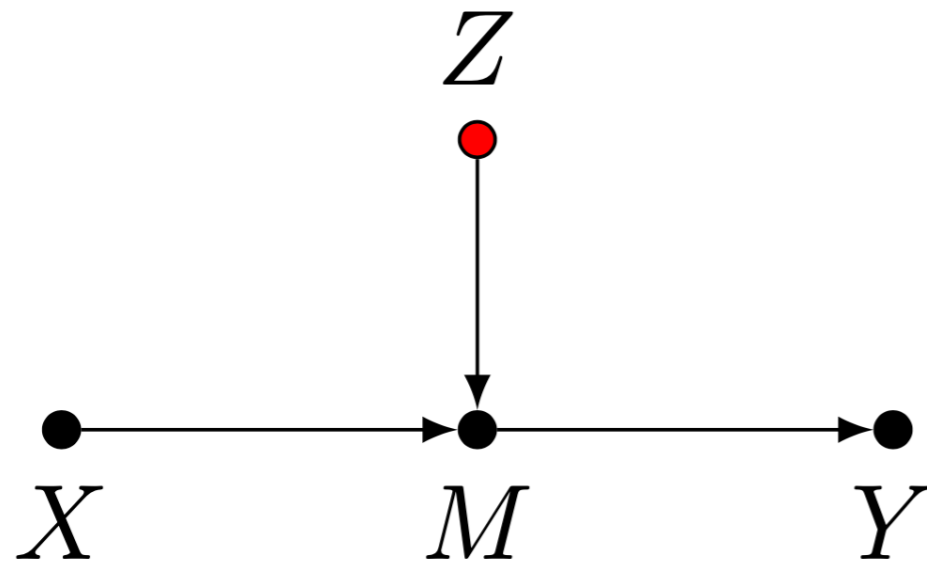
Model 13



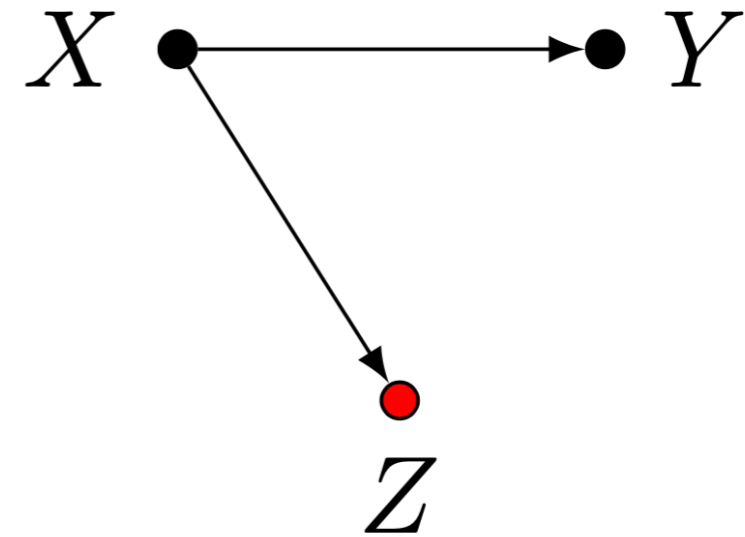
Model 14

At first look, one may think that adjusting for Z in Model 13 would bias the estimate of the ATE, by restricting variations of the mediator M.

“Neutral” Controls (but different impacts on precision)



Model 13

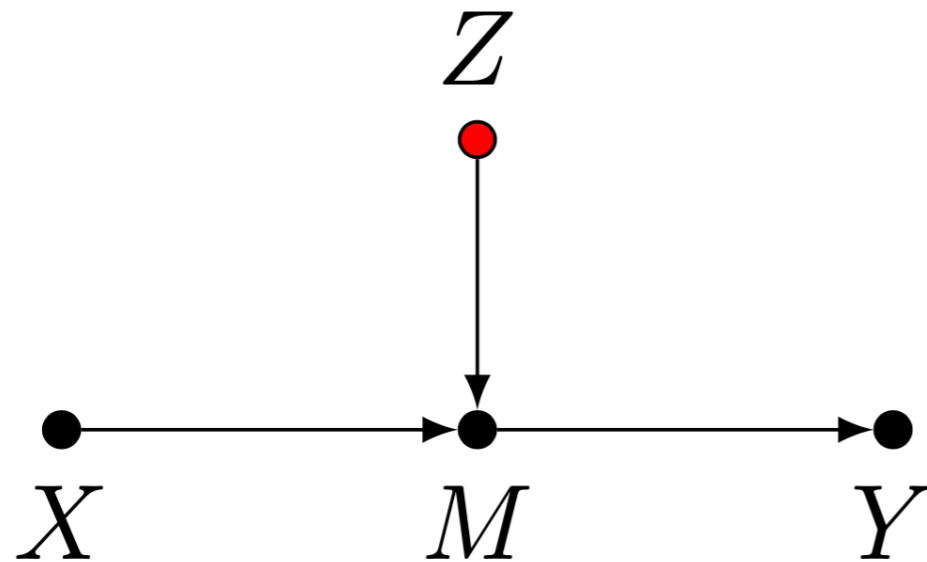


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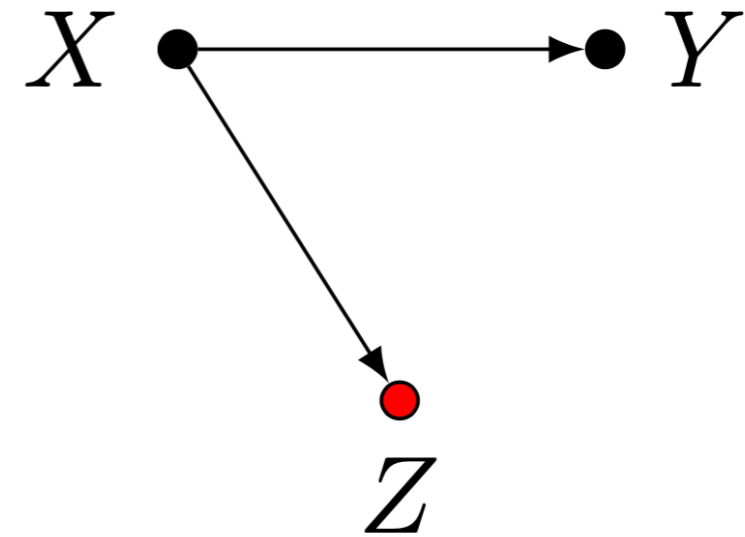
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However, the key difference here is that Z is a *cause*, not an *effect*, of M . Thus, Model 13 is analogous to Model 8, and controlling for Z will be neutral in terms of bias, and may *improve* the precision of the ATE estimate in finite samples.

“Neutral” Controls (but different impacts on precision)



Model 13



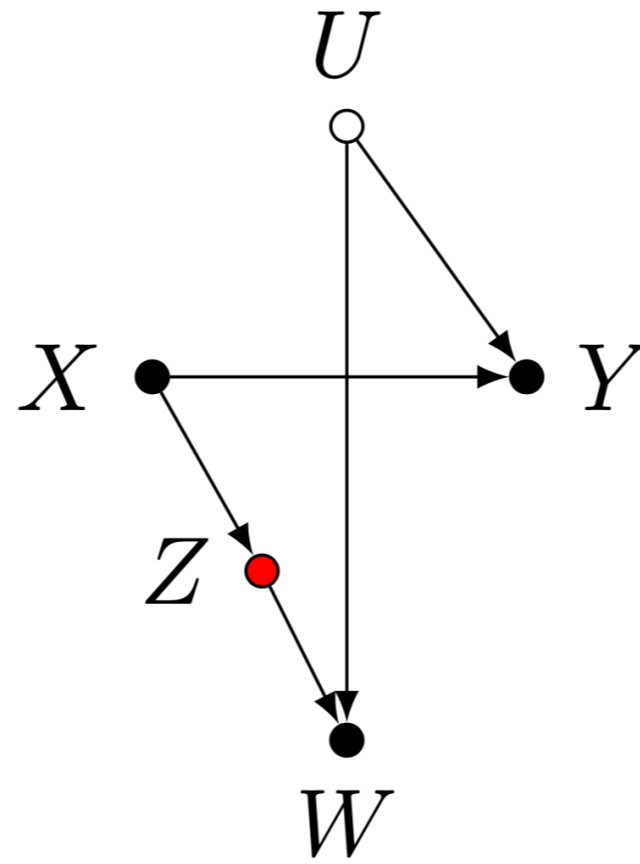
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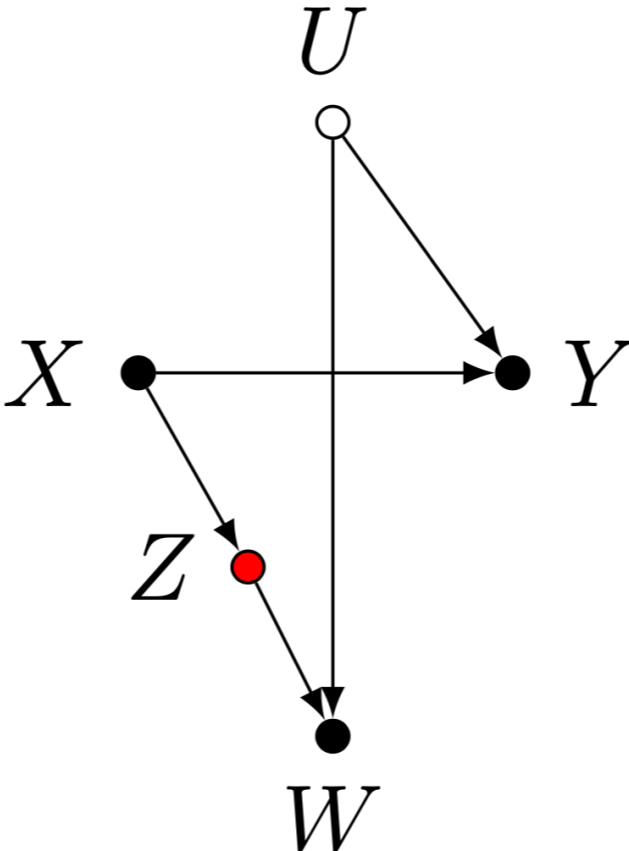
Contrary to folklore, not all “post-treatment” variables are inherently bad controls. In Model 14, Z is post-treatment, and controlling for Z does not open any confounding paths between X and Y . However, as before, controlling for Z may *hurt* the precision of the ACE estimate in finite samples.

“Good” control (in case of selection bias)



Model 15

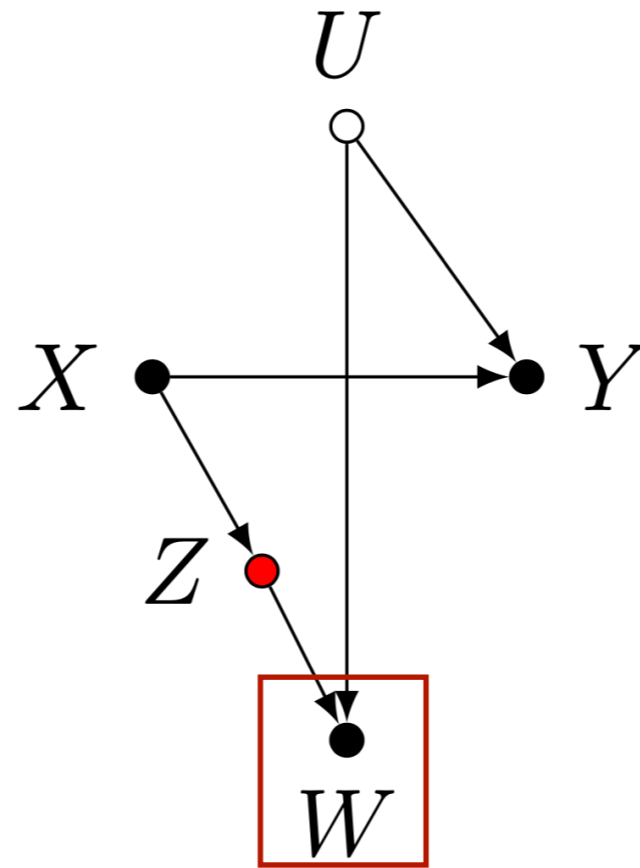
“Good” control (in case of selection bias)



Model 15

We now encounter our first “good” post-treatment variable.

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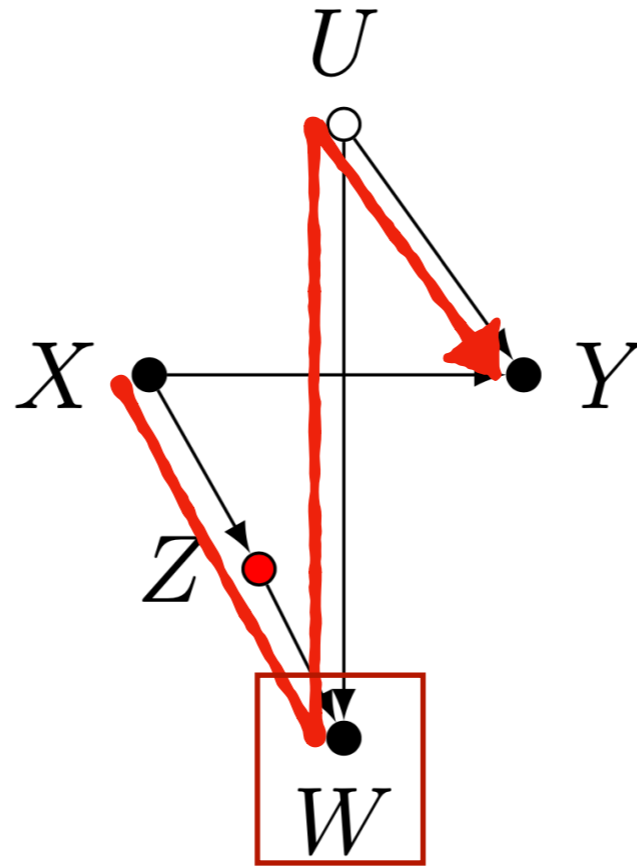


Model 15

We now encounter our first “good” post-treatment variable.

Suppose we only have observations with $W=1$ recorded. This means the colliding path $X \rightarrow Z \rightarrow W \leftarrow U \rightarrow Y$ is opened.

“Good” control (in case of selection bias)

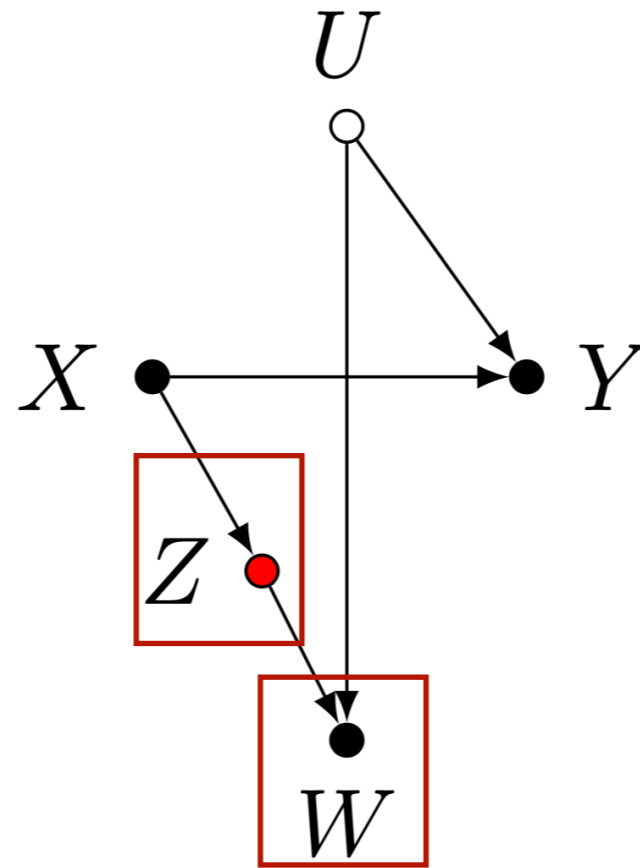


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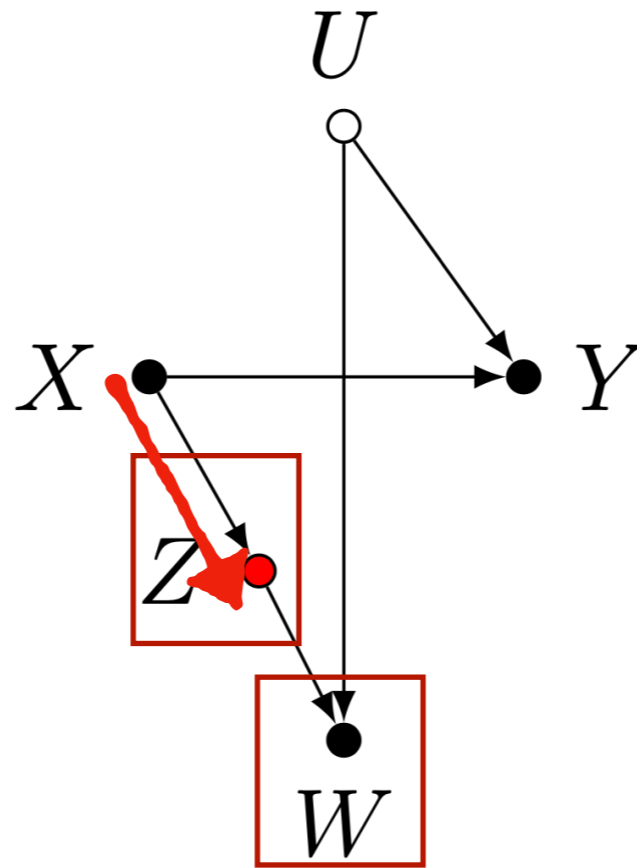
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However, further adjusting for Z blocks the colliding path. This allows us to estimate the conditional ATE (conditional on $W=1$). In linear models, we actually recover the full ATE.

“Good” control (in case of selection bias)



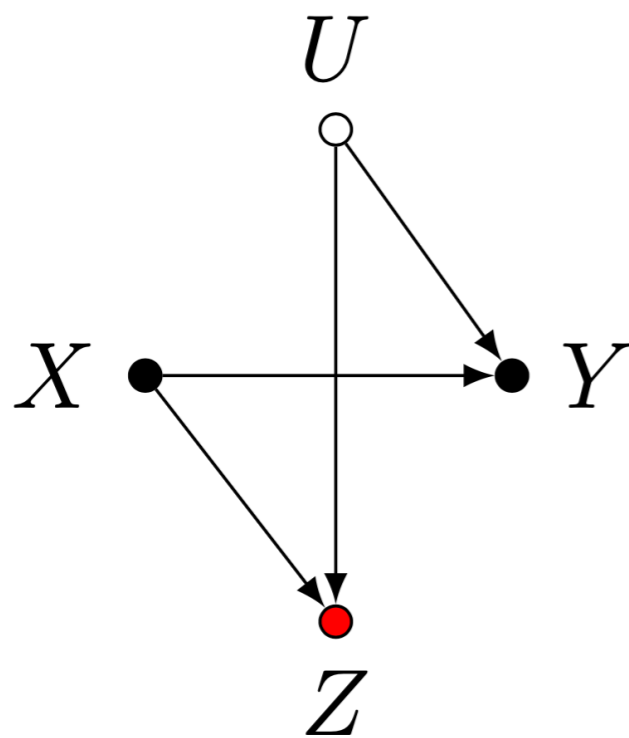
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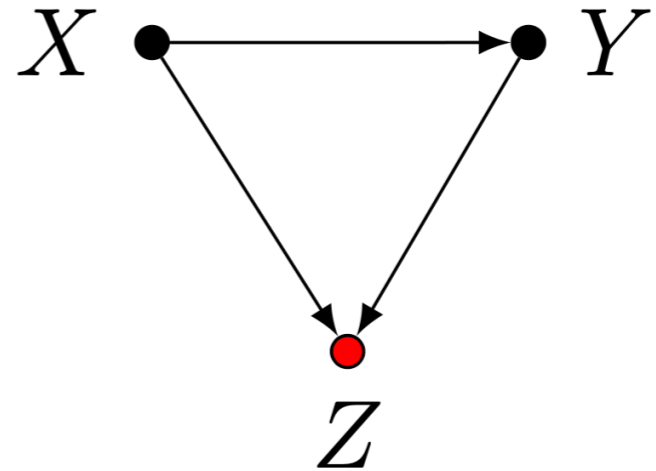
Suppose we only have observations with $W=1$ recorded. This means the colliding path $X \rightarrow Z \rightarrow W \leftarrow U \rightarrow Y$ is opened.

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“Bad” Controls (selection bias)

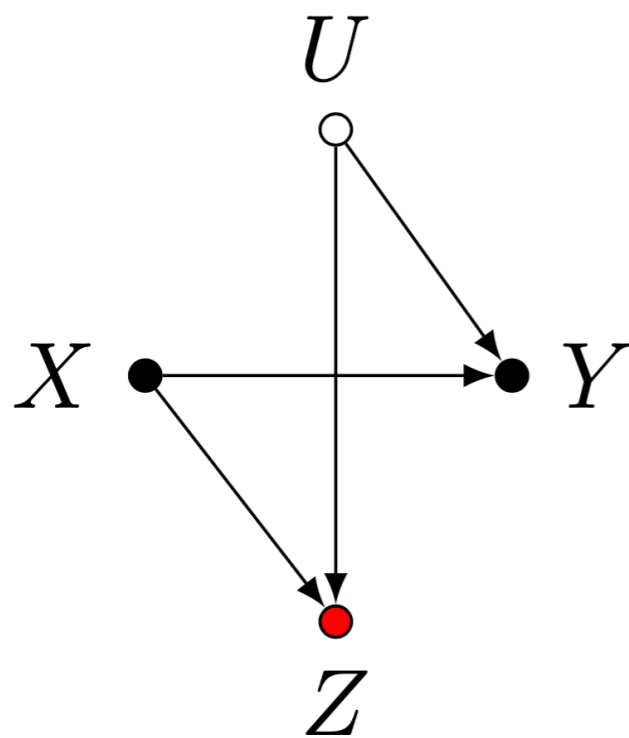


Model 16

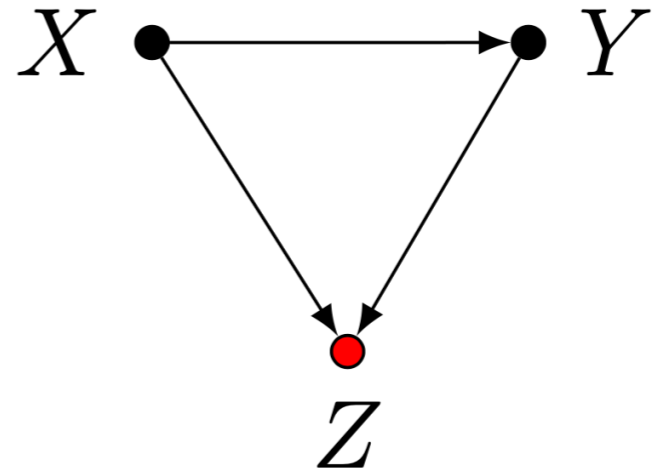


Model 17

“Bad” Controls (selection bias)



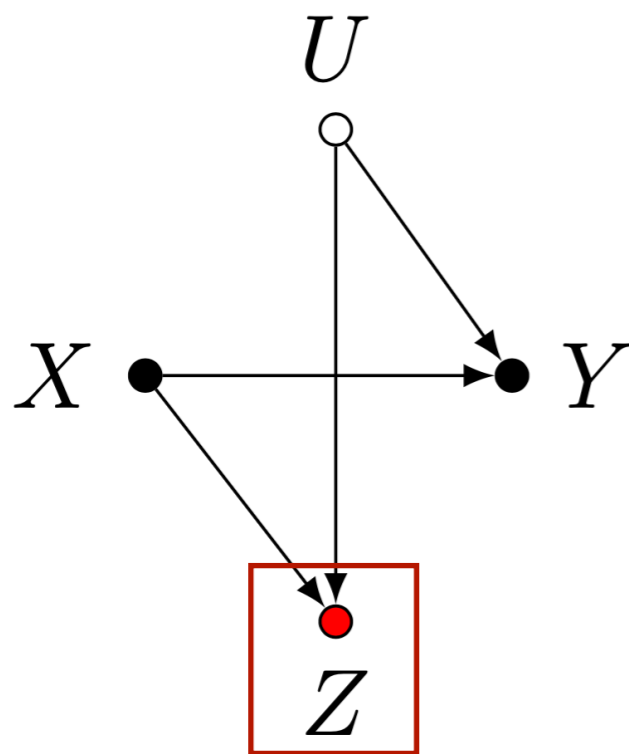
Model 16



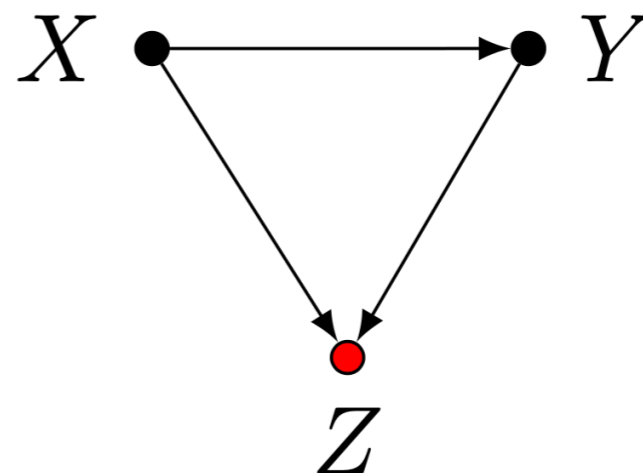
Model 17

Contrary to Models 14 and 15, here controlling for Z is no longer harmless, and induces what is classically known as “selection bias” or “collider bias.”

“Bad” Controls (selection bias)



Model 16

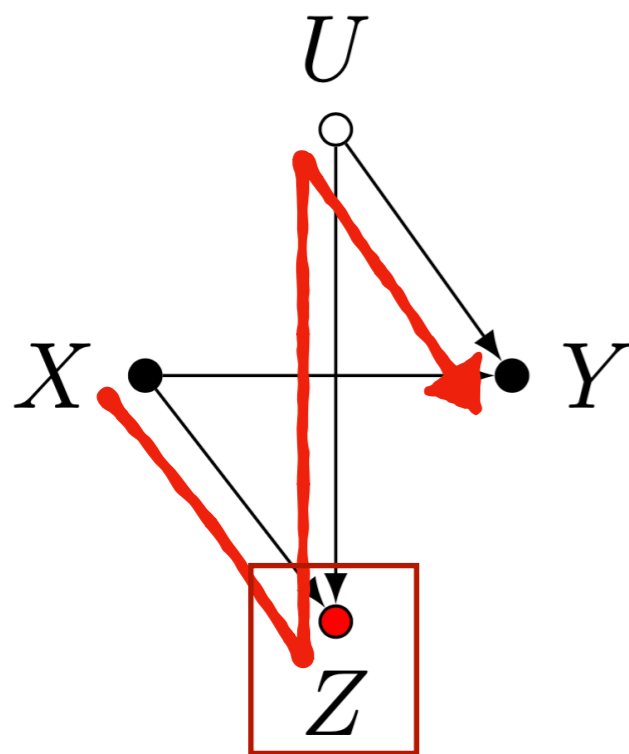


Model 17

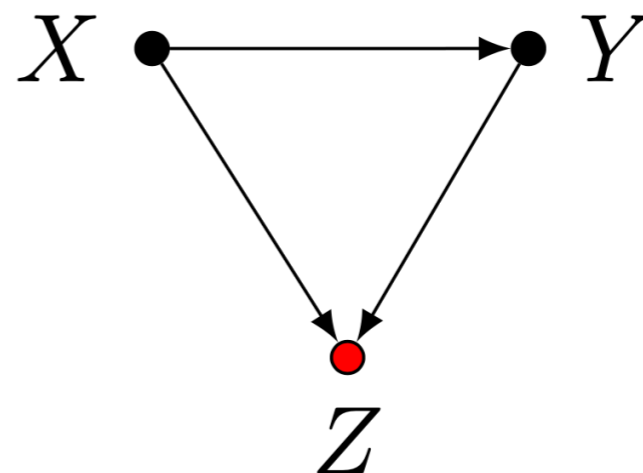
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Adjusting for Z in Model 16 opens the colliding path $X \rightarrow Z \leftarrow U \rightarrow Y$ and so biases the ATE.

“Bad” Controls (selection bias)



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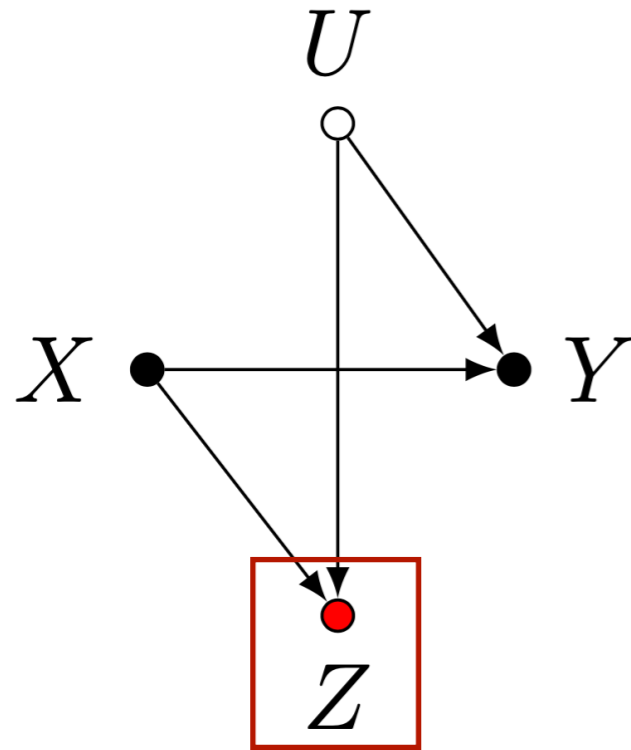


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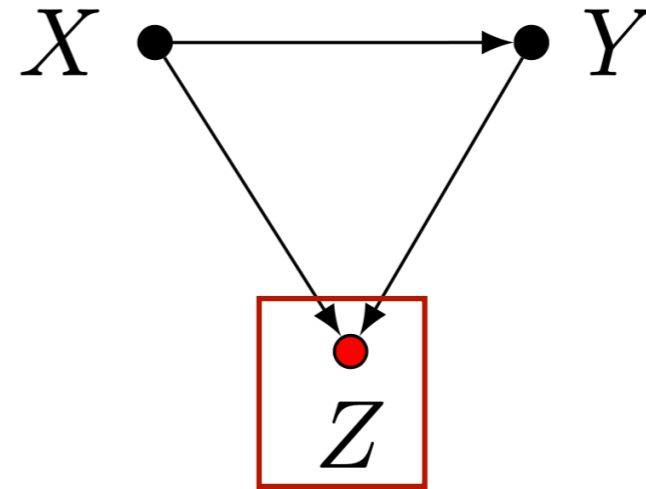
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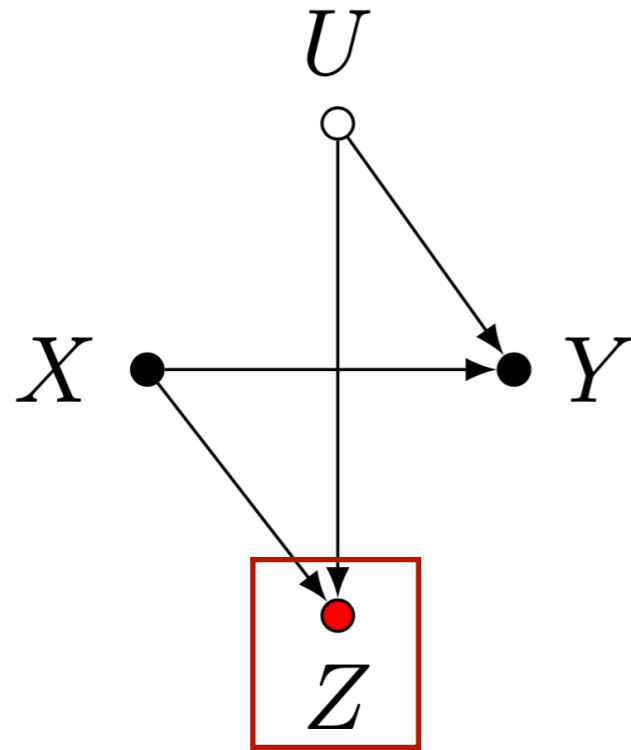
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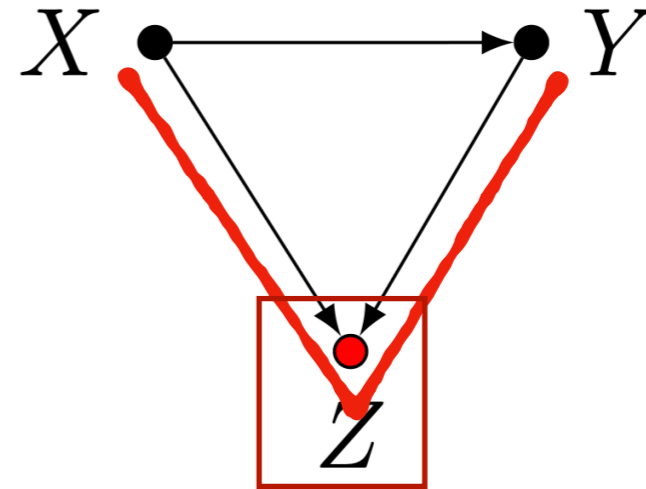
Adjusting for Z in Model 16 opens the colliding path $X \rightarrow Z \leftarrow U \rightarrow Y$ and so biases the ATE.

In Model 17, adjusting for Z not only opens the path $X \rightarrow Z \leftarrow Y$, but also the colliding path due to the latent parents of Y , thus biasing the ACE and motivating our final example.

“Bad” Controls (selection bias)



Model 16



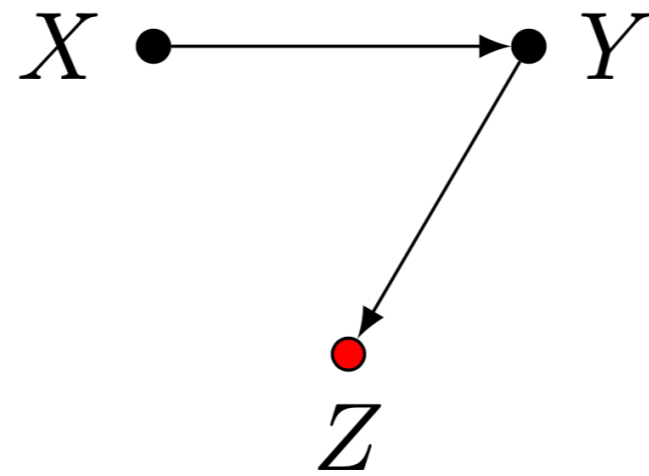
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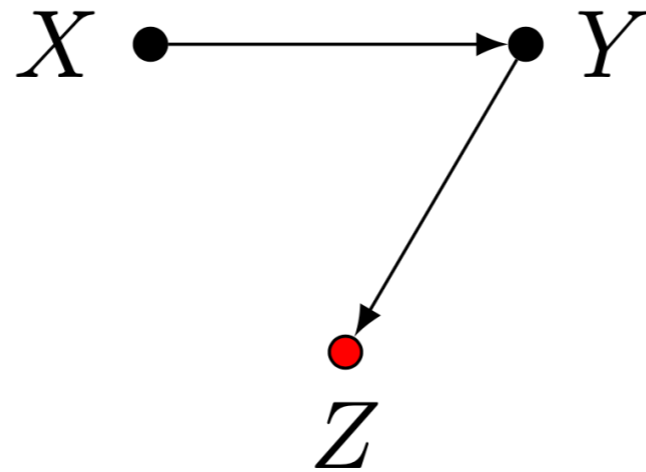
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“Bad” Controls (case-control bias)



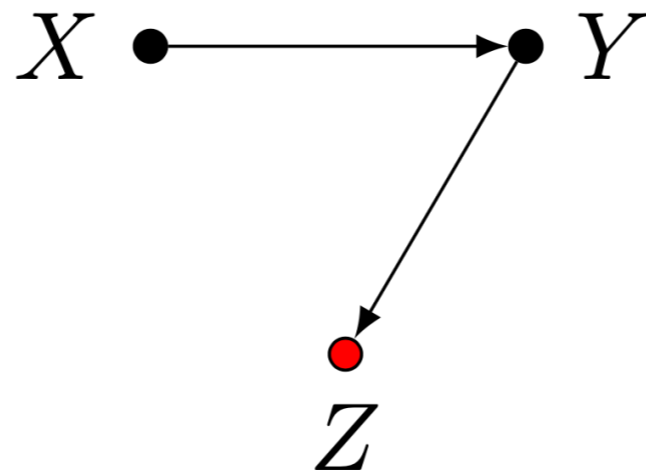
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In our last example, Z is not in the causal pathway from X to Y, Z is not a direct cause of X, and Z is connected to Y.

Thus, one might surmise that, as in Model 8, controlling for Z is harmless for identification, and perhaps beneficial for finite sample efficiency.

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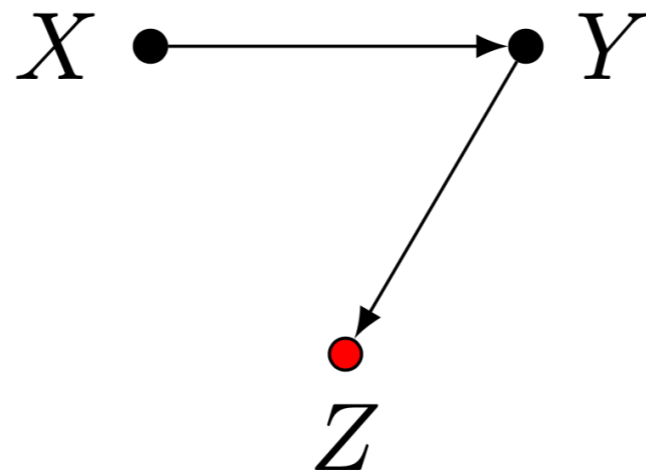


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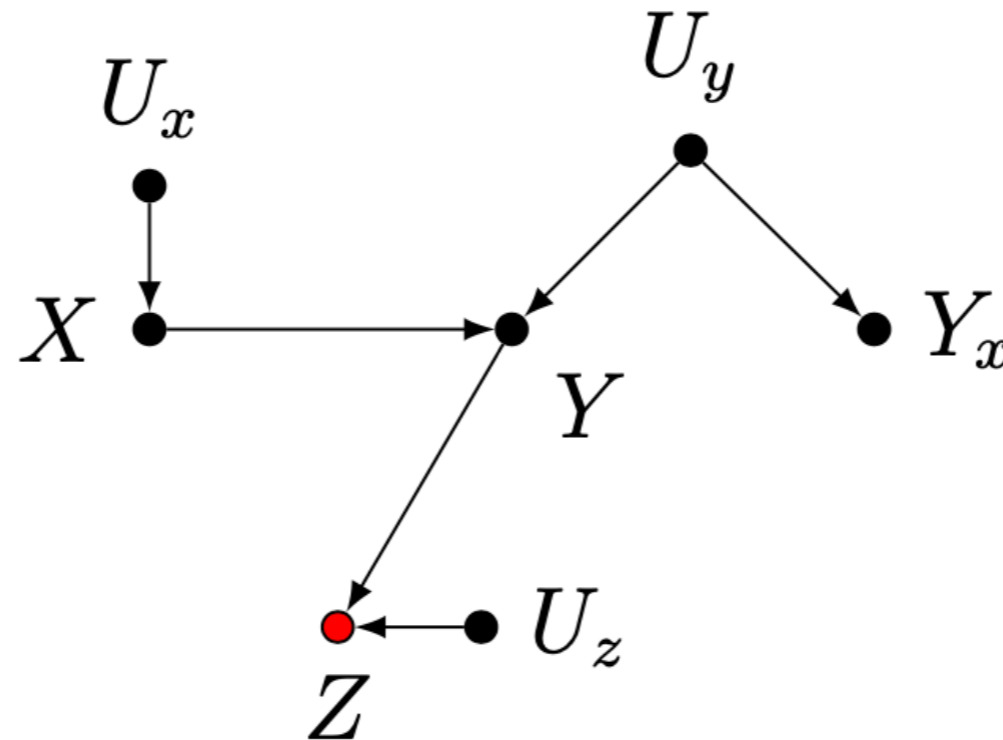
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Some Real Examples of Bad Controls

The birth-weight paradox

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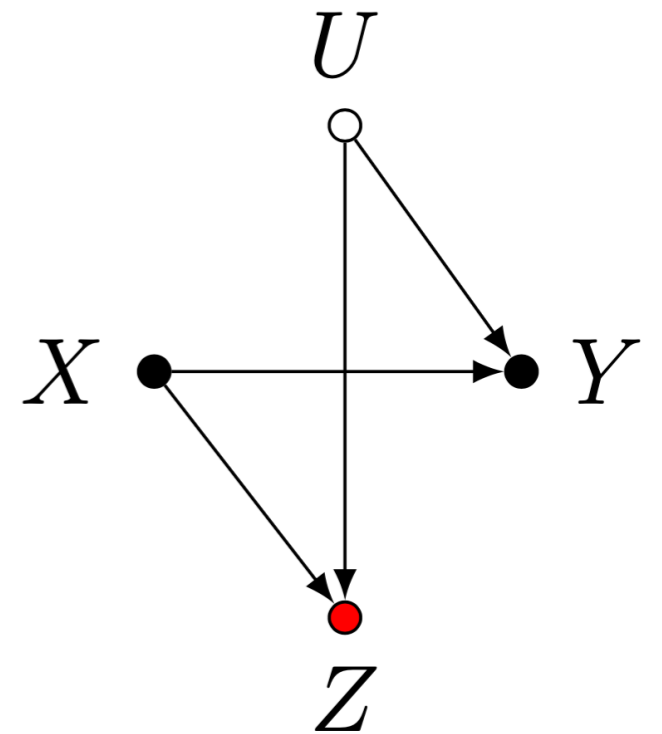
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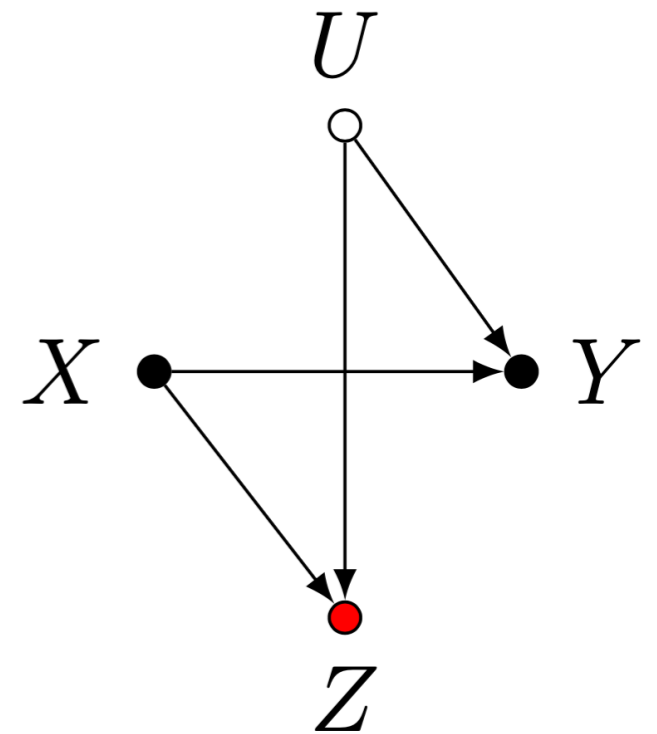
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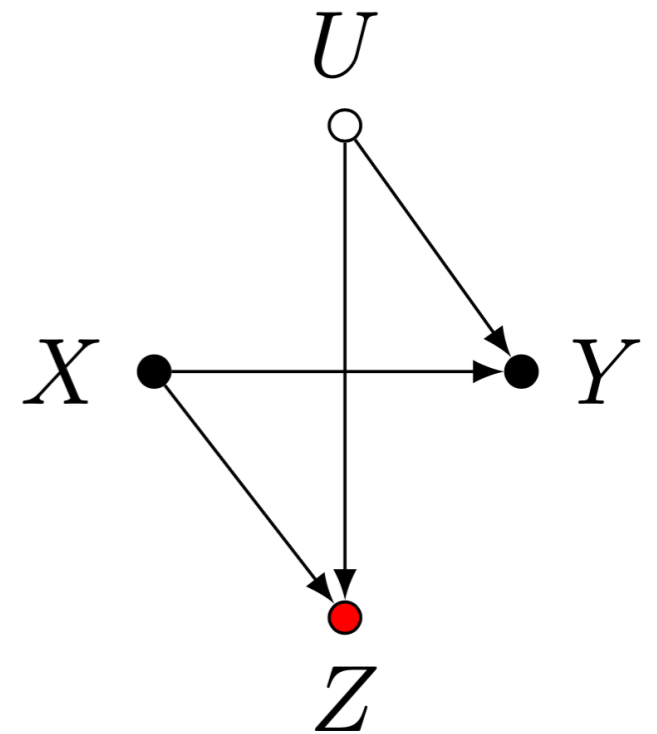
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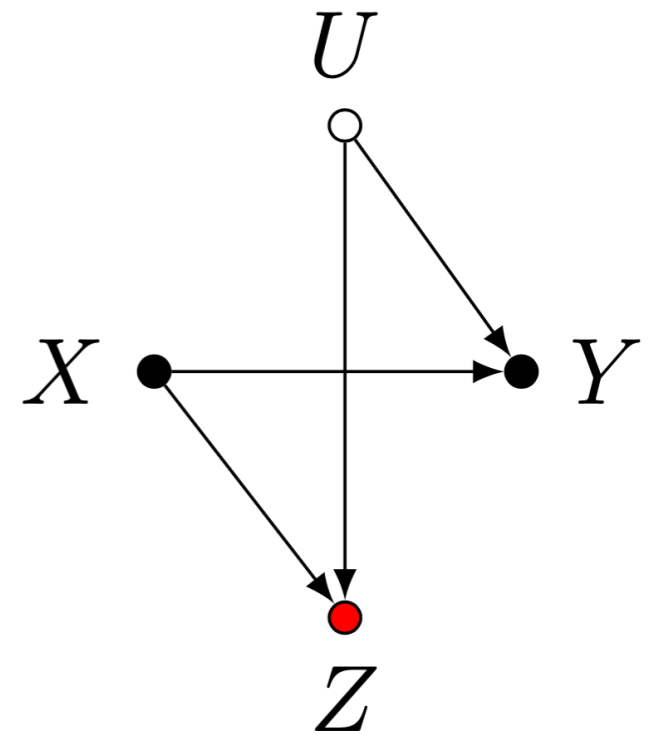
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LBW infants of non-smokers need to have alternative causes for their LBW (such as malnutrition), and such causes could also lead to higher mortality.



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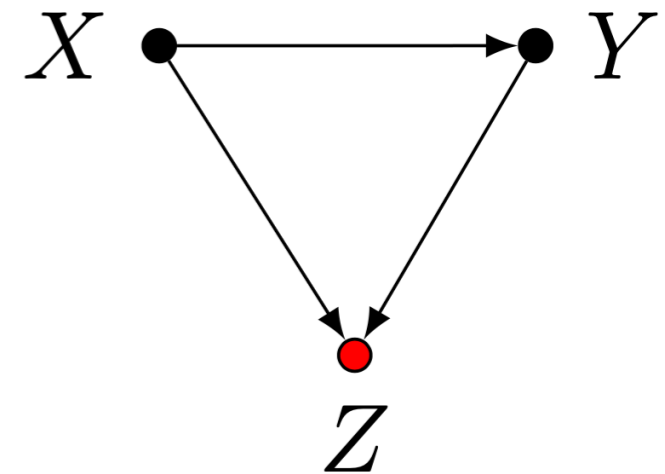
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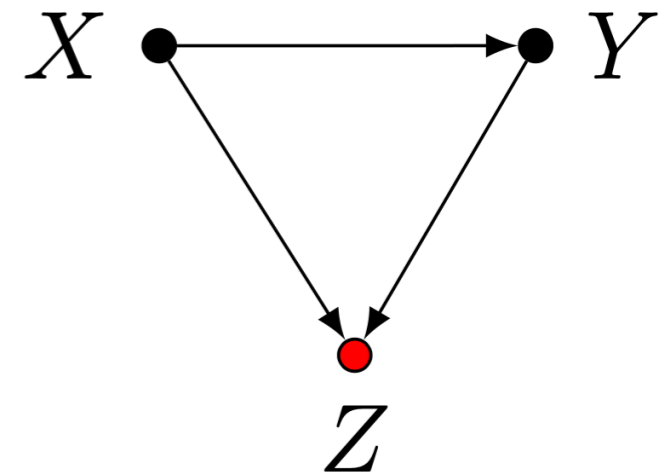
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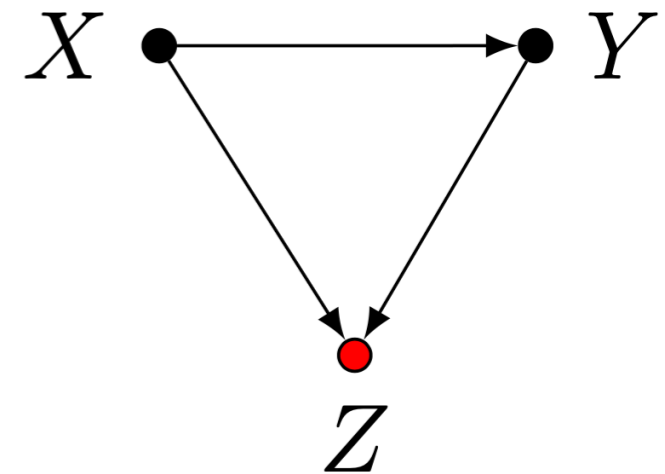
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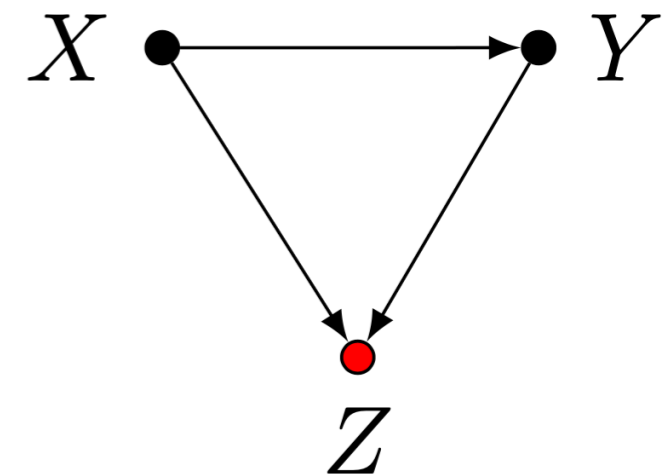
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Here one could argue that both childhood nutrition and adult height have pathways to committing a crime through socioeconomic opportunities, leading to selection bias.



Why you should not adjust for reading grade when estimating the causal effect of class size on math grade?



Jeffrey Wooldridge
@jmwooldridge



Suppose I have two 4th grade test scores, math4 and read4. I want to estimate the causal effect of class size on performance. Assume I have convincing controls. Is there a way to use a DAG to illustrate why I shouldn't include read4 in the equation for math4?

Why you should not adjust for reading grade when estimating the causal effect of class size on math grade?



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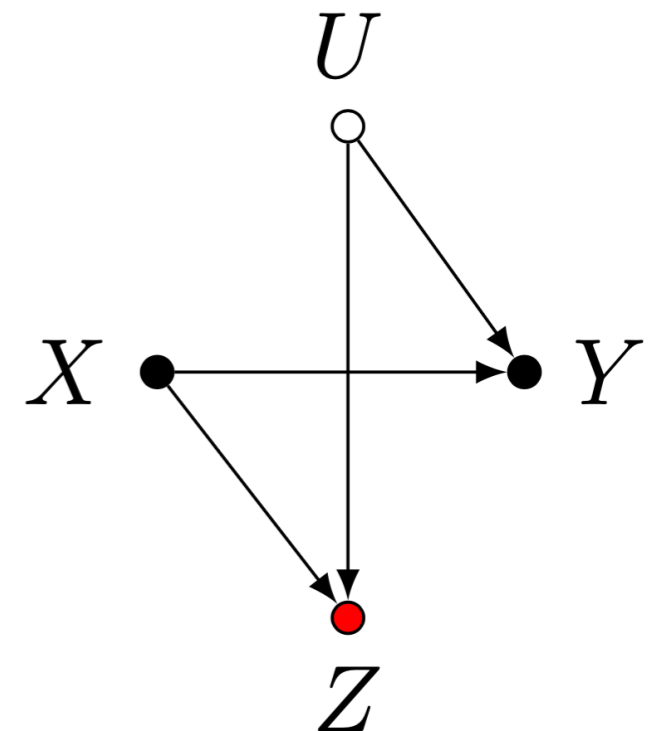
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Análise Real
@analysereal

...

We can illustrate this with Model 16 of the "Crash Course in Good and Bad Controls" (papers.ssrn.com/sol3/papers.cf...). Here X = class size, Y = math4, Z = read4, and U = student's ability. Conditioning on Z opens the path $X \rightarrow Z \leftarrow U \rightarrow Y$ and it is thus a "bad control."



What About Multiple Controls?

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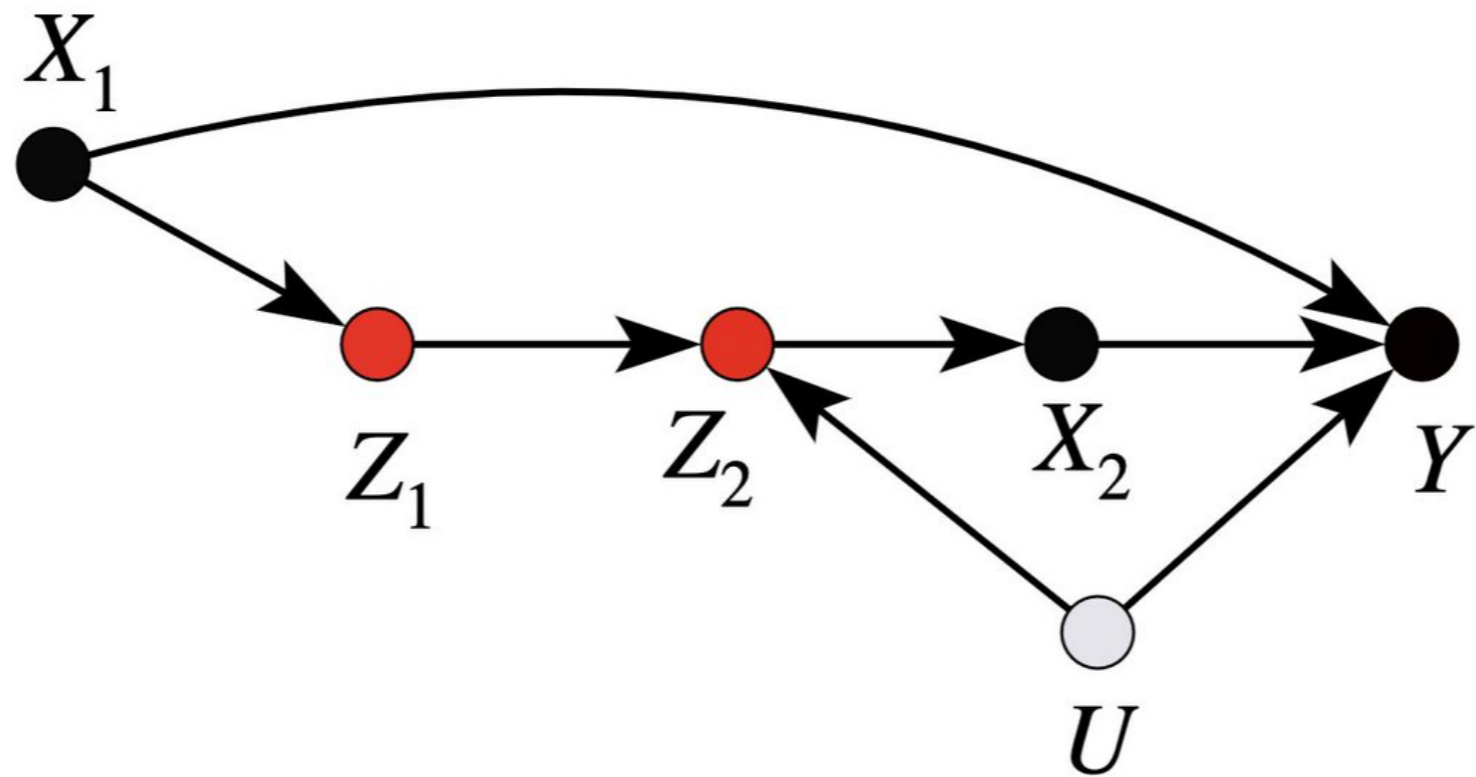
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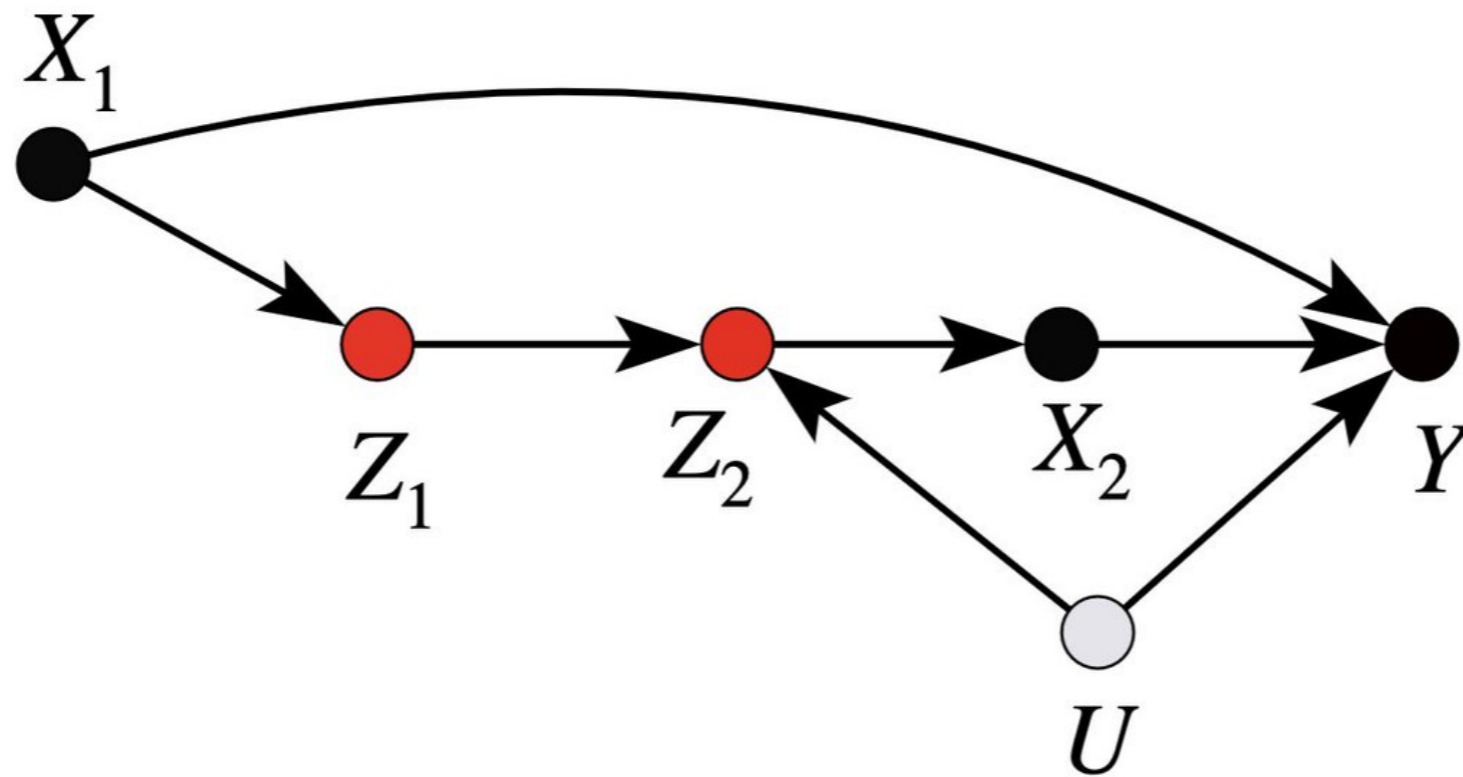
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There is open-source software with efficient procedures to identify (optimal) adjustment sets for you (*dagitty*, *causal fusion*, etc).

Challenge:

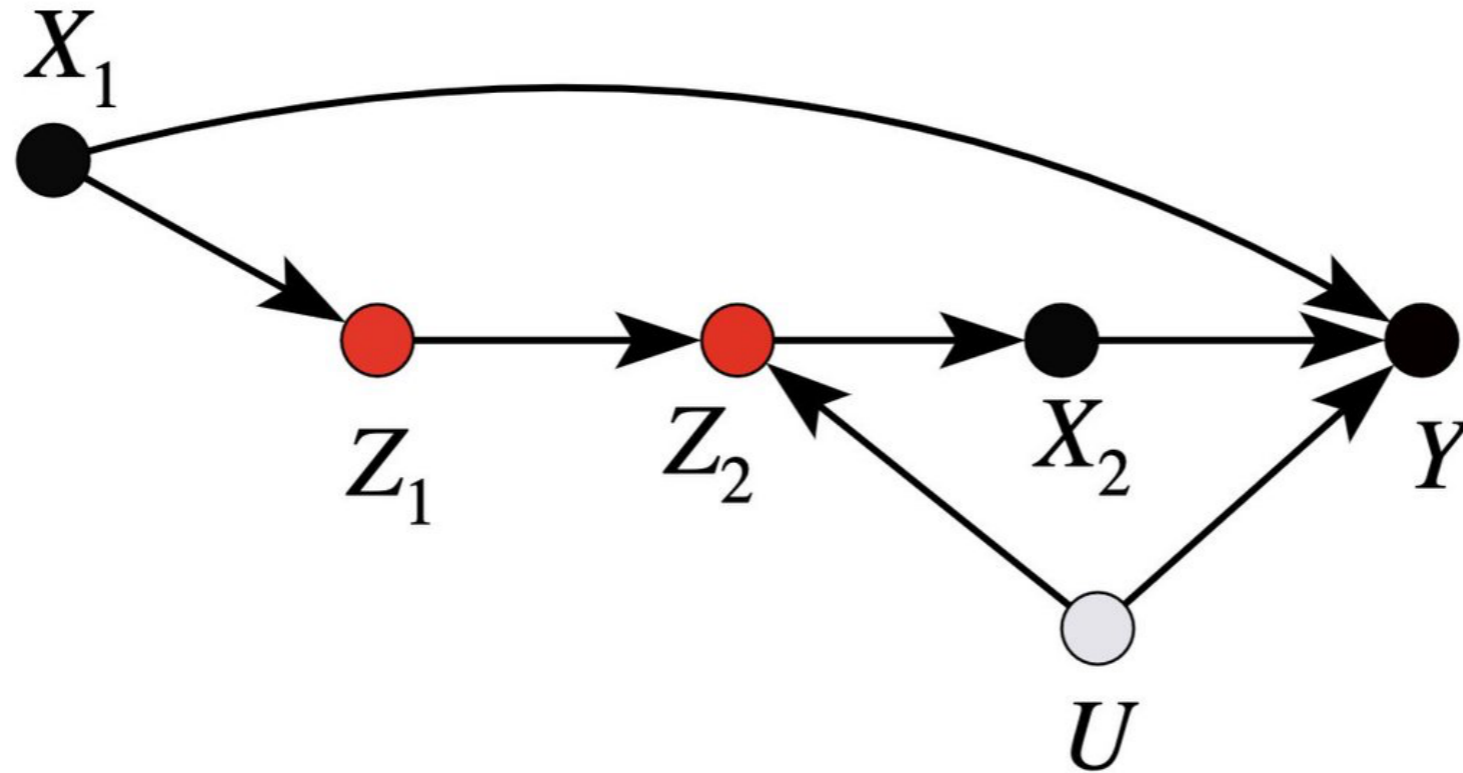


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Goal: estimate the *joint effect* of X_1 and X_2 on Y .

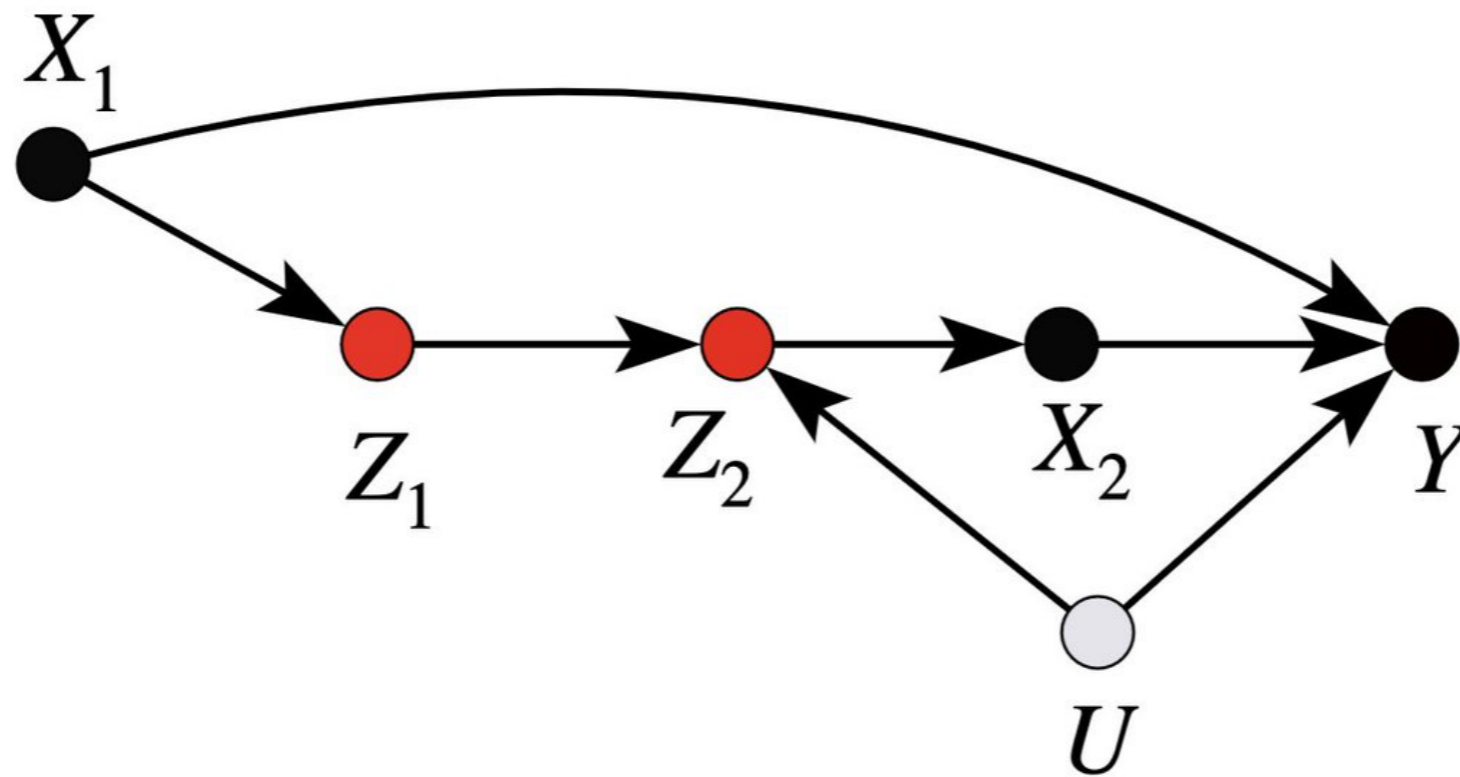
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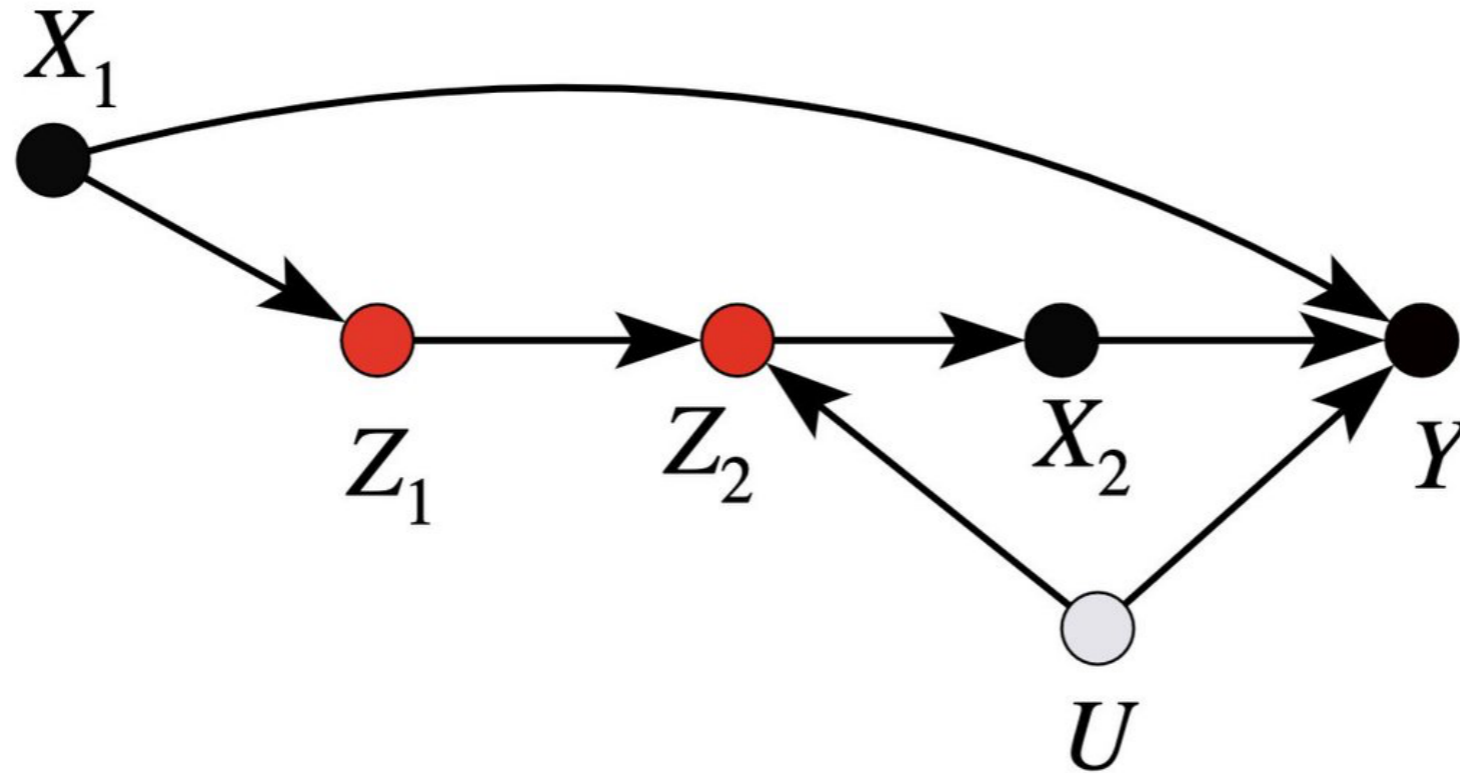


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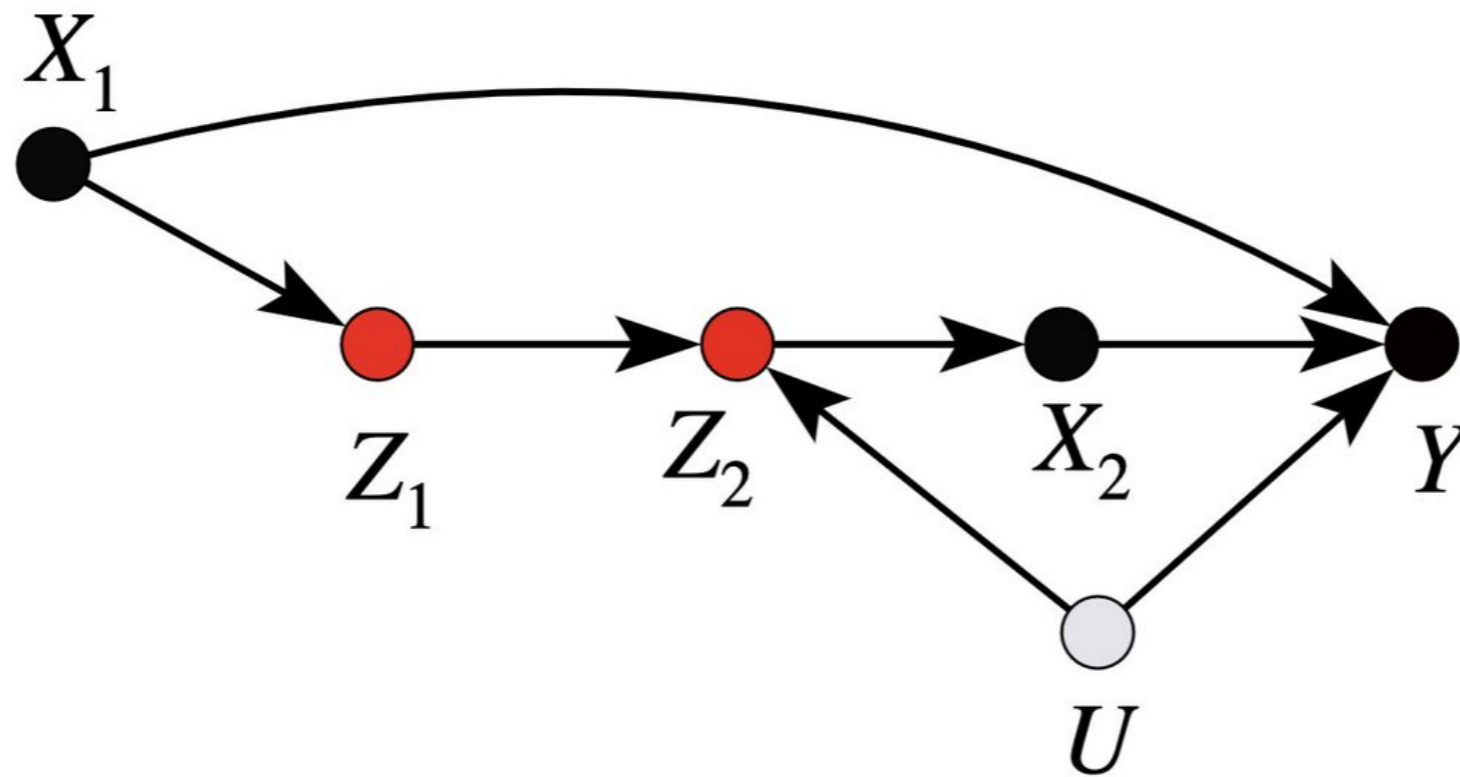
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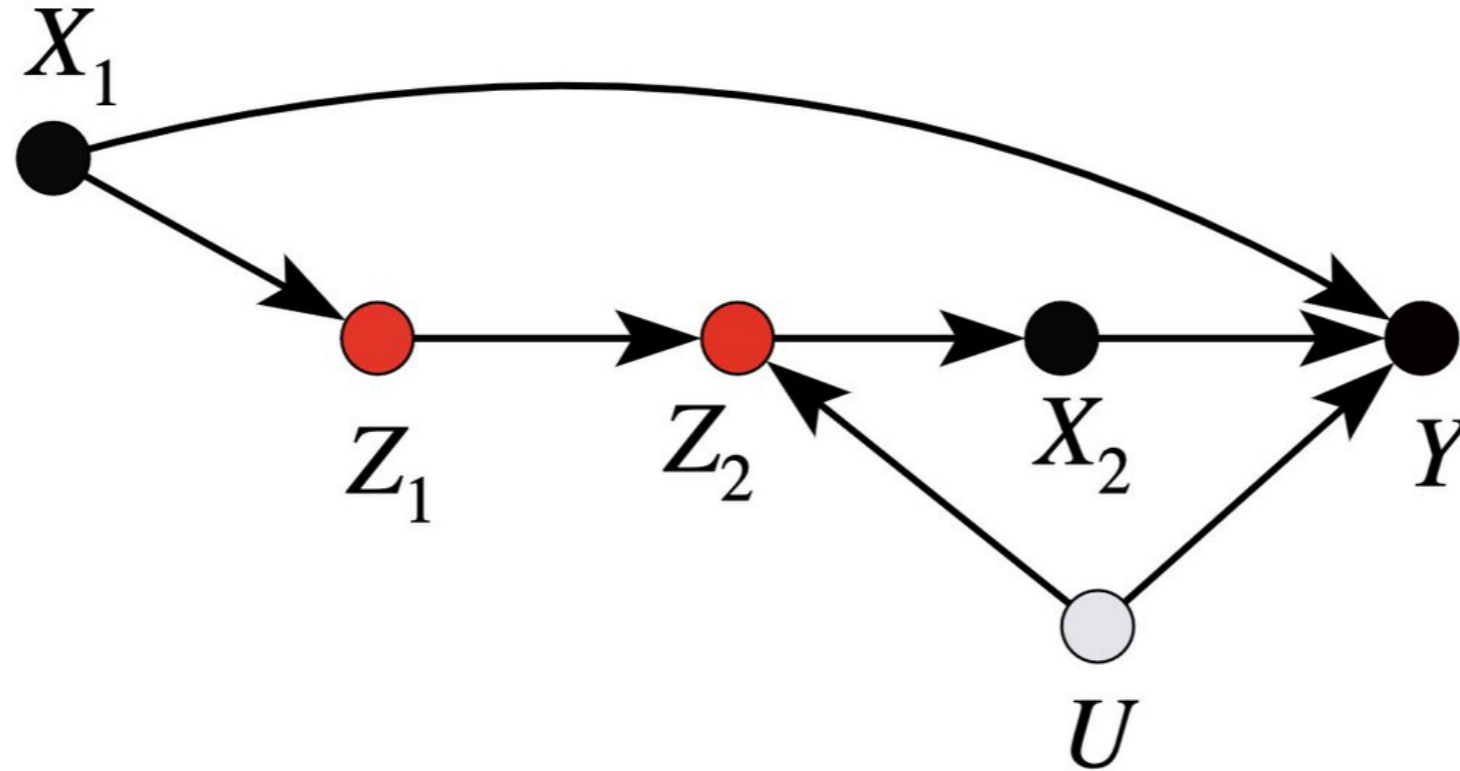
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Answer: include both Z_1 and Z_2 . Note again another example where post-treatment variables are *necessary* for identification.

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In all cases, structural knowledge is indispensable for deciding whether a variable is a good or bad control.

Graphical models provide a natural language for articulating such knowledge, as well as efficient tools for examining its logical ramifications.

Thank you!

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