

# Using Causal Diagrams to Understand Covariate Balance in Propensity Score Models

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# BACKGROUND

The propensity score (PS) is increasingly being used to estimate causal effects in observational studies.

The PS quantifies the probability that a subject receives the exposure or treatment being studied. The PS maps numerous covariates into one score which substitutes for the high-dimensional covariate set in subsequent analysis of the effect of exposure on the outcome.

A central concern in any PS application is achieving covariate balance between exposure groups. Balancing (all) covariates is seen by many as an important yardstick for judging how successful a PS application is.

For high-dimensional covariate set  $\mathbf{Z}$ , treatment  $X$ , and outcome  $Y$ , the PS is estimated as  $e(\mathbf{Z}) = \Pr(X=1|\mathbf{Z})$ .

If  $\mathbf{Z}$  is sufficient to block all biasing (confounding) paths between  $X$  and  $Y$ , and the PS,  $e(\mathbf{Z})$ , is successful in rendering  $X$  and  $\mathbf{Z}$  independent, then  $e(\mathbf{Z})$  should be able to block the biasing paths between  $X$  and  $Y$ .

## OBJECTIVE

This study shows how to use traditional causal diagrams as well as those augmented with the functional mapping encoded in the propensity score to explain covariate balance in propensity score models.

# METHODS

The researcher uses functional mapping augmentation to replace **solid** edges between the exposure and other covariates with **dashed** edges interconnecting the selected covariates ( $Z$ ) with  $e(Z)$  and subsequently  $e(Z)$  with the exposure ( $X$ ):  $Z \rightarrow X$  becomes  $Z \text{---} \rightarrow e(Z) \text{---} \rightarrow X$ .

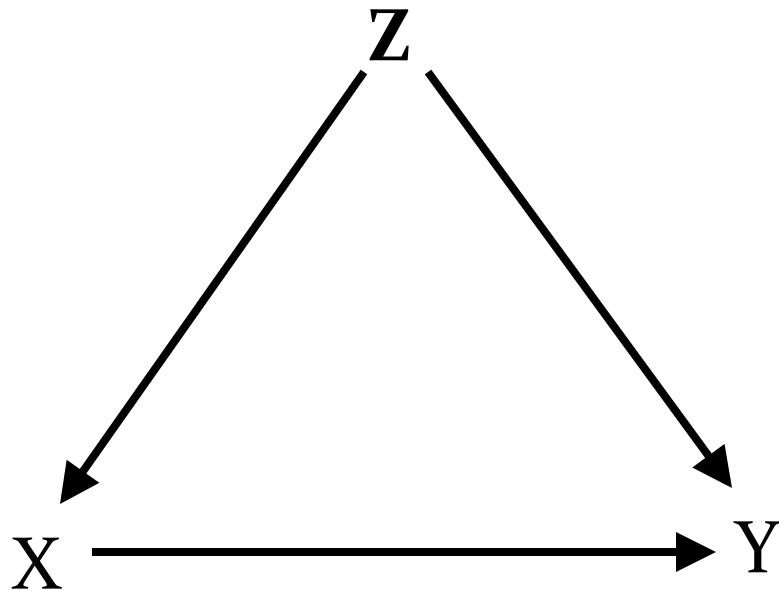
The graphical rules of causal diagrams combined with the conditional independence properties of the propensity score can then be applied to check for covariate balance.

# Simple Confounding

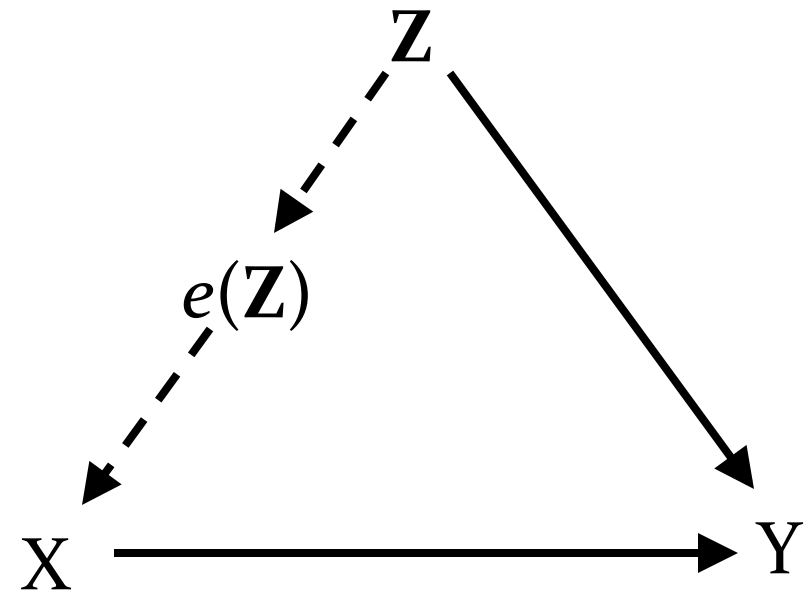
Path  $X\text{-}Z\text{-}Y$  sufficiently blocked by  $Z$ , and if the edge connecting  $Z$  and  $X$  is successfully functionally mapped as  $e(Z)$ , then

- given  $e(Z)$ , the selected covariate  $Z$  will be balanced within  $X$ , and
- $e(Z)$  should block the path  $X\text{-}e(Z)\text{-}Z\text{-}Y$  if PS correctly applied.

DAG augmented with the functional mapping  $e(Z)$



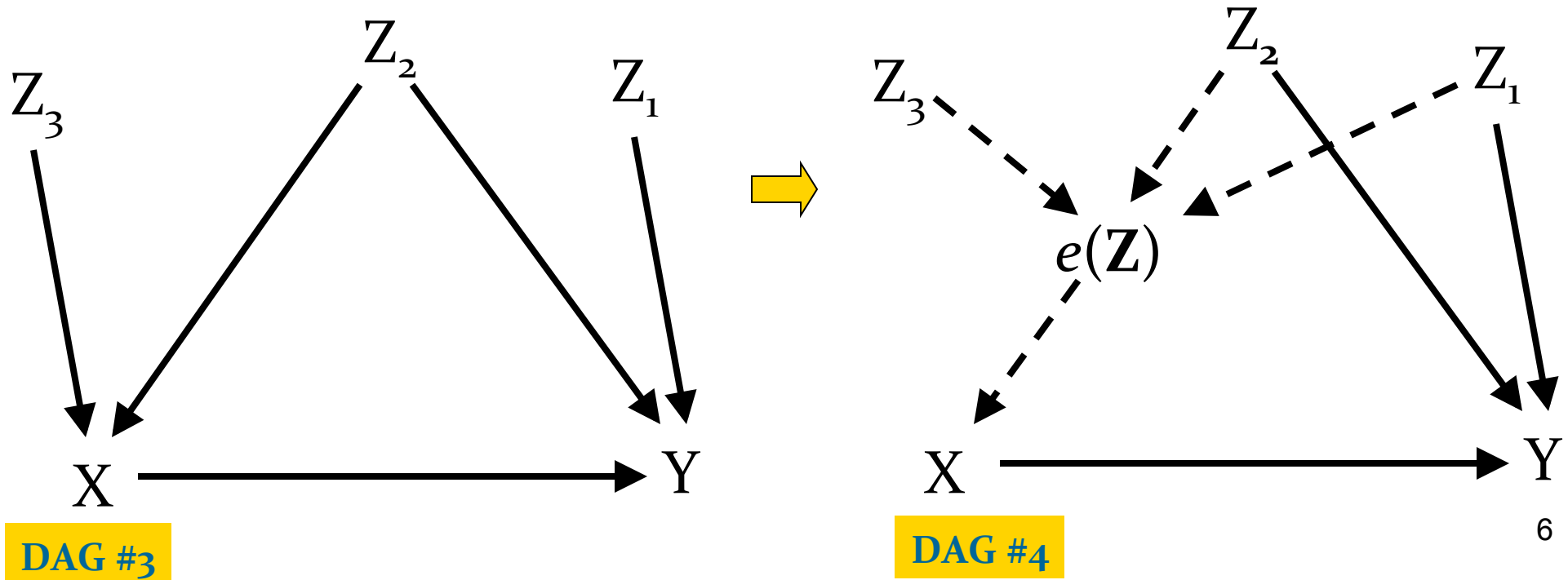
DAG #1



DAG #2

# Confounding variables plus outcome-only and exposure-only predictors

- Prior to applying the PS model (DAG #3), the outcome-only predictor  $Z_1$  set (but not the exposure-only predictor  $Z_3$  or confounding variable  $Z_2$ ) is balanced within  $X$ . Neither  $Z_1$  nor  $Z_3$  is on a biasing path between  $X$  and  $Y$ .
- When all three covariate types are included in the PS model  $e(Z)$ , all three should be balanced within  $X$  given  $e(Z)$ . See DAG#4.

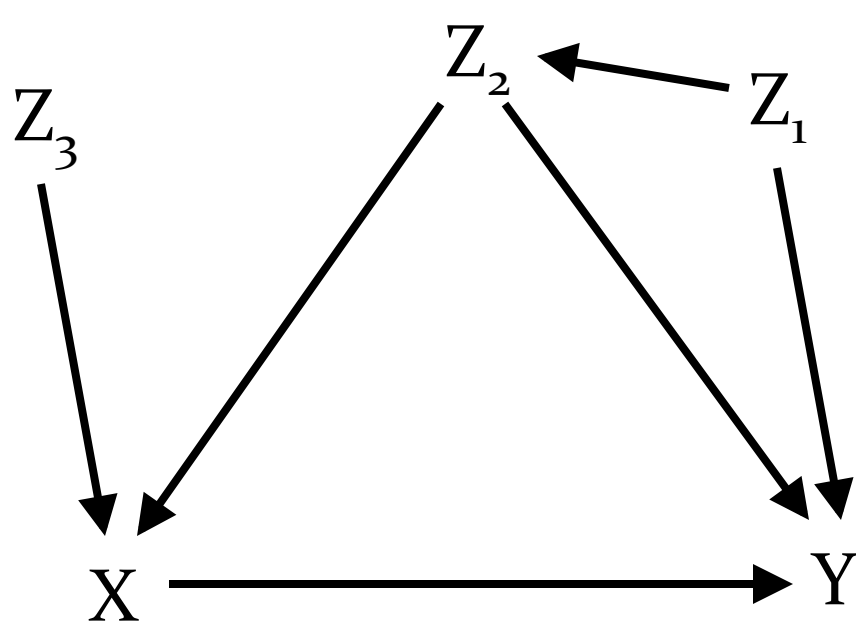


- Potential covariate balance between exposed and unexposed after applying (e.g. matching on) PS

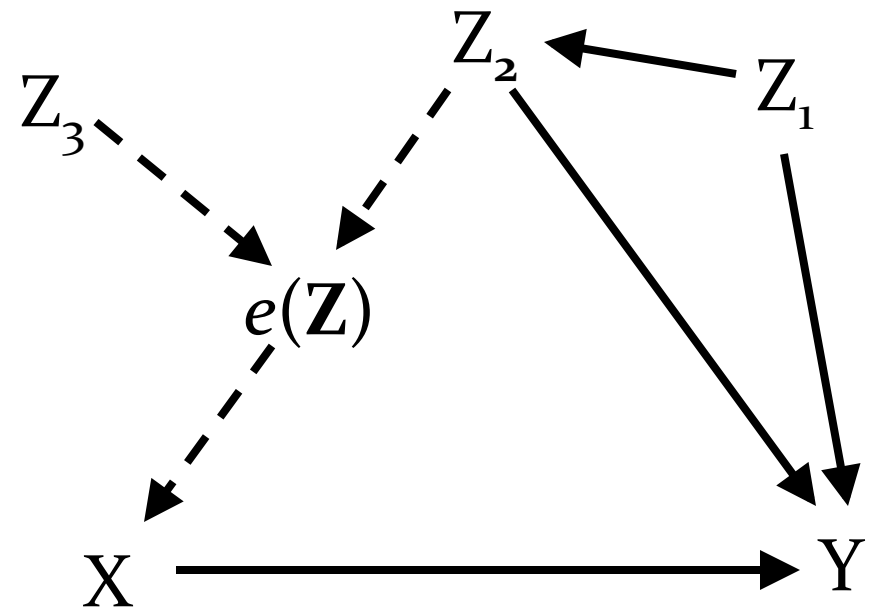
PS model $e(Z)$	Balance of $Z_3$ (variables related only to treatment)	Balance of $Z_2$ (confounders, i.e. variables related to both treatment and outcome)	Balance of $Z_1$ (variables related only to outcome)
$P(X=1 Z_2=z_2)$	No	Yes	Yes
$P(X=1 Z_2=z_2, Z_1=z_1)$	No	Yes	Yes
$P(X=1 Z_2=z_2, Z_3=z_3)$	Yes	Yes	Yes

# Confounding variables plus outcome-confounder and exposure-only predictors

- Prior to applying the PS model (DAG #5), the outcome  $Y$  and the confounder  $Z_2$  share a common cause  $Z_1$  which, without controlling for  $Z_2$ , is not balanced within  $X$ .  $Z_2$  can block the biasing path left open by  $Z_1$  and  $Z_2$  between  $X$  and  $Y$ .
- If  $Z_2$  (but perhaps not  $Z_1$ ) were included in the PS model  $e(\mathbf{Z})$ , then  $Z_1$  can be expected to be balanced within  $X$  given  $e(\mathbf{Z})$ . See DAG#6.



DAG #5

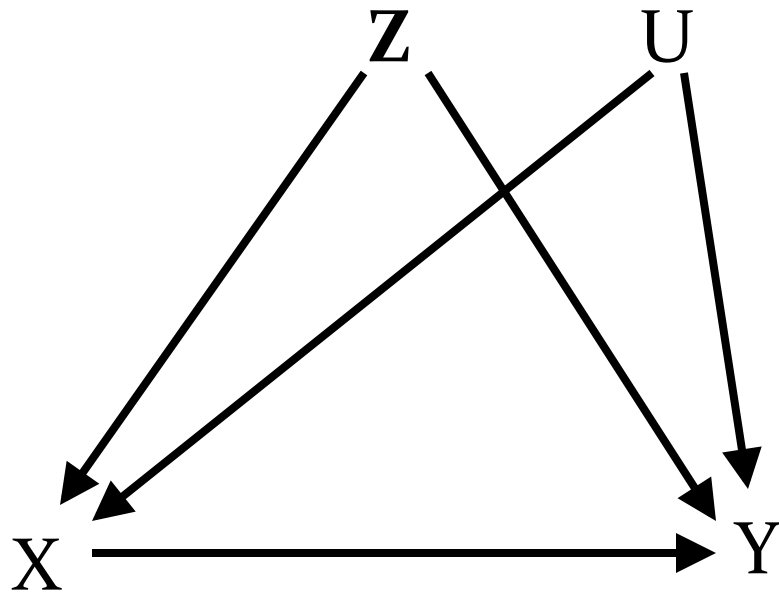


DAG #6

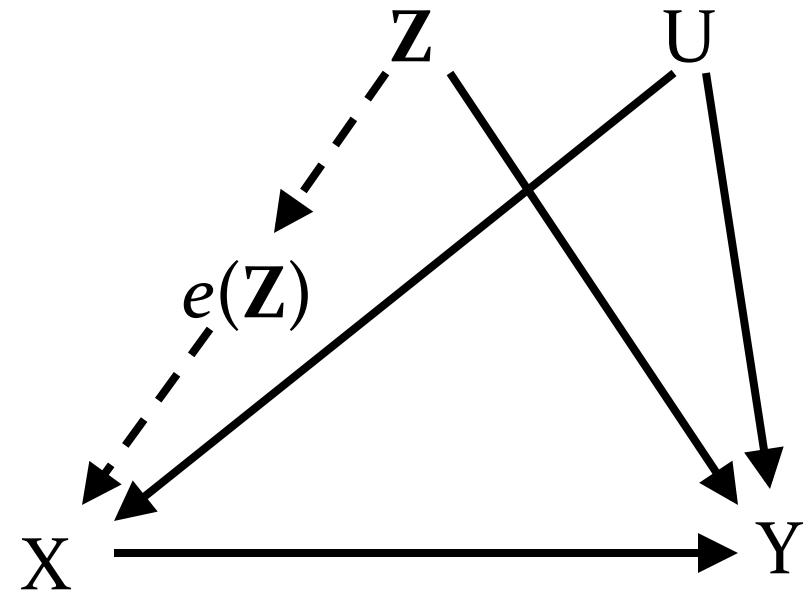


# Uncontrolled Confounding

- Representing uncontrolled confounding is also straightforward: when a covariate  $U$  is unmeasured (DAG#5) and is, therefore, not included in the PS model  $e(\mathbf{Z})$ , the biasing or backdoor path left open by the uncontrolled  $U$  remains open even after applying  $e(\mathbf{Z})$  as would be intuitively expected (DAG #6).



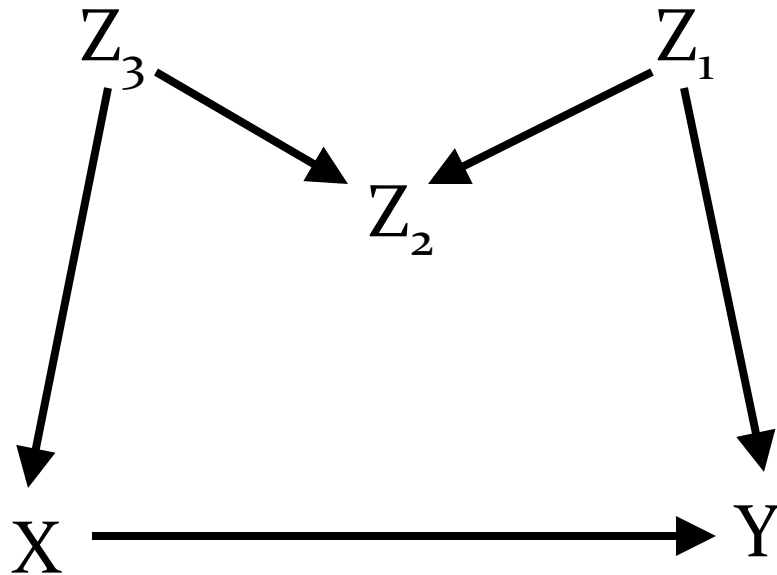
DAG #7



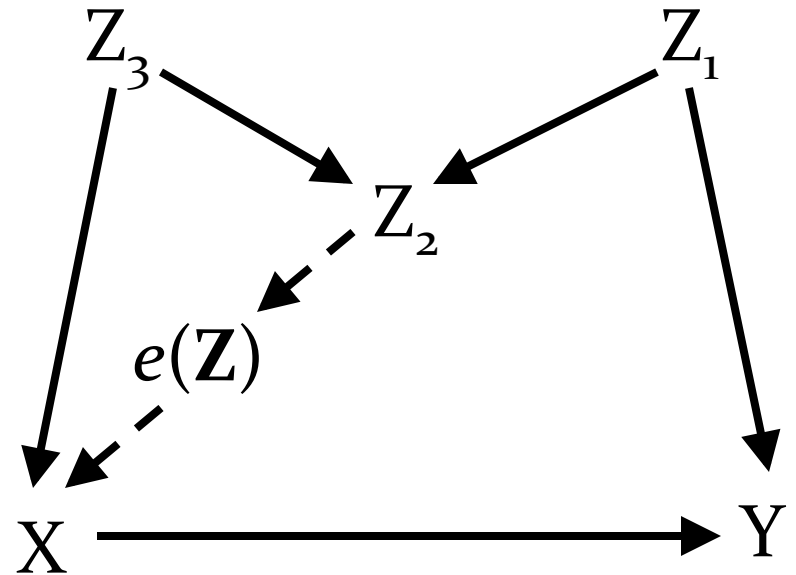
DAG #8

## M-Bias Structure

- It is well-known that adjusting for just the collider  $Z_2$  in DAG #9 opens up a biasing path  $X-Z_1-Z_2-Z_3-Y$  between  $X$  and  $Y$ .
- Now, if the collider  $Z_2$  (but not the covariates  $Z_1$  and  $Z_3$ ) were included in the PS model  $e(\mathbf{Z})$ , then a similar collider bias would be induced in the relationship between  $X$  and  $Y$ . This happens because adjusting for  $e(\mathbf{Z})$  which acts as a “descendant” of  $Z_2$  opens up the colliding path at  $Z_2$ , also leading to marginal dependence between  $Z_1$  and  $X$  (DAG #10).



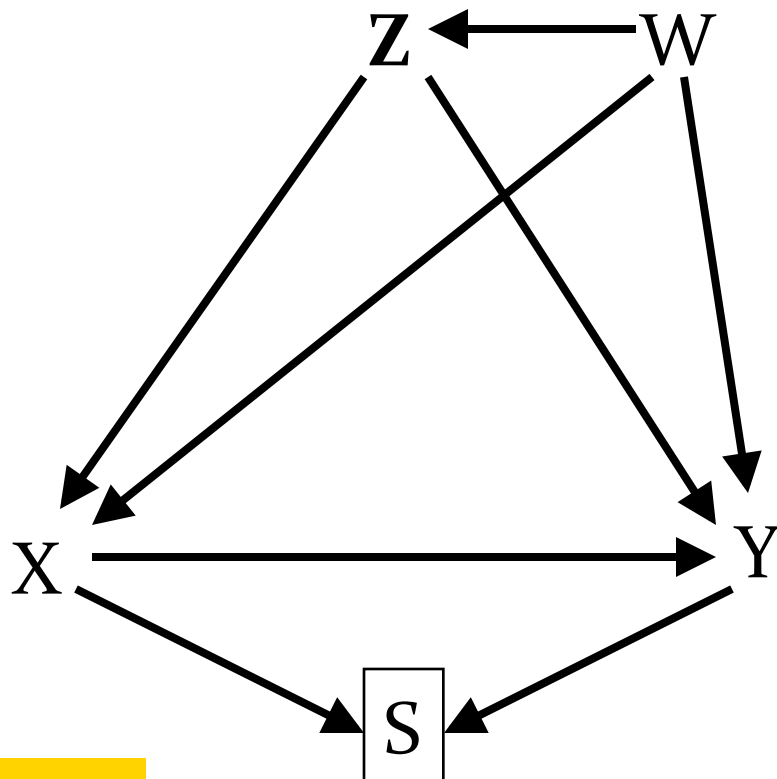
DAG #9



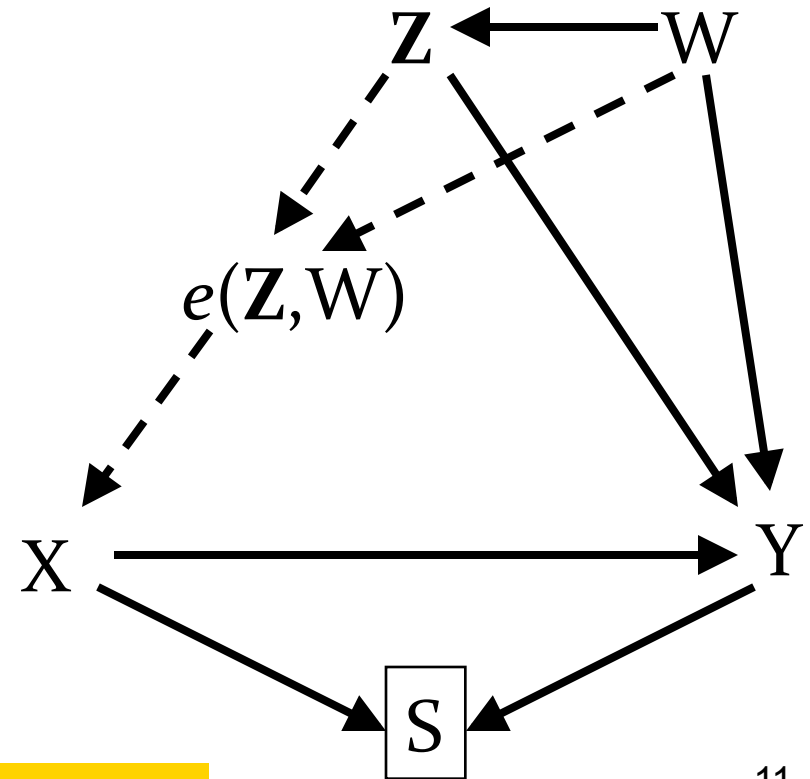
DAG #10

# Selection Bias

- If there is selection bias (from, say, loss to follow-up in cohort study) as depicted in DAG#11, that selection bias will still persist even after applying the PS  $e(Z, W)$  which adjusts for the confounders  $Z$  and  $W$  (DAG #12).



DAG #11



DAG #12

# CONCLUSIONS

Using both traditional and augmented causal diagrams, this study demonstrates how outcome-only predictors are balanced within the exposure whether or not they are included in the propensity score model.

Other types of variables such as confounders and exposure-only predictors must be included in the propensity score model in order to achieve covariate balance between the exposed and unexposed. Nonetheless, only imbalances in covariates which are on confounding paths lead to biased estimates of the causal effect of the exposure. These theoretical results were supported by extensive Monte Carlo simulations.

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