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The Janus face of statistical adjustment: confounders versus colliders

Imre Janszky · Anders Ahlbom · Anna C. Svensson

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Abstract It has long been established that controlling for confounders is essential to delineate the causal relationship between exposure and disease. For this purpose, statistical adjustment is widely used in observational studies. However, many researchers don't acknowledge the potential pitfalls of statistical adjustment. The aim of the present paper was to demonstrate that statistical adjustment is a double edged sword. By using numerically identical examples, we show that adjustment for a common consequence of the exposure and the outcome can lead to as much bias as absence of necessary adjustment for a confounder.

Keywords Confounder · Collider · Bias

In recent decades, there has been an intense theoretical research which has revolutionized our understanding of causal inference [1]. However, little of this research has penetrated into epidemiological practice. One myth about statistical adjustment is that it is a conservative approach, i.e. that it can lead to under—but not overestimation of the magnitude of a causal relationship. Thus, due to lack of enough prior knowledge many researchers prefer to control for every factor they can “to be on the safe side”. However, adjustment for a common consequence of the disease and exposure does not remove, but creates bias. Common consequences of two variables are referred to as colliders in the

modern causal inference literature [2]. For many researchers it is not intuitive why statistical adjustment for colliders induces a bias. The aim of the present paper was to demonstrate and highlight collider bias. By using numerically identical examples to illustrate confounding and collider bias, we will show that adjustment for a collider can lead to as much bias as absence of necessary adjustment for a confounder. The importance of understanding collider bias is further highlighted by the fact that it is also the basis for understanding the recently developed and increasingly popular directed acyclic graphs (DAGs) [2, 3].

Table 1 presents two hypothetical case-control studies with identical numerical results. There are three binary, or dichotomised, variables of interest. E and D represent exposure and disease, respectively. The data are further divided according to a third variable, C. We assume that C is not in the causal pathway between E and D. For simplicity, we neglect the impact of other variables. Numerically the two studies are identical, hence the crude odds ratios (OR_{ED}) for the exposure-disease associations are equal:

$$OR_{ED} = (1500 \times 500) / (1000 \times 1000) = 0.75$$

The OR for the association between E and D when adjusted for C ($OR_{ED \text{ given } C}$) is 0.5. This is also the result in each of the two strata. The adjusted result can be derived from logistic regression but simple stratified analyses also provide the same results (Table 1). Our discussion below applies equally to regression and stratified analyses, as both methods are based on conditioning on C.

Despite the numerically identical results, the interpretation of the crude and the adjusted ORs are the opposite in the two studies.

Study 1. Manual job status (C) is associated with both physical activity at work (E) and heart failure (D). Manual

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Table 1 Identical numerical results of two hypothetical studies

| | C = Yes | | C = No | | All | |
|----------|---------|-----|---------|-----|------|------|
| | Disease | | Disease | | | |
| | Yes | No | Yes | No | Yes | No |
| Exposure | | | | | | |
| Yes | 1200 | 600 | 300 | 400 | 1500 | 1000 |
| No | 400 | 100 | 600 | 400 | 1000 | 500 |
| OR | 0.5 | | 0.5 | | 0.75 | |

In *Study 1*: E, exposure, is physical activity at work, D, disease, is heart failure, covariate C is manual work status (common cause for disease and exposure, i.e. confounder), unbiased result is OR = 0.5. In *Study 2*: E, exposure, is being on diet, D, disease, is heart failure, covariate C is weight loss (common consequence of both the disease and the exposure, i.e. collider), unbiased result is OR = 0.75. It is assumed that C is not in the causal pathway between E and D. The impact of other variables is neglected

status causes the work to be more physically demanding and manual job status is a marker for low socioeconomic status that leads to elevated risk for heart failure. Thus, C is a *common cause* for the exposure and the disease and a confounder. Failure to control for manual job status would lead to an erroneous underestimation of the protective effect of physical activity, i.e. $OR_{ED} = 0.75$ instead of the adjusted $OR_{ED \text{ given } C} = 0.5$. Considerable part of the positive effect of physical activity at work would be masked by the fact that physically active workers are often socially disadvantaged.

Study 2. Weight loss (C) is numerically associated both with being on a diet (E) and with heart failure (D) to the same extent as manual job status is associated with physical activity and heart failure in Study 1 (Table 1). Both heart failure and being on a diet can lead to weight loss. Thus, in this study, C is a *common consequence* of the exposure and the disease. Although it might be tempting to adjust for weight loss, this would clearly be wrong. Sharing a common consequence C does not confound the relation between E and D. To see this, consider an ideal randomized study with very large sample size and perfect compliance. The crude measure of association would show the true causal effect of the exposure on the disease. The consequence of exposure or disease, even if common, could not possibly play any role. If the crude OR correctly reflects the causal association then it follows that the adjusted OR, and the stratum specific ORs, must be biased.

So what happened here? How could adjustment introduce a downward bias that results in an overestimation of the protective effect of diet?

Imagine that only two things cause weight loss: heart failure and diet. If a person not being on a diet loses weight it follows that he/she must have heart failure. The reason is that we know that the weight loss was not due to the only

other cause. As a result, there will be a negative association between diet and heart failure among those who lose weight regardless of whether diet and heart failure are causally related or not.

In the real world, there are many other causes of weight loss. Nevertheless, in the subgroup of those who have lost weight, the association between diet and heart failure will be more negative than in the total population. In the other stratum, comprised by those who did not lose weight, a negative association will also appear. If someone has not lost weight despite being on a diet, then the probability that this person would suffer from another exposure causing weight loss, such as heart failure, is even smaller.

Generally, the bias introduced by adjustment for a common consequence mirrors the bias created by confounding and just as for confounding it can go in either direction depending on how the variables are associated. Study 1 and 2 illustrated how negative confounding corresponds to a bias due to adjusting for a common consequence leading to an overestimation of a true effect. In Table 2, we show that this latter bias can go to the other direction as well, and then it corresponds to a positive confounding. In Study 1', old age is a risk factor for heart failure and prevents physical activity at work. Failure to control for age would result in an overestimation of the true causal effects. In contrast, in Study 2', elevated heart rate is a consequence of heart failure and diet lowers heart rate. Erroneous control for heart rate would lead to an underestimation of the true causal association.

Interestingly, it was discovered a long time ago, and is widely known, that selection of study participants can lead to bias if the selection is influenced by both the disease and the exposure [4]. However, it is not equally intuitive that statistical adjustment leads to the same problem when the

Table 2 Identical numerical results of two hypothetical studies

| | C = Yes | | C = No | | All | |
|----------|---------|------|---------|-------|------|-------|
| | Disease | | Disease | | | |
| | Yes | No | Yes | No | Yes | No |
| Exposure | | | | | | |
| Yes | 1500 | 2000 | 2500 | 10000 | 4000 | 12000 |
| No | 495 | 550 | 105 | 350 | 600 | 900 |
| OR | 0.83 | | 0.83 | | 0.5 | |

In *Study 1'*: E, exposure, is physical activity at work, D, disease, is heart failure, covariate C is old age (common cause for disease and exposure, i.e. confounder), unbiased result is OR = 0.83. In *Study 2'*: E, exposure, is being on diet, D, disease, is heart failure, covariate C is elevated heart rate (common consequence of both the disease and the exposure, i.e. collider), unbiased result is OR = 0.5. It is assumed that C is not in the causal pathway between E and D. The impact of other variables is neglected

Table 3 The summary of the comparison between confounders and colliders

| Confounder | Collider |
|---|---|
| Common cause | Common consequence |
| Causes bias | Does not cause bias |
| Effect of stratification: Removes bias | Effect of stratification: Creates bias |

Only expert knowledge can differentiate colliders from confounders

covariate is a common consequence of the exposure and the disease.

In this paper, collider bias was contrasted with confounding by using numerically identical results from hypothetical studies. In Table 3, we contrast confounders and colliders in relation to statistical adjustment. Confounders cannot be identified empirically but expert knowledge is required for the decision of whether to

consider a covariate a confounder and to include it in a multivariate model or not. Erroneous adjustment for covariates might be just as detrimental as failure to control for proper confounders.

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