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Methods Matter: Beware of collider stratification bias when analyzing recurrent injuries

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MANUSCRIPT

A major objective of sport medicine research is to assess causes of injuries. Studying causes of subsequent injuries, including the effects of rehabilitation programs, presents unique challenges to provide appropriate guidance for treatment. In this commentary, we highlight the issue of "collider stratification bias".

At the 2015 First World Congress in Sports Physical Therapy, Witvrouw[1] presented preliminary findings suggesting that following a 1st injury, strong hamstrings are associated with an increased risk of a 2nd hamstring injury. These results might discourage a clinician from recommending strengthening exercises to a patient as part of their rehabilitation. In actuality, this analysis is subject to "collider stratification bias", leading to a non-causal association between hamstring strength and subsequent injury. The reason for the bias is analogous to the obesity paradox bias whereby obesity (analogous to weakness after 1st injury) causes heart failure (analogous to subsequent injury), but obesity (weakness) appears protective when studies are restricted to only people with heart failure (1st injury).[2,3]

Figure 1 is a simplified causal directed acyclic graph (DAG) for such a study. Variables at the base of the solid arrows are assumed to cause variables at the arrowheads. Consider a surveillance study on risk factors for subsequent injury that follows all athletes from the beginning of the season. To enter the study, participants must have had a previous hamstring injury (box around 1st Hamstring Injury=Yes). We assume that a hamstring injury will cause hamstring weakness post-injury, and will increase the risk of a 2nd hamstring injury through restricted range of motion, etc. We also assume that baseline hamstring weakness is a cause of the 1st hamstring injury. Participants who are stronger at baseline are also assumed to be stronger post-injury. Finally, other causes of the 1st injury aside from weakness (Other Baseline Frailty), are also causes of the 2nd hamstring injury.

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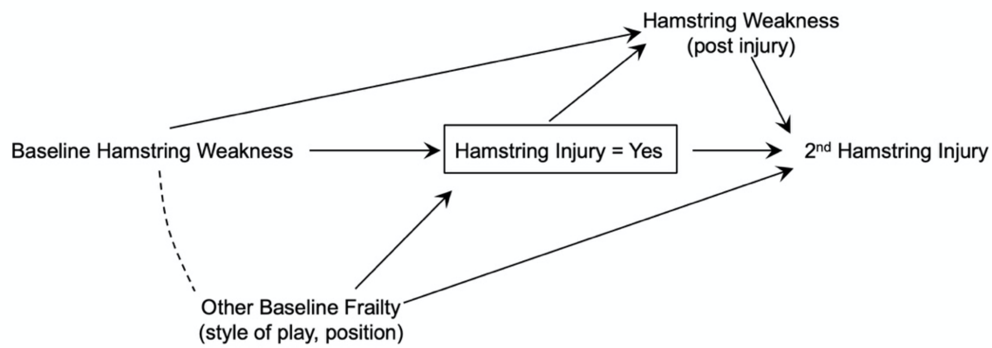

Figure 1: A simplified causal directed acyclic graph (causal DAG) for a study examining time to 2nd injury among participants who had a 1st injury. The box around Hamstring Injury=Yes indicates that we are restricting our study population to those with a hamstring injury. Solid arrows indicate a causal effect between variables. The dashed line indicates that Baseline Hamstring Weakness and Other Baseline Frailties become non-causally associated when one controls for or restricts the population to those with "Hamstring Injury = Yes". Hamstring Injury = Yes is called a collider because it has two arrows point towards it. If this were the true causal DAG, the bias due to the non-causal association could be "blocked" by including accurate measures of either Baseline Hamstring Weakness or Other Baseline Frailty in the regression model,[5 6] or with more advanced methods.[5]

In this causal DAG, the arrows from Baseline Hamstring Weakness and Other Baseline Frailty "collide" (arrowheads meet) on Hamstring Injury = Yes. When we restrict the population based on a collider, or "control" for the collider in a regression analysis, we create a non-causal association between the various causes of the collider (Baseline Hamstring Weakness and Other Baseline Frailty; dashed line in Figure 1). Why?

Consider a simpler example where both rain and automatically timed sprinklers cause grass to be wet.[4] Wet grass is a collider in this situation. An association exists between variables if knowing the value of one variable (rain) provides information about the value of another variable (sprinklers). If you do not know if the grass is wet, there is no association because knowing whether it rained or not does not provide information about the sprinklers. However, if one knew the grass was wet, knowing it did not rain would make it very likely that the sprinklers were on. It is a non-causal association because rain has no effect on automatically timed sprinklers.

From a traditional epidemiology perspective, causal inference requires "exchangeability"; comparison groups must have the same prognosis for the outcome except for the exposure of interest.[5] Participants with strong hamstrings who suffer a hamstring injury must have had another cause for their injury. For instance, they may play positions requiring excessive sprinting (e.g a striker) compared to participants who do not have to sprint (goalies), or have other differences such as an aggressive style of play. Because the participants being compared are not exchangeable, an association between strength after rehabilitation and subsequent injury cannot be interpreted causally.

Once we control/restrict based on Hamstring Injury = Yes, we have the following associations: (1) causal association between Baseline Hamstring Weakness and Hamstring Weakness post injury, (2) non-causal association between Baseline Hamstring Weakness and Other Baseline Frailty, and (3) causal association between Other Baseline Frailty and 2nd Hamstring Injury. This chain of associations (known as back-door path in causal inference) leads to the non-causal (biased) association between Hamstring Weakness post injury and 2nd Hamstring Injury.

Can we estimate the causal effect using traditional regression-based analyses? If the causal DAG in figure 1 is correct, all we need to do is include either Baseline Hamstring Strength or Other Baseline Frailty as a covariate in our regression model. Including either variable blocks the "backdoor path" responsible for the association. The regression model is then estimating the only remaining association between Hamstring Weakness post injury and 2nd Hamstring Injury, which is the causal association directly linking the two variables.

Collider stratification bias is a major concern whenever we restrict populations based on colliders. Yet, restriction of populations is a common strategy in surveillance programs studying subsequent injuries using surveillance programs. Although we could obtain an unbiased estimate using regression in our example, other contexts may require more advanced methods.[5] We strongly encourage investigators to seek out the appropriate statistical expertise when conducting analyses on subsequent injuries.

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REFERENCES

1. Witvrouw E. Return to play in hamstring injuries. First World Congress in Sports Physical Therapy; 2015; Bern, Switzerland.https://www.youtube.com/watch?v=9uvSBRYAA5A
2. Banack HR, Kaufman JS. Does selection bias explain the obesity paradox among individuals with cardiovascular disease? *Ann Epidemiol* 2015;25(5):342-9.
3. Stovitz SD, Banack HR, Kaufman JS. Structural Bias in Studies of Cardiovascular Disease: Let's Not Be Fooled by the "Obesity Paradox". *Can J Cardiol* 2018;34(5):540-542.
4. Pearl J. The art and science of cause and effect. *Causality: models, reasoning and inference*. Cambridge University of Cambridge, 2000:331-358.
5. Hernán MA, Robins JM. Causal inference: What if. Boca Raton: Chapman & Hall/CRC, 2020. 231 pages. https://www.hsph.harvard.edu/miguel-hernan/causal-inference-book/
6. Shrier I, Platt RW. Reducing bias through directed acyclic graphs. *BMC Med Res Methodol* 2008;8:70.