Journal of Communicable Diseases
Volume 58, Issue 1 - 2026, Pg. No. 145-152
Peer Reviewed & Open Access Journal

Review Article

Collider Bias and Infectious Disease Studies: A Review

Pratap Kumar Jena[1], Jugal Kishore[2], Sabyasachi Shukla[3], Debashis Mohapatra[4], Arpita Patel[5]

[1]Associate Professor, School of Public Health, Kalinga Institute of Industrial Technology (KIIT) Deemed to be University, Bhubaneswar, India
[2]Director-Professor, Department of Community Medicine, Vardhman Mahavir Medical College & Safdarjung Hospital, New Delhi, India
[3,4]PhD Scholar, School of Public Health, Kalinga Institute of Industrial Technology (KIIT) Deemed to be University, Bhubaneswar, India
[5]Department of Community Medicine, Kalinga Institute of Medical Sciences, Bhubaneswar, India
DOI: https://doi.org/10.24321/0019.5138.202619

I N F O

Corresponding Author:
Pratap Kumar Jena, School of Public Health, Kalinga Institute of Industrial Technology (KIIT) Deemed to be University, Bhubaneswar, India
E-mail Id:
drpratapjena@gmail.com
Orcid Id:
https://orcid.org/0000-0002-1236-3103
How to cite this article:
Jena P K, Kishore J, Shukla S, Mohapatra D, Patel A. Collider Bias and Infectious Disease Studies: A Review. J Commun Dis. 2026;58(1):145-152.

Date of Submission: 2025-11-07
Date of Acceptance: 2026-03-15

A B S T R A C T

Collider bias is the distortion of the observed association between an exposure and an outcome that occurs when analysis conditions (by design or adjustment) on a variable that is a common effect of the exposure and outcome, or their causes, thereby opening a noncausal pathway that induces a spurious association or alters effect estimates. This review explains the mechanism, distinguishes collider bias from confounding and broader selection bias, and illustrates its impact in infectious disease research, particularly in the context of COVID-19, HIV, tuberculosis (TB), and malaria. During the COVID-19 pandemic, for example, studies restricted to hospitalised patients suggested smoking might protect against severe disease, an artefact later understood as collider bias. Similar issues have emerged in HIV and TB research, where restricting analyses to patients in care or clinical trials created misleading associations, and in malaria studies, where hospital-based sampling distorted genetic findings. We conducted a structured narrative review of the literature published in the past decade, identifying methodological and applied papers that described collider bias in infectious disease contexts. Key findings show that collider bias can produce false protective effects, reverse expected associations, and either exaggerate or attenuate true effects. Practical solutions include the use of directed acyclic graphs (DAGs) to identify colliders, application of inverse probability weighting, sensitivity analyses, and reweighting with external population data. The broader implications extend beyond methodology: collider bias can shape public perception, misinform clinical practice, and obscure health disparities. Recognising and addressing collider bias is therefore essential for generating accurate, equitable, and actionable evidence in infectious disease research.

Keywords: Collider Bias, Infectious Diseases, HIV/TB; COVID-19, Epidemiology, Causal Inference

Introduction

Infectious disease research often relies on observational data collected under urgent and challenging conditions. While these studies provide invaluable insights, they are also prone to subtle biases that can distort findings. One such bias, collider bias, has gained increasing recognition in epidemiology over the past decade. Collider bias occurs when an exposure and outcome (or factors that cause these) each influence a common downstream variable, and that variable is controlled by design (e.g., restriction, selection) or through statistical adjustment, which creates a noncausal pathway and induces a spurious association. This creates associations that do not exist in the population and can lead to incorrect conclusions.[1,2]

In the context of infectious diseases, collider bias frequently arises in hospital- or clinic- based studies, where inclusion is inherently tied to factors such as hospitalization, diagnostic testing, or survival outcomes. For example, early in the COVID-19 pandemic, analyses restricted to hospitalised patients suggested that smoking might be protective against severe COVID-19.[3] This was later explained as an artefact of collider bias, since both smoking status and severity of disease influenced the likelihood of hospitalization.[4] Restricting analyses to hospitalised patients effectively "opened a backdoor" correlation between smoking and disease outcomes that did not exist in the general population. A schematic illustration of this collider structure is presented in Figure 1.

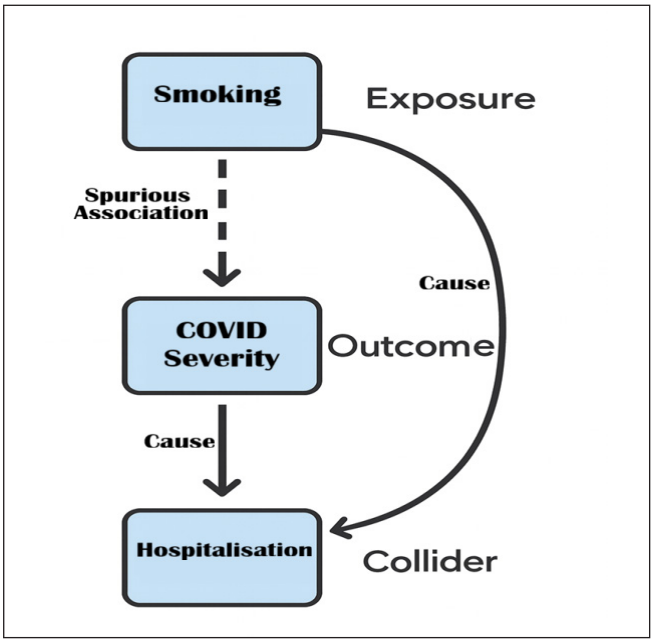

Figure 1.Illustration of Collider Bias in the Association Between Smoking and COVID-19 Severity
Source: Adapted from Holmberg & Andersen[1]

Both smoking status and the severity of COVID-19 influence whether a person is hospitalised. When researchers include only hospitalised patients in their analysis, they are effectively conditioning on a variable, hospitalization, that is affected by both factors. This conditioning creates what is known as collider bias. In this situation, hospitalisation acts as a "collider" because it is an outcome of both smoking and disease severity. Analysing only those who are hospitalised, we unintentionally introduce a statistical link between smoking and severity, even if no causal relationship exists in the general population. As a result, smoking may appear to protect against severe COVID-19, not because it truly does, but because smokers with mild disease are under-represented among non-hospitalised individuals, distorting the observed relationship.

Critically, individuals who experienced mild or asymptomatic COVID-19 and never presented for hospital care are entirely absent from such analyses. Because this non-hospitalised group is disproportionately composed of non-smokers with mild disease, their systematic exclusion compounds the collider effect and further amplifies the spurious protective association between smoking and severe disease - a form of selection bias formally described by Westreich,[5] and Hernan et al.[6]

Similar issues have long been present in other areas of infectious disease epidemiology. In HIV research, conditioning on clinic-based samples can make behavioural factors appear associated with treatment outcomes when they may reflect who presents for care.[7] In tuberculosis (TB), collider bias can occur when both HIV status and genetic susceptibility influence who progresses from latent infection to active disease and is subsequently enrolled in treatment trials.[8,9] Because participation in such trials is limited to individuals with active TB, this selection process conditions the analysis on a variable, trial enrolment, that depends on both HIV status and host genetics. As a result, HIV status and genetic susceptibility, which may be unrelated in the general population, appear correlated within the selected trial group. This induced association can distort estimates of treatment response or disease progression, giving the false impression that certain genetic profiles modify the effect of HIV on TB outcomes when, in reality, the relationship is created by conditioning on who was eligible for the study. In malaria, restricting analysis to diagnosed cases seen in hospitals may create spurious links between resistance genes and socioeconomic status, even if no such relationship exists in the wider community.[5]

These examples highlight the importance of carefully considering colliders when designing, analysing, and interpreting studies. Directed acyclic graphs (DAGs) are a

ISSN: 0019-5138
DOI: https://doi.org/10.24321/0019.5138.202619

powerful tool for visualising these structures. In a DAG, a collider is represented as a node with at least two arrows pointing into it. Conditioning on the collider opens a pathway that connects variables that were otherwise independent.[9,10] This graphical approach facilitates the identification of potential sources of collider bias prior to conducting an analysis.

The consequences of collider bias are not trivial. Misleading associations can shape clinical guidance, influence public health messaging, and even alter policy decisions. The so-called "obesity paradox," where obesity appeared to protect against worse infectious disease outcomes, has been partly attributed to collider bias in hospitalised samples.[11] Similarly, the smoking-COVID narrative gained traction early in the pandemic, despite being based on flawed evidence.[4] In both cases, the risk of public misunderstanding and policy misdirection was substantial.

To move forward, infectious disease research must incorporate strategies to avoid or address collider bias. These include resisting the reflex to adjust for every available variable, using DAGs to identify colliders, and applying corrective methods like inverse probability weighting or sensitivity analyses when conditioning is unavoidable.[12] External population data can also be used to reweight hospital-based samples, improving representativeness.[13] Transparency in reporting, as recommended in guidelines like STROBE, ensures that readers can assess the potential for collider bias in published studies.[14]

Infectious disease epidemiology is especially vulnerable to collider bias because it is particularly susceptible to this bias due to the numerous studies conducted in urgent clinical settings, where rapid answers are required. As the COVID-19 experience has shown, the consequences of overlooking collider bias can be profound. This review examines how collider bias emerges in infectious disease research, its implications for inference, and strategies for mitigating its effects. Bringing these issues to the forefront aims to strengthen the reliability of future research in global health.

Methods

We conducted a structured narrative review to summarise how collider bias has been described and addressed in infectious disease research.

- **Search strategy:** PubMed, a popular database for medical science researchers was searched for peer-reviewed articles containing the phrase "collider bias" in the title or abstract, limited to publications from January 2014 to June 2024. Searches were restricted to human studies published in the English language. To ensure completeness, we manually reviewed the reference lists of included papers and relevant methodological reviews.
- **Inclusion and exclusion criteria:** Studies were included if they (i) explicitly discussed collider bias, (ii) related to infectious disease research or employed methods applicable to infectious disease contexts, and (iii) provided empirical examples or methodological insights. We excluded (i) studies unrelated to infectious diseases, (ii) papers that mentioned collider bias only tangentially, and (iii) unreviewed conference abstracts or preprints.
- **Screening and data extraction:** Two reviewers independently screened titles and abstracts, followed by full-text review of eligible studies. Data were extracted using a structured form that captured the study design (theoretical, methodological, or applied), disease context (COVID-19, HIV, TB, malaria, or others), examples of collider bias, and mitigation strategies. Discrepancies were resolved through discussion.
- **Synthesis:** Given the conceptual and methodological heterogeneity among studies, findings were synthesised thematically rather than quantitatively. The review followed PRISMA guidelines, where applicable, to reviews, ensuring transparency in the search, selection, and synthesis processes.

Results

Our review yielded 56 articles with "Collider Bias" in the title over the past decade. After applying the eligibility criteria, 12 articles including methodological papers, and applied contagious disease studies, as reported in the Table 1.

Contexts of Collider Bias

COVID-19 dominated the applied literature. Several studies described paradoxical findings, such as smoking or obesity appearing protective against severe COVID outcomes.[3,4,15–17] These paradoxes were linked to collider bias caused by conditioning on hospitalisation or diagnostic testing.[3,15]

In HIV research, collider bias often appeared when analyses were restricted to patients already in care or clinical trials. This conditioning created misleading associations between behavioural risk factors and outcomes.[7] TB studies highlighted how HIV status and genetic susceptibility both affected progression to active TB, biasing studies of enrolled cohorts.[8] In malaria, hospital-based genetic studies produced spurious associations between resistance genes and socioeconomic status due to conditioning on diagnosed cases.[18]

ISSN: 0019-5138
DOI: https://doi.org/10.24321/0019.5138.202619

Table 1.Collider Bias in Infectious Disease Research as reported in the PubMed indexed articles during past decade

Author (Year)	Disease Context	Collider Bias Mechanism
Tattan-Birch H et al., (2021)[3]	COVID-19, Smoking	People who cough are more likely to get tested for COVID-19, and since both smoking and COVID-19 can cause coughing, smokers may get tested even when they don't have the virus. Because only tested people are included, smokers appear more often among those who test negative, falsely suggesting that smoking protects against COVID-19.
Taheri Soodejani et al. (2023)[15]	Covid-19	Because both vaccination status and factors like age and comorbidities influence who gets hospitalized, studying only hospitalized patients creates collider bias. This can hide the true protective effect of vaccination on death, making it appear weaker or absent unless key factors like age and underlying diseases are properly adjusted for
Lee et al. (2022)[19]	Sepsis	In sepsis research, studying only hospitalized or ICU patients can introduce collider bias because both the exposure (e.g., a risk factor or treatment) and the outcome (such as severity or death) influence whether a patient is included in the sample. As a result, restricting analyses to these selected patients can create misleading associations that may hide or even reverse the true effect seen in the broader population.
Leite et al. (2020) [20]	Periodontitis	When studies include only specific groups (e.g., those who attend dental clinics or remain in long-term follow-up), because both the exposure (such as risk factors or behaviors) and the outcome (oral health status) influence who is included in the sample. As a result, this selection process can distort associations, sometimes creating or masking relationships, especially in longitudinal studies where loss to follow-up or selective participation acts as a collider.
Griffith et al. (2020) [4]	COVID-19	Many COVID-19 studies use non-representative samples (such as only tested, hospitalized, or volunteer participants), where both risk factors and disease outcomes influence who gets included, creating collider bias. This selection process can distort or even create false associations between risk factors and COVID-19 outcomes, meaning observed relationships in the sample may not reflect the true relationships in the general population
Watson et al. (2019) [18]	Malaria	The study shows that an apparent protective effect of G6PD deficiency against cerebral malaria is actually due to collider bias created by how cases were defined and selected. By excluding patients who had both severe malarial anaemia and cerebral malaria, the analysis artificially induced a negative association, making G6PD deficiency seem protective even though this effect can be fully explained by selection bias rather than a true causal relationship
Frickmann et al. (2024)[21]	Infectious GI disorder	In this study, collider bias was explored by examining how multiple microorganisms contribute to the same condition (chronic gastroenteric disorder), where the disease itself acts as a collider influenced by different pathogens. The results showed that negative associations (expected under collider bias) were rare, and instead many pathogens co-occurred, suggesting that the disease is driven by complex interactions of multiple microbes rather than a single causal pathogen, making simple causal conclusions difficult

ISSN: 0019-5138
DOI: https://doi.org/10.24321/0019.5138.202619

Learoyd et al. (2023) [16]	COVID-19	The study shows that analyzing only hospitalized COVID-19 patients can introduce collider bias because hospitalization is influenced by both ethnicity (exposure) and disease severity or mortality (outcome), creating artificial associations between them. As a result, observed ethnic differences in outcomes may be distorted, and adjusting for this selection bias can reduce or change these apparent disparities, giving a more accurate estimate of the true relationship
Herbert et al. (2020) [17]	COVID-19	The article explains that collider bias occurs when researchers focus on a selected group (such as hospitalized patients) where two unrelated conditions both influence inclusion in the sample. This selection can create a false association between those conditions, making them appear related in the selected group even though no real relationship exists in the general population.
Learoyd et al. (2024) [14]	COVID-19	The study explains that many COVID-19 analyses focus only on hospitalized (severe) patients, where both the exposure (e.g., ethnicity or other risk factors) and the outcome (death) influence hospital admission, creating collider bias. This selection can produce misleading associations, such as apparently lower risk in some groups, which can be corrected using methods like inverse probability weighting with external data to better reflect the true population relationships.
Zhang et al. (2025) [22]	COVID-19, Air pollution	In the context of that study, air pollution is treated as an exposure that may affect COVID-19 outcomes (such as severity or death), but analyses restricted to hospitalized patients can introduce collider bias because both air pollution exposure and disease severity influence hospitalization. As a result, the relationship between air pollution and outcomes may appear weaker, stronger, or even reversed unless the selection bias is properly adjusted for.
Raittio (2021)[23]	Cardiovascular/ Oral infections	Factors like patient characteristics or risk factors (exposures) and treatment outcomes (outcomes) can both influence whether a tooth with oral infection is included in a study (e.g., only treated cases), so restricting analysis to these infected/treated cases can introduce collider bias and distort the true relationships.

Study design and Collider Bias

Holmberg and Andersen (2022) described how collider bias can occur in both observational studies and randomized clinical trials.[1] In clinical trials, they highlighted that differential loss to follow-up may act as a collider when both the intervention and outcome influence who remains in the study, potentially creating false associations. In observational studies, it has been explained that restricting analyses to individuals with a particular condition, such as COVID-19, can similarly induce spurious relationships between exposure and outcome.[15] Nevertheless, collider bias can occur in any study design.

Consequences of Collider Bias

Three key consequences were consistently reported: (i) spurious protective or harmful associations, (ii) reversal of expected relationships (e.g., the obesity paradox), and (iii) exaggeration or attenuation of true effects. These consequences were observed in vaccine effectiveness studies using test-negative designs, where conditioning on testing resulted in biased estimates.[22,24]

Restriction to control confounding as a source of collider bias

Restrictions intended to reduce confounding (e.g., limiting analyses to hospitalized cases or test-positive individuals) can inadvertently condition on colliders and induce bias, potentially altering or even reversing exposure-outcome associations.[1,4] In COVID-19 research, case- or hospital-based designs often make exposures like smoking or obesity appear protective because hospitalization or testing status functions as a collider between exposure and outcome determinants.[4] Directed acyclic graphs (DAGs) are critical for distinguishing true confounders from colliders before applying restrictions or adjustments, as conditioning on colliders opens noncausal pathways.[1,10] When such

ISSN: 0019-5138
DOI: https://doi.org/10.24321/0019.5138.202619

restrictions are unavoidable, inverse probability weighting, reweighting, or sensitivity analyses can help mitigate, but not fully eliminate, the induced bias. Analogous bias can arise in randomized trials when analyses are restricted to participants retained in follow-up, since retention itself may become a post-randomization collider.[1,12]

Mitigation Strategies

Mitigation strategies emphasised prevention and correction. DAGs were widely recommended for identifying colliders during study design.[9,10] Inverse probability weighting and sensitivity analyses were applied when conditioning could not be avoided.[12,13] Population-based data were used to correct biases in hospital cohorts.[14,15] Transparency in reporting conditioning and selection processes was consistently emphasised.[14,17]

Discussion

Collider bias is not a rare technical nuisance but a significant threat to the validity of infectious disease research. This review highlights how easily collider bias can arise, particularly in hospital-based studies, test-negative designs, and genetic epidemiology. Its consequences, false associations, paradoxical findings, and distorted effect sizes pose risks not only to science but also to clinical practice and policy.

The COVID-19 pandemic exposed how collider bias can distort evidence and influence public perception.[17] Early findings suggesting that smoking protected against severe COVID-19 were later disproven as artefacts of conditioning on hospitalization, yet the narrative persisted. Similar issues have arisen in HIV, TB, and malaria studies, where analyses limited to clinical or diagnostic cohorts created spurious associations that misinformed treatment and prevention strategies.

The path forward involves shifting from a culture of automatic adjustment to one of careful causal reasoning. More adjustment is not always better; in fact, adjusting for colliders can make estimates worse. DAGs and causal inference principles should become standard tools in infectious disease research training. Embedding these frameworks into curricula will equip future researchers more effectively to recognise and avoid collider bias.

Practical mitigation also requires methodological tools. Inverse probability weighting, external reweighting with population data, and sensitivity analyses are all promising strategies. Simulations can also be used to estimate the potential magnitude of collider bias under plausible scenarios, offering transparency to readers and policymakers.

Addressing collider bias requires strategies applied at two stages: prevention during study design and mitigation during analysis. At the design stage, the most powerful tool is the use of directed acyclic graphs (DAGs) to map the causal relationships among all relevant variables before data collection begins, enabling researchers to identify colliders and decide whether conditioning on them is necessary.[10,25] Wherever possible, researchers should prefer population-based or randomly sampled study designs over hospital- or clinic-restricted samples, since such sampling eliminates the selection mechanism that creates colliders.[5] When conditioning on a collider is unavoidable by design (for example, in clinical trials or hospital cohorts), several analytical remedies exist. Inverse probability weighting (IPW) re-weights individuals in the selected sample by the inverse of their probability of selection, effectively reconstructing a sample representative of the target population.[12] External reweighting using population registers or surveillance data achieves the same goal by anchoring the hospital sample to known population distributions, as demonstrated by Learoyd et al. in a COVID-19 cohort.[16] Negative control analyses, including negative control exposures and outcomes, can reveal the presence of bias by detecting associations that should not exist under a correctly specified causal model.[24]

There are also broader implications. Collider bias intersects with equity: who gets hospitalised, tested, or included in studies is shaped by structural determinants such as socioeconomic status and race. Raittio et al., highlight collider bias in oral-systemic disease studies, where selection into clinical cohorts distorts exposure-outcome relationships influenced by social factors, mirroring patterns in infectious disease epidemiology.[9] If collider bias interacts with these factors, health disparities can be exaggerated or obscured, leading to misinformed policy. Recognising collider bias as not only a methodological issue but also an ethical concern is critical for public health.

Finally, interdisciplinary collaboration is vital. Infectious disease research increasingly depends on large datasets and rapid analyses. Statisticians, epidemiologists, clinicians, and data scientists must work together to anticipate, detect, and address collider bias. Only through such collaboration can we ensure that methodological warnings are translated into real improvements in applied research.

In summary, collider bias is a central challenge in infectious disease epidemiology. Learning from the lessons of COVID-19 and integrating causal inference frameworks into research culture can help the field move toward more reliable, equitable, and actionable evidence. Addressing collider bias by use of DAGs, careful avoidance of conditioning on colliders, inverse probability weighting, sensitivity analyses, use of population-based or representative data, appropriate adjustment for confounders, transparency in reporting selection and conditioning processes is not just a technical

ISSN: 0019-5138
DOI: https://doi.org/10.24321/0019.5138.202619

refinement; it is essential to safeguarding the accuracy and trustworthiness of infectious disease science.

Authors' Contribution Statement: All authors contributed equally to the conception, design, literature review, analysis, drafting, and critical revision of the manuscript. Each author approves the final version for submission and agrees to be accountable for all aspects of the work.

Source of Funding: None

Conflict of Interest: None

References

1. Holmberg MJ, Andersen LW. Collider Bias. JAMA. 2022 Apr 5;327(13):1282–3. doi:10.1001/jama.2022.1820.
2. Mansournia MA, Nazemipour M, Etminan M. Interaction Contrasts and Collider Bias. Am J Epidemiol. 2022 Sep 28;191(10):1813–9. doi:10.1093/aje/kwac103.
3. Tattan-Birch H, Marsden J, West R, Gage SH. Assessing and addressing collider bias in addiction research: the curious case of smoking and COVID-19. Addiction. 2021 May;116(5):982–4. doi:10.1111/add.15348.
4. Griffith GJ, Morris TT, Tudball MJ, Herbert A, Mancano G, Pike L, Sharp GC, Sterne J, Palmer TM, Davey Smith G, Tilling K, Zuccolo L, Davies NM, Hemani G. Collider bias undermines our understanding of COVID-19 disease risk and severity. Nat Commun. 2020 Nov 12;11(1):5749. doi:10.1038/s41467-020-19478-2
5. Westreich D. Berkson's bias, selection bias, and missing data. Epidemiology. 2012 Jan;23(1):159–64. doi:10.1097/EDE.0b013e31823b6296
6. Hernan MA, Robins JM. Causal Inference: What If. Boca Raton (FL): Chapman & Hall/CRC; 2020.
7. Ma Z, Chen Q, He J, et al. The causal relationship between HIV-1 infection and the spectrum of Coronavirus disease 2019: a Mendelian randomisation study. Research Square; 2022. DOI: 10.21203/rs.3.rs-1483723/v1.
8. Rodriguez CA, Lodi S, Horsburgh CR, et al. Selection bias in multidrug-resistant tuberculosis cohort studies assessing sputum culture conversion. PLoS One. 2022; 17(11):e0276457. doi:10.1371/journal.pone.0276457.
9. Greenland S, Pearl J, Robins JM. Causal diagrams for epidemiologic research. Epidemiology. 1999 Jan;10(1):37–48.
10. 10. M. M. Glymour and S. Greenland, "Causal Diagrams," In K. J. Rothman, S. Greenland and T. L. Lash, Eds., Modern Epidemiology, 3rd Edition, Lippincott Williams and Wilkins, Philadelphia, 2008, pp. 183-209.
11. Pearl J. *Causality: Models, Reasoning, and Inference*. 2nd ed. Cambridge: Cambridge University Press; 2009.
12. Banack HR, Kaufman JS. The obesity paradox: understanding the effect of obesity on mortality among individuals with cardiovascular disease. Prev Med. 2014 May;62:96–102. doi:10.1016/j.ypmed.2014.02.003.
13. Cole SR, Hernán MA. Constructing inverse probability weights for marginal structural models. Am J Epidemiol. 2008 Sep 15;168(6):656–64. doi:10.1093/aje/kwn164.
14. Learoyd A, Nicholas J, Hart N, Douiri A. Application of information from external data to correct for collider bias in a Covid-19 hospitalised cohort. BMC Med Res Methodol. 2024 Jul 16;24(1):149. doi:10.1186/s12874-023-02129-7.
15. Taheri Soodejani M, Tabatabaei SM, Lotfi MH, Nazemipour M, Mansournia MA. Adjustment for collider bias in the hospitalized Covid-19 setting. Glob Epidemiol. 2023 Dec;6:100120. doi:10.1016/j.gloepi.2023.100120.
16. Learoyd AE, Nicholas J, Hart N, Douiri A. Revisiting ethnic discrepancies in COVID-19 hospitalized cohorts: a correction for collider bias. Journal of Clinical Epidemiology. 2023 Sep 1;161:94–103. doi:10.1016/j.jclinepi.2023.06.014
17. Herbert A, Griffith G, Hemani G, Zuccolo L. The Spectre of Berkson's Paradox: Collider Bias in Covid-19 Research. Signif (Oxf). 2020 Jul 29;17(4):6–7. doi:10.1111/1740-9713.01413.
18. Watson JA, Leopold SJ, Simpson JA, Day NP, Dondorp AM, White NJ. Collider bias and the apparent protective effect of glucose-6-phosphate dehydrogenase deficiency on cerebral malaria. Elife. 2019;8:e43154. doi:10.7554/eLife.
19. Lee CC, Ho MP, Huang AH, Tan J, Yo CH, Hsu WT. Collider Bias Rather Than a Healthy Condition Leads to the Unfavorable Outcome of Sepsis. Chest. 2022;162(1):e63–4. doi:10.1016/j.chest.2022.02.046.
20. Leite FRM, Nascimento GG, Peres KG, Demarco FF, Horta BL, Peres MA. Collider bias in the association of periodontitis and carotid intima-media thickness. Community Dent Oral Epidemiol. 2020 Aug;48(4):264–70. doi:10.1111/cdoe.12525.
21. Frickmann H, Backhaus J, Hoerauf A, et al. Collider Bias Assessment in Colombian Indigenous Wiwa and Kogui Populations with Chronic Gastroenteric Disorder of Likely Infectious Etiology Suggests Complex Microbial Interactions Rather Than Clear Assignments of Etiological Relevance. Microorganisms. 2024;12(5):970. doi:10.3390/microorganisms12050970.
22. Zhang J, Andersen ZJ, Napolitano GM, Lim YH. Investigation of potential collider bias in estimating the association between long-term exposure to air pollution and COVID-19 mortality. Environmental Epidemiology. 2025;9(3):e394. doi:10.1097/EE9.0000000000000394
23. Raittio E. Be aware of collider bias - comment on the association of carotid artery calcification with oral infections and mortality amongst symptomatic cardiovascular disease patients. Int Endod J. 2021 Apr;54(4):639–40. doi:10.1111/iej.13452.

24. Jackson ML, Nelson JC. The test-negative design for estimating influenza vaccine effectiveness. Vaccine. 2013 Apr 19;31(17):2165–8. doi:10.1016/j.vaccine.2013.02.053.
25. Lipsitch M, Tchetgen Tchetgen E, Cohen T. Negative controls: a tool for detecting confounding and bias in observational studies. Epidemiology. 2010 May;21(3):383–8. doi:10.1097/EDE.0b013e3181d61eeb.

ISSN: 0019-5138
DOI: https://doi.org/10.24321/0019.5138.202619