

Title: The Evolution of Selection Bias in the Recent Epidemiologic Literature—A Selective Overview

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Running Head: Evolution of Selection Bias Concept

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Abbreviations: SWIG, Single World Intervention Graph; DAG, directed acyclic graph

ABSTRACT

Selection bias has long been central in methodological discussions across epidemiology and other fields. In epidemiology, the concept of selection bias has been continually evolving over time. In this issue of *the Journal*, Mathur and Shpitser (*Am J Epidemiol.* XXXX;XXX(X):XXXX–XXXX) present simple graphical rules for using a Single World Intervention Graph (SWIG) to assess the presence of selection bias when estimating treatment effects in both the general population and a selected sample. Notably, the authors examine the setting in which the treatment affects selection, an issue not well-addressed in the existing literature on selection bias. To place the work by Mathur and Shpitser in context, we review the evolution of the concept of selection bias in epidemiology, with a primary focus on the developments in the last 20-30 years since the introduction of causal directed acyclic graphs (DAGs) to epidemiologic research.

Keywords: selection bias; collider bias; causal directed acyclic graph; single world intervention graph; causal inference; epidemiologic research

In epidemiology, the concept of selection bias has arguably proven more challenging to articulate than that of other systematic biases (i.e., confounding and measurement bias).¹ Moreover, the definition of selection bias varies across fields. In both clinical trials and economics, the term “selection bias” is commonly used to refer to what epidemiologists define as confounding.^{2–6} Similarly, in comparative effectiveness research, confounding bias is frequently labeled as “treatment selection bias.”^{7–9} These terminological discrepancies hinder effective communication and collaboration among researchers from diverse training backgrounds. In this issue of *the Journal*, Mathur and Shpitser¹⁰ present simple graphical rules for a Single World Intervention Graph (SWIG)^{11,12} to assess the presence of selection bias when estimating treatment effects in both the general population and a selected sample. Notably, the authors examine the setting in which the treatment affects selection, an issue not well-addressed in the existing literature on selection bias. This contribution deepens the field of epidemiology’s understanding of selection bias. To place the work by Mathur and Shpitser in context, we review the evolution of the concept of selection bias in epidemiology, with a primary focus on the developments in the last 20-30 years since the introduction of causal directed acyclic graphs (DAGs) into epidemiologic research¹³.

Before proceeding, it should be emphasized that selection bias, unlike confounding, can arise outside the context of causal effect estimation. Selection bias is relevant to descriptive and predictive studies as well.^{14,15} In this commentary, however, our primary focus is on selection bias in causal contexts.

Evolution of the Concept of Selection Bias Over the Last 20-30 Years

Selection bias has long been a key topic in methodological discussions across epidemiology and other fields.^{16–19} The introduction of DAGs over two decades ago provided a clearer and more structured way to represent biases in epidemiologic research.^{13,20} DAGs are particularly effective for

illustrating the structure and the mechanism of collider bias.^{13,21–23} This is especially highlighted when compared to other non-graphical methodologies, such as the potential outcomes framework, which fall short in elucidating collider bias.

In 2004, a pivotal paper by Hernán et al.²⁴ linked selection bias with collider bias using DAGs, and illustrated that selection bias can arise from conditioning on a common effect of two other variables, of which one is either the exposure or a cause of the exposure, and the other is either the outcome or a cause of the outcome (i.e., conditioning on a collider). The authors showed that such selection bias can occur for various reasons (e.g., differential loss to follow-up and non-response). Hence, their proposed definition of selection bias offered a structured perspective that helped unify various forms of selection bias in the epidemiologic literature. In their work, Hernán et al. also differentiated between confounding and selection bias using distinct causal structures; specifically, they argued that confounding arises from the presence of a common *cause* of both the exposure and the outcome, whereas selection bias results from conditioning on a common *effect*. Given that some fields conflate selection bias with confounding (in particular some describe confounding as “treatment selection bias”),^{2,6,7} the work by Hernán et al. helped carve out a unique identity for epidemiology by explicitly linking selection bias with collider bias and distinguishing selection bias from confounding using DAGs, thereby setting epidemiology apart from other fields in its approach to selection bias. Following the landmark paper by Hernán et al., additional articles helped elucidate the concept of selection bias, with the majority specifically addressing collider selection bias.^{1,25–33} Today, the concept of collider selection bias has been widely recognized in epidemiologic and medical research, as evidenced by its application in various domains, including genetic epidemiology^{34,35}, infectious diseases³⁶, health disparities³⁷, addiction³⁸, obesity³⁹, and reproductive epidemiology⁴⁰, among others. Furthermore, this concept has gained traction in other fields⁴¹.

The work by Hernán et al.²⁴ has led some epidemiologists to directly equate selection bias with collider bias. However, in the Special Issue of the *Journal* marking the 50th Anniversary of the Society for Epidemiologic Research in 2017, Hernán reviewed the article by Greenland in 1977 on non-response bias in cohort studies, and explained using DAGs that selection bias can also arise without colliders.^{16,42} Further, Hernán suggested that such occurrences of selection bias without colliders are closely tied to effect measure modification. However, since this work by Hernán was a short commentary, it inevitably had to omit certain technical details and nuances. Perhaps as a result, there continued some lack of clarity surrounding the concept of selection bias.

To harmonize and unify selection biases with colliders and without colliders, recently Lu et al. proposed a refined definition of selection bias with a simple taxonomy: type 1 selection bias owing to restricting to one or more level(s) of a collider (or a descendant of a collider), and type 2 selection bias owing to restricting to one or more level(s) of an effect measure modifier.³² For illustration, consider a common selection process that involves the general population and a selected sample as shown in Figure 1. Assume we aim to examine the effectiveness of a new medication for opioid use disorder in the general population (in Mathur and Shpitser's terminology; Lu et al. refer to this as the "target population"). Yet, due to certain reasons (e.g., individuals decline to give consent during study enrollment), we may only include a (oftentimes nonrandom) sample of the general population in the study, which is labelled as the selected sample (in Mathur and Shpitser's terminology; Lu et al. refer to this as the "study sample"). If we proceed without recognizing the potential for selection bias and analyze only the data from this selected sample, our effect estimate may account for all other biases (e.g., confounding) but overlook selection bias, either type 1 or type 2. Specifically, Lu et al. defined selection bias as "any bias away from the true causal effect in the

referent population, due to selecting the sample from the referent population.” Briefly, based on their definition, the referent population – that is, in the presence of selection bias, the population to which the true causal effect should refer – is the general population before the selection process, rather than the selected sample. Then the (overall) selection bias is defined as the difference between the true causal effect in the general population and the effect estimated from the selected sample (see Figure 1). This refined definition offered by Lu et al. helps unify the existing definitions of selection bias in epidemiology (i.e., selection bias with colliders which is named type 1 selection bias, and selection bias without colliders which is named type 2 selection bias). In addition, they delineated the relationships between the target population, the study sample, and the analytic sample as well as their implications for internal and external validity.

While Lu et al. provided a more unified definition of selection bias, these authors likewise did not fully clarify certain key technical and mathematical details. More recently, Sjölander shed light on some of the more technical aspects of Lu et al.’s definition of selection bias, focusing on outcome-dependent selection.⁴³ Specifically, Sjölander explored two forms of outcome-dependent selection: outcome-influenced selection (related to type 1 selection) and outcome-associated selection (related to type 2 selection), and evaluated whether these forms of outcome-dependent selection allow for causal effect estimation in the selected sample and in the general population. He demonstrated that while causal effects in the selected sample can be estimated under outcome-associated selection, they are not estimable under outcome-influenced selection. He also showed that the observed data in the selected sample have no information about causal effects in the general population under outcome-associated selection, but the data do contain some information about causal effects in the general population under outcome-influenced selection.

Mathur and Shpitser's Graphical Rules for Assessing Selection Bias

In this issue, Mathur and Shpitser propose graphical rules for evaluating selection bias when estimating treatment effects in the general population (the population in whom we ultimately want to make a decision) and in a sample selected from that general population (a selected sample).¹⁰ The authors extend Lu et al.'s work and examine the scenario when the treatment affects selection, a phenomenon that has been less explored previously. The contributions of Mathur and Shpitser are at least three-fold.

First, the authors use SWIGs to identify treatment effects in the presence of selection bias. SWIGs are a graphical approach that unifies causal DAGs and potential outcomes.^{11,12} Briefly, in SWIGs, we begin with a standard DAG for the effect of treatment A , and then the treatment A is split into two separate nodes: one node that represents a random component of A prior to intervening on it and inherits all incoming edges into A , and the other node that represents a fixed component (value) after the intervention of setting $A = a$ and inherits all the outgoing edges from A . Any node W that is a descendant of A is labeled as $W(a)$. SWIGs are particularly useful to describe the scenarios when the treatment A affects selection S , because as a descendant of A , the distribution of selection S and subsequently the selected sample ($S = 1$) will vary by different interventions on treatment A .^{44,45} Hence, it is more intuitive that we label the selection node as $S(a)$ to denote such dependence. For example, consider a randomized controlled trial evaluating the effect of a new treatment model for opioid use disorder (compared to standard-of-care) on mortality (see Figure 2), the probability of participants remaining in the trial during the 1-year study period depends on whether they are assigned the new treatment model, and thus we say the treatment A affects selection S . Subsequently, the counterfactual selected sample (those remaining in the trial) in the

hypothetical intervened world where everyone enrolled in the trial was assigned to the new treatment model ($A = 1$) will be different from the counterfactual selected sample in the hypothetical intervened world where everyone enrolled in the trial was assigned to the standard-of-care ($A = 0$). To capture this subtlety, Mathur and Shpitser adopt the concept of “net treatment difference,” which have long been used in statistics to describe the difference in mean potential outcomes comparing the treatment $A = 1$ with the treatment $A = 0$ after adjusting for a posttreatment variable.^{46,47} The net treatment difference, in the context of selection bias, is defined as “the net change in outcomes that would occur for the [counterfactual] selected samples if all members in the general population were treated versus not treated.”¹⁰ Put another way, the net treatment difference not only captures the effect of the treatment A on outcome Y , but also considers the impact of the treatment A on selection S (i.e., membership in the selected sample). It merits attention, however, that the net treatment difference does not represent a traditional causal effect if the treatment affects selection, because it does not compare potential outcomes for a fixed sample. Hence this noncausal estimand has uncertain utility for policymaking which considers a policy in a fixed population, and thus we urge caution and close consideration before using a net treatment difference in policymaking.

Second, by adopting the estimand of net treatment difference, Mathur and Shpitser decompose total selection bias into two parts: internal bias manifested as a discrepancy between naïve estimand (i.e., effect estimated from the factual selected sample without accounting for possible selection bias) and net treatment difference, and net-external bias manifested as a discrepancy between net treatment difference and conditional average treatment effect in the general population. The internal bias and net-external bias can be viewed as a generalized version of Lu et al.’s type 1 and type 2 selection bias³², adapted to encompass instances wherein the treatment affects selection. When the treatment

does not affect selection, internal bias and net-external bias are equivalent to type 1 and type 2 selection bias, respectively. This equivalency occurs because when selection S is independent of the treatment A , the counterfactual selected sample ($S(a) = 1$) used in estimating the net treatment difference in the hypothetical world by setting $A = a$ for everyone is identical to the factual selected sample ($S = 1$), and thus the net treatment difference is equal to the causal effect in the factual selected sample. However, when the treatment affects selection, internal bias and net-external bias differ from type 1 and type 2 selection bias, as the counterfactual selected sample used in estimating the net treatment difference is different from the factual selected sample. While decomposing overall selection bias into type 1 and type 2 is still a valid characterization even when the treatment affect selection, Mathur and Shpitser argue that the decomposition of bias into internal and net-external bias, by considering the net treatment difference, enhances conceptual clarity and facilitates assessment of each source of bias through a distinct graphical rule.

Last, Mathur and Shpitser offer two simple graphical rules for assessing the presence of selection bias in SWIGs, which are applicable regardless of whether the treatment affects selection. The first rule is $Y(a) \perp\!\!\!\perp A \mid S(a), Z$; where Z is a conditioned set of measured covariates. If the first rule holds, there will be no internal bias. Put another way, in a SWIG, if the potential outcome $Y(a)$ is independent of the random component of the treatment A given the selection $S(a)$ and a set of covariates Z , the naïve estimand from the factual selected sample will be equivalent to the net treatment difference. The second rule is $Y(a) \perp\!\!\!\perp S(a) \mid Z$. If the second rule holds, there will be no net-external bias. That is, in a SWIG, if the potential outcome $Y(a)$ is independent of the selection $S(a)$ given a set of covariates Z , the average treatment effect in the general population will be equivalent to the net treatment difference. If both Rules 1 and 2 hold, then there will be no selection

bias at all and the average treatment effect in the general population becomes identified using the naïve estimand from the factual selected sample. Mathur and Shpitser further demonstrate that if Z is a sufficient set of confounders for the treatment-outcome relationship in the general population, then average treatment effect in the general population is identifiable once Rule 2 holds by conditioning on Z . This robust proposition offers a pragmatic tool for pinpointing and addressing selection bias in nuanced situations, exemplified by the case of selection bias discussed in Breskin et al.⁴⁴ It should be noted that oftentimes g-computation, inverse probability weighting or doubly robust estimators are needed to account for the full distribution of covariates Z in order to address selection bias and recover the marginal treatment effect in the general population.

Conclusion

Mathur and Shpitser provide a novel perspective for assessing selection bias, leveraging SWIGs and adopting the "net treatment difference" concept.¹⁰ Similarly, Kenah has recently employed SWIGs as a potential outcomes approach to selection bias, delineating two separate analytic conditions for evaluating selection bias: the analytic cohort condition and the analytic case-control condition.⁴⁸ Both works underscore the valuable role of methodological advancements—such as SWIGs—in further clarifying longstanding and important concepts within epidemiologic research. It is anticipated that as we embrace new methodologies, our comprehension of selection bias will deepen and mature.

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1 Figure 1. A common selection process involving the general population and a selected sample. It also depicts Lu et al.'s definition of
2 selection bias³², which is a combination of type 1 selection bias (i.e., selection bias with colliders) and type 2 selection bias (i.e., selection
3 bias without colliders). For the purpose of illustration, we assume the effect estimated from the selected sample has accounted for all forms
4 of biases (e.g., confounding and measurement bias) except for selection bias.

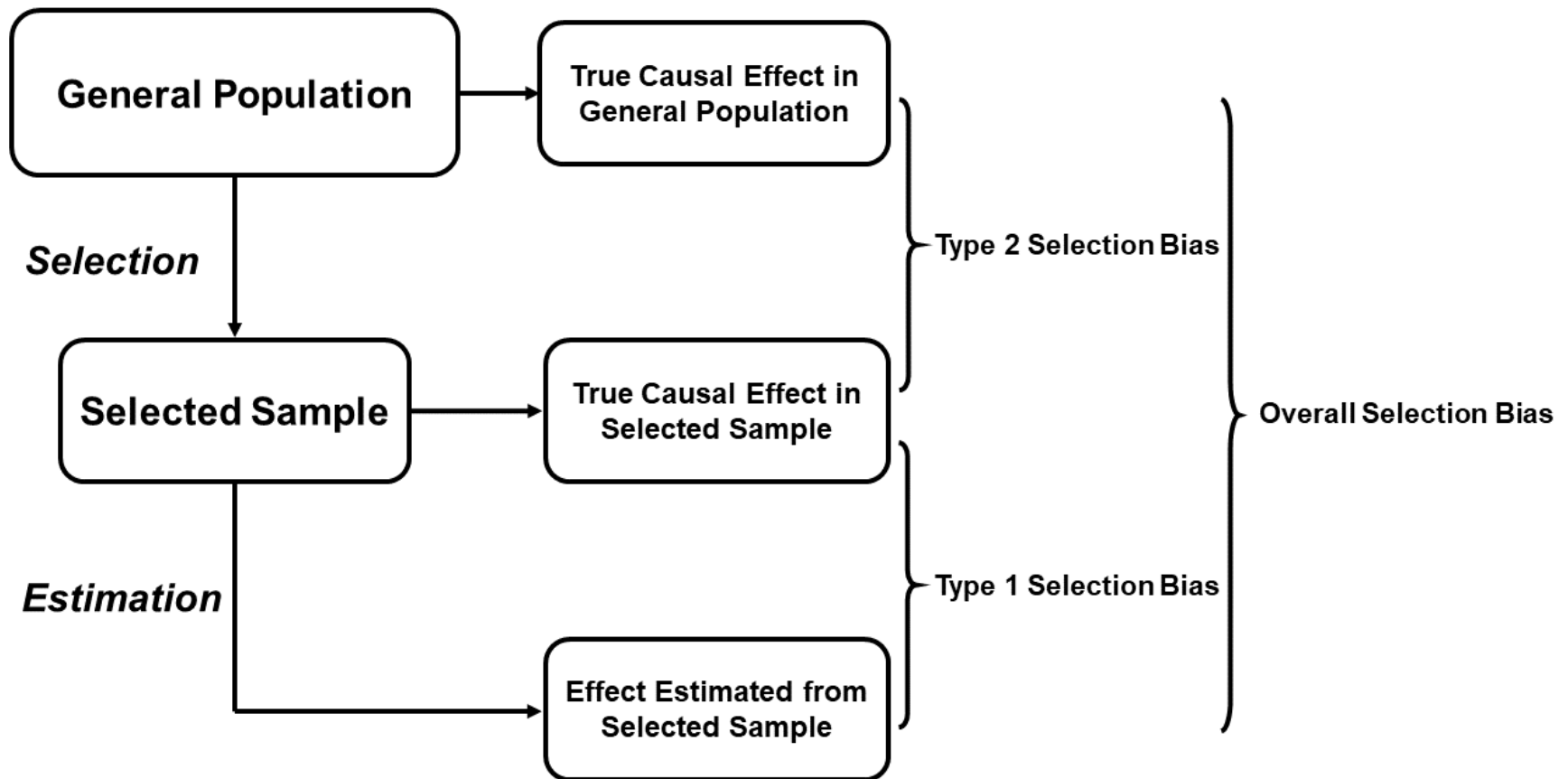
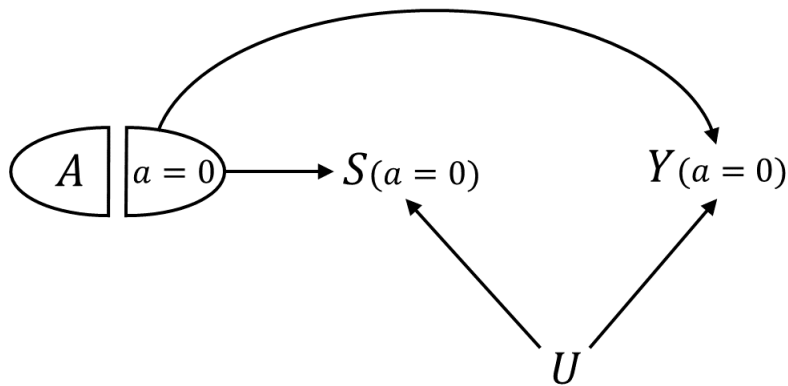


Figure 2. A randomized controlled trial, where A is certain new treatment model versus standard-of-care for opioid use disorder, Y is the 1-year mortality, S is selection node (i.e., remaining in the trial during 1-year study period), and U is an unmeasured common cause for selection S and outcome Y (e.g., access to healthcare). Figure 2A is a SWIG that represents the hypothetical intervened world by setting $A = 0$ for everyone in the general population; Figure 2B is a SWIG that represents the hypothetical intervened world by setting $A = 1$ for everyone in the general population. SWIGs are often expressed using templates. Here, for illustrative purposes, two SWIGs are depicted to represent two different counterfactual selected samples under different interventions on A .

(A)



(B)

