Letters to the Editor

Nonrandom Selection and the Attributable Cost of Surgical-Site Infections

To the Editor:

In the April 2002 issue of *Infection Control and Hospital Epidemiology*, Hollenbeak et al.[1] claimed to control for selection bias in a model of the additional cost of surgical-site infection. The authors developed a two-stage model to additionally control for a variable that was derived from the risk factors for wound infection. They argued that these risk factors would also independently increase cost, thus acting as true confounders. Presumably, the purpose of including this risk index on the right-hand side of their regression model was to minimize confounding, which Haley describes as severity of illness bias.[2]

We understand selection bias to occur only when common factors determine participation in the research and the likelihood of acquiring the disease or outcome. For example, selection bias might occur in case–control studies to explore the additional cost of hospital infection when cases are excluded because no match can be found for them, for all variables, from the controls. Because all patients were included in the data set used by Hollenbeak et al., selection bias should not be a problem. We suggest that they controlled for severity of illness (to be welcomed), not selection bias.

REFERENCES

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2. Haley RW. Cost-benefit analysis of infection control activities. In: Brachman P, Bennett J, eds. *Hospital Infections*. Philadelphia: Lippincott-Raven; 1998:249-267.

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The authors reply.

Drs. Birrell and Graves raise an important point about the language that is used to describe selection bias. As they mention, one form of selection bias can occur when common factors determine participation in a study. More generally, selection bias may arise whenever there is a systematic mechanism that determines both outcomes and study participation or the distribution of treatment. For example, dropouts from randomized trials may lead to selection bias if there is a variable that is related to both the decision to drop out and the treatment effect. In this case, the selection mechanism is self-selection, and the selection bias would be observed in the estimated treatment effect.

In the case of nosocomial infections, we hypothesized that estimates of the attributable cost of surgical-site infections may suffer from selection bias, and proposed a model that would allow us to test for its presence. We framed our discussion in terms of selection bias because there is an underlying mechanism that selects some patients to develop surgical-site infections, and the variables that drive the selection process are correlated with costs. For example, obesity and diabetes have both been shown to increase a patient's risk for surgical-site infections, and have been associated with increased costs independent of infection. The selection mechanism we hypothesized was not standard; it goes without saying that infections are not assigned based on self-selection. However, there is an underlying natural process that results in a systematic distribution of infections; therefore, it is appropriately modeled as a selection mechanism, and its impact on the treatment effect is appropriately called a selection bias.

Note that the selection bias we hypothesize is not due to the systematic deletion of observations, as Drs. Birrell and Graves suppose, but rather because risk factors for infection have two effects on costs: a direct effect, which can be controlled by including the risk factor as a covariate, and an indirect effect, which inflates the treatment effect or the coefficient on the binary infection indicator. Simply controlling for the risk factor as a confounder in, for example, a regression context would address the direct effect but would not mitigate the indirect effect. The purpose of including the inverse Mills ratio was not to minimize confounding, but rather to absorb the selection effects.

It is important to contrast this notion of selection bias with severity bias, which arises when patients who develop infections are "sicker" than patients without infections, even before they developed an infection. It is hoped that it is clear why we described the effect for which we attempted to control as a selection bias and not a severity bias. The variables that contribute to the selection mechanism for infections are not necessarily related to disease severity, although they could be. If they were, disease severity could be included in the first stage probit regression as well as a covariate in the second stage regression.

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Surveillance of Nosocomial Surgical Wound Infections: A Few Suggestions

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