The Association of Body Mass Index With Health Outcomes: Causal, Inconsistent, or Confounded?

Eyal Shahar

Initially submitted October 8, 2008; accepted for publication July 30, 2009.

According to the definition of confounding in a causal diagram, the association of body mass index (weight (kg)/height (m)^2) with health-related outcomes is almost always noncausal, attributable to confounding by weight and perhaps height. The same conclusion holds for any other deterministic derivation from weight and height. No causal knowledge is gained by estimating a nonexistent effect of body mass index.

bias (epidemiology); body mass index; causality; confounding factors (epidemiology)

Abbreviation: BMI, body mass index.

Editor’s note: An invited commentary on this article appears on page 959, and the author’s response appears on page 963.

Body mass index (BMI; weight (kg)/height (m)^2) is a widely used variable in causal analysis, often assumed to be a cause of health-related outcomes, including death. The index has been criticized recently from various methodological viewpoints (1–3), but its most fundamental shortcoming has not been recognized: Rare examples aside, BMI is not a cause of health-related outcomes. This conclusion is quickly reached when causal assumptions are displayed in a directed acyclic graph (4).

Although surely incomplete, Figure 1 shows relevant causal relations with mortality, most of which are not controversial. The causes of measured weight and height are the true versions of these variables, as well as every source of measurement error (5). The arrow emanating from “True Weight” and pointing to “Mortality” corresponds to a widely accepted theory, whereas the arrow from “True Height” to “Mortality” is controversial. Notice that computed BMI must be a deterministic effect of measured weight and measured height: There are no other immediate causes of this variable.

Most important, no arrow is pointing from “True BMI” to “Mortality,” because an arithmetic derivation (weight/height^2) should not have a purely pathophysiologic effect on survival. Otherwise, the very same causal hypothesis could be proposed for an infinite number of arithmetic derivations: weight/height, weight^2/height, and so on. Likewise, no arrow is pointing from “Computed BMI” to “Mortality,” because we don’t usually expect that computing or measuring something would affect survival (except via cognitive-behavioral pathways).

Previous writers have rejected BMI for failing to meet the consistency assumption (1) (or requirement?), which states that every causal contrast must be defined on a well-defined intervention. They would therefore object to the drawing of an arrow from weight to mortality or to estimating that effect—because many types of interventions could lead to a given weight. Nonetheless, the requirement to reduce effect estimation to well-defined interventions seems far too restrictive, immediately raising philosophical questions about “acceptable counterfactuals” (6) and hidden causal reality.

For example, did Nature create only causal connections that correspond to the human conception of a randomized trial? Or alternatively, did Nature divide our postulated causal arrows into those that carry a causal parameter and those that do not? The effect of a causal contrast between weighing 300 pounds (factually) and weighing 200 pounds (counterfactually) over time is a logical theory for which causal inference is possible (6), regardless of any well-defined intervention that would
change anybody’s weight. Of course, estimating an effect is always a fallible exercise—even in a valid randomized trial where the estimated effect might miss the causal parameter. Regardless of the consistency assumption, almost all associations between BMI and mortality are not causal. As Figure 1 shows, they are fully attributable to confounding (7) by weight and perhaps height, which are the common causes of BMI and mortality. BMI itself has no effect on mortality. To study the effect of true weight, we should use measured weight as the best proxy we can get, not BMI, which is 2 steps removed from the causal agent. If effect-measure modification is suspected, interaction terms between weight and height should be added, not a ratio of one variable to the (squared) other (8, 9). Finally, weight/height\(^2\) does not capture any aspect of adiposity that is missed by weight. Weight/height\(^2\) is no more than a deterministic function of 2 measured variables. Its association with adiposity is fully explained by the effect of adiposity on weight (Figure 1).

In summary, BMI may be a useful variable for the layperson and the clinician, but there are no compelling arguments for modeling BMI in causal inquiry. In fact, there is a good reason to abandon dogmatic research practice, which is anchored in tradition more than in critical thinking.

**ACKNOWLEDGMENTS**

Author affiliation: Division of Epidemiology and Biostatistics, Mel and Enid Zuckerman College of Public Health, University of Arizona, Tucson, Arizona.

Conflict of interest: none declared.

**REFERENCES**