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.

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.
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.

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