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REPLY

We appreciate the interest by Drs. Henrikson and Chandra-Strobos in our recent study (1). They disagree with the conclusion that lower levels of troponin are likely analytical false positives and are more likely related to necrosis. This is based on the results of their recent study (2), in which patients with minor troponin T (TnT) elevations had a higher event rate than those without detectable levels, and were closer to those with clearly elevated levels (2).

However, the two studies are not directly comparable. The range of TnT levels, which they called “marginal” (defined as levels between 0.01 ng/ml and 0.09 ng/ml), was fairly broad. The TnT levels from 0.01 to 0.03 ng/ml (between the lower limit of detectability and 10% coefficient of variation [CV]) (3) are equivalent to our low troponin I values, whereas those from 0.03 to 0.09 ng/ml (from the 10% CV to the prior myocardial infarction [MI] diagnostic criteria) would be equivalent to our intermediate TnI levels. The fact that their marginal TnT values are a combination of low and intermediate TnT values likely explains the higher event rate that was found. For example, the proportion of patients who had elevated creatine kinase-MB fraction (CK-MB) was 15% in our TnI intermediate group, comparable to 14% in their marginal TnT group; in contrast, only 1.1% of patients in our TnI low group had increased CK-MB.

In addition, we did not call patients with detectable TnI values “normal.” We made the distinction between low elevations, in which some represent necrosis; this is seen by the higher event rate. However, as we and others have noted (4), this is a mixture of a small number of patients who truly have necrosis and a much higher number who have analytical false positives. As we noted, labeling a patient who has atypical chest pain, no-ischemic electrocardiographic changes, low levels of CK-MB not near the diagnostic cut-off, and with minor troponin elevations as having an myocardial infarction has significant implications for the patient’s long-term health care, and we believe this is inappropriate. As none of the currently available assays conform to recommended standards (5), our data, as well as other recommendations (6), are that these values may be indicative of necrosis, and therefore should be further evaluated. This evaluation should be dependent on the clinical scenario. Rather than routinely performing coronary angiography as recommended by the American College of Cardiology/American Heart Association guidelines for patients who have troponin elevations, we believe that stress testing would be an appropriate evaluation, for many of these patients.

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The Diet–Heart Hypothesis: An Evolutionary Support

Nothing in biology makes sense except in the light of evolution.
Theodosius Dobzhansky, 1973

Considering that humankind and its metabolic physiology are biological products of evolution, one might well ask why Weinberg’s report (1) not only ignores the evolutionary arguments supporting the diet–heart hypothesis, but also advances the evolutionarily untenable thesis that the low-fat, high-carbohydrate diet is responsible for the current epidemics of obesity, type II diabetes, and the metabolic syndrome.

Weinberg seems to forget that the low-fat, high-carbohydrate diet represents the diet “for which human beings are in essence genetically programmed” (2), because their metabolic physiology has been evolutionarily molded by a nutritional environment in which, for millions of years, that diet was practically the sole one available to our ancestors (3). For 99% of its evolution, humankind indeed lived mainly on fruits and vegetables, which consist essentially of carbohydrates, and consumed little fat, because game was very lean and cattle-breeding, chicken farming, butter, dairy products, margarines, and oils did not exist (2).