The fat mass index: why its height exponent should be 3 and not 2

Dear Sir:

Calculated as (fat mass)/height^2, the fat mass index (FMI) is a measure of relative fat content. It has an advantage over percentage body fat (%BF) in that it is independent of fat-free mass. Weber et al (1) recently published FMI reference curves for children and adolescents aged 8–20 y. However, the FMI is not generally valid as an index of fatness, and a more appropriate formulation is (fat mass)/height^3 (2). The results of Weber et al (1) are reinterpreted below in terms of the latter index.

The FMI was first conceived as being 1 of 2 components of the BMI, the other being the fat-free mass index (1), and in that context it can be useful. However, the BMI is a special case of the Benn index, mass/height^2 (3), with p approximated as the round number 2. The actual value of p is determined statistically for a given population. Both indexes are thus statistical constructs and lack the physical meaning of, say, height, mass, or density. This must be true also of the BMI’s component, the FMI.

What is needed is a measure of fatness that is not founded in population statistics. In addition, it needs to be dimensionless (ie, unit-free) (4), such as %BF, and identical for individuals of different sizes who share the same body build and relative fat content. One such index is the ratio (fat mass)/(D × height^3), where D is the density of fat (2). Because D is virtually constant, it may be dropped from the index to give (fat mass)/height^3 (2). This, then, is a proper index expressing fatness relative to height. A similar argument applies to the fat-free mass index.

We may now address the relations between FMI and age as shown in Figure 1 of Weber et al (1), which shows smoothed centile curves for males and females. For females, all of the curves show a steady increase in FMI with increasing age from 8 to 20 y. For males, the 75th to 95th centile curves also show an increase, although less steep, whereas the 50th centile curve increases only a little. The 5th to 25th centile curves are almost horizontal. The question now is how would the picture change if the index (fat mass)/height^3 were substituted for the BMI? Median values of the former index may be estimated from the tabulated values of the FMI divided by median values for height given by the Centers for Disease Control and Prevention (www.cdc.gov/growthcharts/). Both sets are based on NHANES data. The new index, mass/height^3 behaves more like %BF than does the FMI. Other studies have also shown the decrease in median %BF between the ages of 11 and 16–17 y (6, 7). Calculated from median UK data for males (8), both %BF and (fat mass)/height^3 decline over the age range of 11–16 y.

The index (fat mass)/height^3 shares with the FMI the advantage over %BF that it is independent of fat-free mass and therefore of, for example, muscularity and short-term malnutrition. Instead, it is affected by the ratio of leg length to total height, just as is the BMI. This ratio tends to increase with age in young children before decreasing again from the ages of ~13 y in girls and 15 y in boys (9). The measure of fatness, (fat mass)/height^3 or %BF, that is most appropriately used must depend on circumstances, so that, in general, it may be sensible to report values for both.

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Reply to RF Burton

Dear Sir:

The letter from Burton addresses an important issue regarding the use of a fat mass index (FMI). Similar to BMI, the goal of expressing fat mass relative to height is to have an index of body fat that is independent of overall body size. Burton proposes that \((\text{fat mass})/\text{height}^3\) is a more appropriate formulation for FMI compared with \((\text{fat mass})/\text{height}^2\). The changes in body proportions and body composition during childhood and adolescence are indeed complex, so the task of identifying the optimal index across the entire age range is challenging. There were multiple considerations in our selection of \((\text{fat mass})/\text{height}^2\) rather than other indexes such as the one proposed by Burton.

The primary rationale for expressing fat mass relative to height squared in our article (1) was to generate reference data for fatness that could be readily compared with existing means of assessing adiposity in the pediatric population. BMI, calculated as \((\text{body mass})/\text{height}^2\), is the most widely used method to screen for excess adiposity in children and adults. Because of the familiarity with BMI as a frame of reference across the research, clinical care, and public health spectrums, FMI is a meaningful measure.

It is important to note that BMI is a measure of body mass that is independent of height in adults (2), but this is not the case in the pediatric population in whom a positive correlation between height and BMI is generally seen (3, 4). The same is true for fat mass. The true value of \(p\) [as in the equation \(\text{FMI} = (\text{fat mass})/\text{height}^p\)] necessary to eliminate the correlation between height and FMI and BMI has been investigated previously and found to vary across the pediatric age range (5, 6). Cross-sectional correlations between height and measures of adiposity including BMI, FMI \([\text{fat mass}/\text{height}^2]\), and FMI3 \([\text{fat mass}/\text{height}^3]\) at each age for children aged 8–20 y in our NHANES sample are shown in Figure 1. For all 3 measures, the correlations with height tend to be highest around the ages of the adolescent growth spurt and are lower at older ages. Although the correlation with height was lower for \((\text{fat mass})/\text{height}^2\), the use of \((\text{fat mass})/\text{height}^3\) did not eliminate the positive correlation with height seen among younger children and resulted in a larger negative correlation among older children.

Most important, it is unclear whether the ideal index of adiposity should be independent of height because this may mask biological associations between adiposity and height (7). Studies have found that height-independent formulations of body mass and fat mass may be inferior for the detection of cardiometabolic risk factors compared with traditional formulations of BMI and FMI (6, 8). To our knowledge, there are no studies that show that \((\text{fat mass})/\text{height}^3\) is superior to FMI or BMI in identifying children at increased cardiometabolic risk.

Burton also maintains that curves for \((\text{fat mass})/\text{height}^3\) are preferable to FMI because they more closely resemble those for percentage body fat (%BF). The rationale for using %BF as the gold standard comparator is unclear, because he acknowledges that %BF fails to take into account the independent contributions of fat and lean body mass. The decrease in the median %BF for boys aged 11–17 y is likely a result of the rapid accumulation of lean body mass during puberty. The use of FMI and lean body mass index allows for the independent evaluation of fat and lean body mass, and thereby would allow for an individual who has accumulated excessive fat mass in addition to lean mass to be identified for screening. That same individual with high fat and lean body mass would be missed if %BF were used as the screening tool.

In summary, the letter by Burton underscores many of the challenges in analyzing body composition in children. Reference curves for lean body mass index and FMI for children and adolescents are now available, so that future body composition studies have a frame of reference with which to evaluate lean and fat mass relative to height and age. Interested clinicians and researchers may use an online calculator to convert dual-energy X-ray absorptiometry measures of whole-body fat and lean body mass into age- and sex-specific z scores (http://www.research.chop.edu/web/zscore/). It is our hope that the scientific community will use this reference data in future studies aimed at the assessment of body composition in diverse patient populations and its relation to health outcomes.

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**Primefructose study misses mark due to preventable design flaws**

Dear Sir:

A recent article by Kavanagh et al (1) concluded that high exposure of fructose to primates (24% of total energy) induces hepatic lipidosis when consumed ad libitum for periods >1 y and significant liver damage brought about by inflammation when consumed under short-term, calorie-controlled conditions. Although the study aims to supply the primary mechanism by which high dietary fructose exposures provoke human metabolic disease, it misses the mark due to preventable design flaws.

First, the presentation of percentage of nutrients in the authors’ Table 1 obscures critical differences in composition between control and high-fructose (HFr) diets. Perhaps for reasons of convenience, but inexplicable from a nutritional standpoint, the authors chose to formulate the purified HFr diet with different carbohydrate, protein, and fat sources than found in the Purina Chow control diet. However, it has been known for nearly half a century that formulation choices can profoundly influence metabolic outcomes: the seminal work of Kritchevsky et al (2–5) thoroughly explored nutrient interactions in animals, including primates, and showed unequivocally that blood lipids and the course of atherosclerosis are materially affected by interactions between type and amount of protein, carbohydrate (including fructose), and nonnutritive fiber.

Second, the authors offer a meaningless comparison of extreme fructose amounts, both too low (control) and too high (HFr) to be within the normal range of human, and surely primate, consumption. Low fructose amounts comparable to the control group (<0.5% of energy as fructose) would be achievable only by those subsisting on a diet of starches, protein, and fat; no fruit or vegetables and no added sugars. The high fructose value in the HFr diet (24% of energy) may have arisen from a misreading of Marriott et al’s (6) fructose exposure data: the authors appear to have selected the 95th percentile of fructose intake as a percentage of carbohydrate intake for adolescent US males (24.6%); Marriott et al’s Supplemental Table 3) instead of the correct 95th percentile fructose intake as a percentage of energy intake (14.6%; Marriott et al’s Supplemental Table 2). For clarification, the population subgroup identified by Marriott et al with the highest fructose intake was young adult women (19–22 y), with 17.9% of energy as fructose. As a result of this oversight, the HFr monkey group was exposed to an amount of fructose >30% above the most extreme human consumers of fructose.

Finally, it is disconcerting to find errors in the article and in the accompanying press release (7) mischaracterizing the composition of experimental and common ingredients and exaggerating the dietary prominence of fructose. According to the authors’ Table 1, protein in the control group diet was composed of whey, grain, and fish meals, not soy protein. Fructose is not the main sugar in corn syrup, a fructose-free food ingredient composed entirely of glucose and glucose oligosaccharides. And whereas fructose is certainly 1 of the 2 most commonly added sugars (with comparable glucose) in the American diet, its metabolic influence is surely diminished by the 5-fold surplus of glucose from all dietary sources (8).

In summary, the study by Kavanagh et al (1) aiming to explain how high dietary fructose provokes human metabolic disease misses the mark. Preventable design flaws in diet formulation and fructose dosing leave the study with little relevance to human health.

As a consultant and advisor to the food and beverage industry in the area of nutritive sweeteners, the author receives compensation from scientific societies, research institutes, food industry councils, trade organizations, and individual companies. Clients have an ongoing interest in nutritive sweetener research, development, production, applications, safety, nutrition, and education.

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Reply to JS White

Dear Sir:

Studies using dietary conditions with exaggerated nutritional compositions are often used to define the potential for biological responses and to act as starting points for understanding nutritional effects on health. The monkeys in the study consumed 30% more fructose than the unhealthiest humans, with fructose approximating the 95th percentile of total carbohydrate consumption (1, 2). This intake was modeled on the clinical study by Stanhope et al (3), which showed changes in adiposity, plasma lipids, and insulin sensitivity. Monkeys supplemented with fructose at this same amount (4) similarly experienced detrimental changes in these variables. Thus, our goal was to evaluate the influence of an extreme change in fructose while holding adiposity constant by careful focus on caloric and body weight control. One advantage of animal models, especially Old World monkeys with gastrointestinal tracts more comparable to humans, is sufficient longer-term environmental control to unmask perturbations induced by manipulation of nutritional variables. A potential next step would be to narrow the intake range over which health effects may be seen, while matching all dietary ingredients including protein source. Casein is known to be proatherogenic in the context of cholesterol-loaded dietary experiments (5); however, we actually reported no elevations in plasma or liver cholesterol concentrations (free or esterified) with high-fructose/casein diets, endpoints that are known risk factors for atherosclerosis (6). We therefore believe that the lack of differences seen in these endpoints in our study suggests that protein source did not primarily influence metabolic health, microbial translocation, and liver injury outcomes. We conclude that consumption of very high amounts of simple carbohydrate is likely to impair intestinal integrity and initiate liver and metabolic changes. Human and rodent studies involving exaggerated diets support the greater potency of fructose compared with glucose in initiation of such changes (3, 7), and future experiments should refine these findings using relevant diets in relevant animal studies.

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REFERENCE


An incorrect link to the ASN public policy update published in the August 2013 issue of *The Journal of Nutrition* appears on page 626. The published link is http://www.jn.org/143/8/1355.full.pdf+html, but the correct link is http://jn.nutrition.org/content/143/8/1355.full.pdf+html. The announcement should read as follows: “The following summary of ASN’s public policy–related activity with regard to the *Dietary Guidelines for Americans* is available in the August issue of *The Journal of Nutrition* (http://jn.nutrition.org/content/143/8/1355.full.pdf+html): The many roles of ASN scientists in the *Dietary Guidelines for Americans* process. Ohlhorst SD.”