different girths. To this end, heights and measures of relative fatness, uncorrelated, can be randomly generated within realistic limits. Values of log(WC) and several differing sets of log(X) are then calculated from Equations 1 and 2 by using chosen values of B, C, E, and F. Scatter is introduced through random variations in C and F. The correlation coefficient for log(WC) and log(height) can be made to equal the real value of 0.04 (1) by adjusting E. The individual regression coefficients produced in this way have the right signs but are otherwise of no interest, being necessarily based on arbitrary input parameters and affected by multicollinearity. More interesting is the less predictable fact that the sum of all the coefficients can be close to the value of 1.15 obtained by Wells et al (1). Moreover, if log(height) is regressed simultaneously on log(WC) and the logarithms of the other girths by using the same simulated data, the sum of all the coefficients is much lower—like the average value of 0.62 based on Table 6 of Wells et al (1).

The apparent negative association between WC and height in men may be approached in a more intuitive way, starting with a familiar analogy. Fatness is correlated with body mass index (BMI) and therefore with log(BMI)—and so also with [log(weight) − 2 : log(height)]. Multiple regression of some fatness measure on log(weight) and log(height) must therefore produce a negative coefficient for log(height) (regardless of whether the relations are actually linear). Fatness and weight obviously tend to vary together, but no one can suppose that height has a direct negative influence on percentage body fat. To make this analogy more immediately relevant, note that WC, like fatness, correlates strongly with BMI in men, with a correlation coefficient of 0.95 for example (6). Therefore, log(WC) must correlate with [log(weight) − 2 : log(height)]. Accordingly, regression of log(WC) on log(weight) and log(height) would produce a negative coefficient for log(height)—without any implication that abdominal girth tends to be less in tall men.

In conclusion, the apparent negative association between WC and height in men illustrates one of the hazards in interpreting multiple regression equations when these are not serving their usual predictive role. WC shows a small but significant positive correlation with height, when weight is also included, must inevitably generate a negative coefficient for height. It seems that he considers waist to be such a proxy for adiposity but not the other girths. However, several girths besides waist increase substantially across the range of body mass index (BMI) in both sexes. Average girths by 4 categories of BMI in 907 men and 1330 women aged 21–30 y from Size UK are shown in Table 1. The BMI categories (in kg/m²) are <20, 20–24.99, 25–29.99, and ≥30. For each girth, we also calculate the percentage difference between the highest and lowest BMI category. It can be seen that although waist increases substantially with BMI group, so too do girths of the arm, hip, thigh, and bust (women only); whereas increases in knee and chest girths are more modest, and those in head girth are negligible. If one assumes that knee and chest girth incorporate little adiposity, such that their percentage increases with BMI represent increasing body frame size, then other girths with greater percentage increases are likely to reflect increasing adiposity as well as frame size. These data thus indicate that several girths reflect positive associations between BMI and adiposity, a point also made by Heymsfield et al (6). However, our multiple regression analysis found that holding other girths constant, waist girth was strongly negatively associated with height in men but only very weakly so in women (1).

Burton argues that "no one can suppose that height has a direct negative influence of percentage body fat," and seemingly transfers this logic to waist girth. It seems that he did not register our previous

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Dear Sir:

We welcome a second opportunity to discuss our findings regarding a significant negative association between height and waist girth in men in the UK National Sizing Survey, which contrasted with positive associations for head, knee, hip, chest, and arm girths with height (1).

Burton argues that body weight should be proportional to height cubed. This may be true across a range of species varying widely in body size (eg, the classic mouse to elephant spectrum), where the correlation between weight and height approximates to 1. It is not true within a species, where the correlation between weight and height is reduced. In the case of the UK Sizing Survey data, for example, the correlation between weight and height was only 0.31 in women and 0.39 in men. These relatively low coefficients reflect the very wide range of nutritional status in this sample. Accordingly, weight has been consistently shown to scale with height squared in humans (2), and indeed many individual tissues and organs likewise scale with height squared (3, 4). For lung volumes in particular, Cole has shown algebraically why this should be (5). The argument that the regression coefficients of girths on heights should sum to 3 is not derived from appropriate theory. It is difficult to understand Burton’s subsequent points in which he similarly predicts certain values from summing regression coefficients presented in our tables.

Burton then argues that regression of an index of body fat on height, when weight is also included, must inevitably generate a negative coefficient for height. It seems that he considers waist to be such a proxy for adiposity but not the other girths. However, several girths besides waist increase substantially across the range of body mass index (BMI) in both sexes. Average girths by 4 categories of BMI in 907 men and 1330 women aged 21–30 y from Size UK are shown in Table 1. The BMI categories (in kg/m²) are <20, 20–24.99, 25–29.99, and ≥30. For each girth, we also calculate the percentage difference between the highest and lowest BMI category. It can be seen that although waist increases substantially with BMI group, so too do girths of the arm, hip, thigh, and bust (women only); whereas increases in knee and chest girths are more modest, and those in head girth are negligible. If one assumes that knee and chest girth incorporate little adiposity, such that their percentage increases with BMI represent increasing body frame size, then other girths with greater percentage increases are likely to reflect increasing adiposity as well as frame size. These data thus indicate that several girths reflect positive associations between BMI and adiposity, a point also made by Heymsfield et al (6). However, our multiple regression analysis found that holding other girths constant, waist girth was strongly negatively associated with height in men but only very weakly so in women (1).

Burton argues that "no one can suppose that height has a direct negative influence of percentage body fat," and seemingly transfers this logic to waist girth. It seems that he did not register our previous
letter, in response to Nevill et al, in which we made exactly this point: relations between size (height) and shape (adiposity) are not due entirely to adult allometry but also reflect patterns of growth and development (7). A number of studies (although not all) have associated stunting in early life with increased central fat, which would translate in an adult population into an inverse association between height and waist girth (7). We also referenced a study of Siberian women, in which short stature was associated with a lower fasting fat oxidation rate, greater weight and adiposity, and higher serum lipid concentrations (8).

We have emphasized in various articles the need to adjust adiposity appropriately for body size (9, 10), but appropriate statistical models cannot afford to ignore biology, and in particular developmental biology, through which adult variability is generated.

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LETTERS TO THE EDITOR 865

Anthocyanin analysis in banana fruit—a mistake

Dear Sir:

Recently, Cassidy et al (1) reported that “participants in the highest quintile of anthocyanin intake (predominantly from blueberries and strawberries) had an 8% reduction in risk of hypertension […] compared with that for participants in the lowest quintile of anthocyanin intake.” In Figure 1 of their article, bananas are shown to be the second or third important source of anthocyanins. The US Department of Agriculture (USDA) database for flavonoids used by the authors (2) provides a delphinidin value for bananas of 7.39 mg/100 g, which is taken from Harnly et al (3), who used acidified methanol (1.2 N HCl) at 75°C for extraction of

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<td>Average values for body girths according to 4 categories of BMI in men and women aged 21–30 y from Size UK</td>
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1 % diff, percentage difference between values for the BMI groups ≥30 and <20, calculated as [{(≥30 value − <20 value)/<20 value} × 100].


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