

## LETTERS

edited by Jennifer Sills

## Health and Obesity: A New Normal?

IN THEIR PERSPECTIVE “THE HEALTH RISK OF OBESITY—BETTER METRICS imperative” (23 August, p. 856), R. S. Ahima and M. A. Lazar ask how a normal body mass index (BMI) could be deleterious to health. A better question might be: Why should a value of BMI be arbitrarily defined as “normal” and used as a benchmark in discussing health risks, when it is possible to define an optimal value of BMI by reference to unequivocal data points such as BMI versus age at death?

Ahima and Lazar cite Flegal *et al.* (1), who found that “overweight was associated with significantly lower...mortality.” That is, subjects in Flegal *et al.*’s “overweight” group (BMI

25 to 30) had a lower mortality rate than subjects in her “normal” group. These data suggest that the optimal BMI is higher than the “normal” BMI. Other studies, which use the unequivocal end point of death to define optimality, confirm this implication. For instance, Durazo-Arvizu *et al.* found that minimal mortality was associated with BMI ranging from 23 to 30 (2). Another study showed that adults with BMIs of 18.5 to 24.9 at the onset of diabetes had higher mortality than those with BMIs of 25 or higher (3). Others have demonstrated that optimal BMI increases with increasing age (4). In a review of 40 studies, Hamer and Stamatakis could not find a confounding factor that could explain the better outcomes in overweight and mildly obese groups (5).

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Health and Obesity:  
Not Just Skin Deep

IN THEIR PERSPECTIVE “THE HEALTH RISK OF obesity—better metrics imperative” (23 August, p. 856), R. S. Ahima and M. A. Lazar discuss findings that suggest positive effects of obesity on mortality and call for better body shape and fat distribution measurements. Other adipose factors may also be important.

It has long been known that the size of

fat cells is important in metabolic obesity complications (1). Recent evidence suggests additional connections to fat cell number and turnover. At any level of body fat, human adipose tissue can have either hypertrophy (a small number of large cells) or hyperplasia (a large number of small cells). Human fat cells turn over at a high rate (2), but turnover is markedly lower in subjects with adipose hypertrophy (3). Hypertrophy is linked to a disadvantageous metabolic profile in all body types: healthy lean, overweight, obese (3), and morbidly obese subjects (4). Genetic predisposition for type 2 diabetes is also associated with adipose hypertrophy (5). In other words, it is possible to look lean and healthy on the outside but have obesity/diabetes-prone adipose tissue on the inside.

Taking early and recent findings together, it is possible that in hypertrophy, adipocyte production rate is low, requiring existing fat cells to accumulate more lipids than in hyperplasia. Supporting this notion is the strong

connection between fat cell lipid turnover, lipid metabolism, and insulin sensitivity (6). Fat cell turnover and morphology may also be linked to insulin resistance through adipose inflammation (7).

Ahima and Lazar point out that weight loss alone may not reduce cardiovascular disease (8). Similarly, a hallmark study found no improvement of metabolic profile after removing large amounts of adipose tissue from the abdominal wall of obese patients (9), further implying that the quality rather than the amount of adipose tissue is important. Perhaps an improvement of metabolic profile is partly dependent on remodeling of adipose tissue from hypertrophy to hyperplasia rather than weight loss alone. Targeted studies of potential effects of weight loss on fat cell morphology and turnover may reveal further links between adipose tissue composition and metabolic risk factors.

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## Letters to the Editor

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Sensing temperature  
for flowering

566

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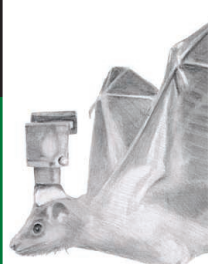
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## Emerging Arsenic Threat in Canada

L. RODRÍGUEZ-LADO *ET AL.*'S GEOSTATISTICAL model ("Groundwater arsenic contamination throughout China," Reports, 23 August, p. 866) has revealed the hidden risk of arsenic exposure in drinking water, particularly in China. The accompanying Perspective by H. A. Michael ("An arsenic forecast for China," p. 852) highlighted the global burden of arsenic-contaminated drinking water and the potential utility of Rodríguez-Lado's model to identify populations exposed to arsenic before the development of symptoms.

In Canada, arsenic is not currently a primary public health concern. However, we would like to point out the urgency of this emerging threat. More than a quarter of a million tons of arsenic generated over 50 years during the operation of the Giant Mine in Yellowknife (Northern Canada) reside underground and in piles aboveground. Because cleanup is deemed too great a health risk, the arsenic will be frozen in the ground, at a cost of nearly one billion Canadian dollars (1). In almost every province, arsenic-contaminated drinking water is a growing health concern in Canada. From Nova Scotia to British Columbia, arsenic levels frequently exceed those deemed safe by the World Health Organization (WHO) (2). Tools such as those described by Rodríguez-Lado *et al.* will not only help identify areas to prioritize for immedi-



Neurobiology  
Prize Essay

573

ate action in Canada and around the world, but also encourage public health agencies to monitor those already chronically exposed.

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## LIFE IN SCIENCE

### Zombiology

I step behind the podium and announce, "Today is zombie biology."

The students look up.

"First things first: What would cause a person to turn into a zombie?"

A few hands rise. "Radioactivity," a student says. "A disease," offers another.

"Excellent. And how would this disease be transmitted?"

Question after question, more and more students join in. Facing a zombie outbreak, how should institutions react?

How would survivors organize? Would they be able to carry on as before, or do careers and mortgages become secondary concerns when a horde of walking dead approaches? Would governments still manage to protect individuals, or should everyone take their safety into their own hands?

Finally, one last question: "When you get home," I say, "think about whether this class was really about zombies. See you next week!"

My "zombie biology" lecture is fictional, but zombie classes have begun to emerge (1, 2). The course descriptions are reassuring enough: Learn how to build a shelter, what kind of food to keep at the ready, and how to lead a group of survivors. Zombie apocalypse prevention may prove useful. Perhaps those who know how to survive a zombie outbreak won't panic when word spreads of a bad strain of the flu or a radioactive leak.

Even so, we need to tread carefully. We don't want students leaving the class convinced that zombies are real. Inviting zombies into a class may be a risky initiative—but if the students survive it, the results could be scarily effective.

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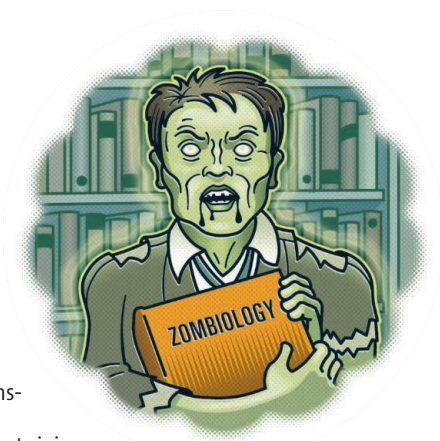
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## CORRECTIONS AND CLARIFICATIONS

**Reports:** "Representation of three-dimensional space in the hippocampus of flying bats," by M. M. Yartsev and N. Ulanovsky (19 April, p. 367). In Fig. 1, some graphic elements were misaligned. The HTML and PDF versions online have been corrected.

**Reports:** "Declining coral calcification on the Great Barrier Reef" by G. De'ath *et al.* (2 January 2009, p. 116). The Report cited a 14.2% decline in coral calcification from 1990 to 2005 based on analyses of 328 *Porites* colonies from the Great Barrier Reef (GBR). The authors now note that some of outermost annual growth bands were incomplete, and thus their estimate of the magnitude of the calcification decline was too high. They have adjusted for the incomplete outermost bands, and the decline in calcification for the period 1990–2005 drops to between 11.4 and 9.0%, depending on the method used. The most efficient adjusted estimate shows a decline of 11.4% [95% CI = (10.4, 12.4)]. The revised estimate of 11.4% still suggests a bleak future for corals of the GBR due to climate change.



#### EDITOR'S NOTE

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## Corrections and Clarifications

*Science* **342** (6158), 559.  
DOI: 10.1126/science.342.6158.559-b

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