The emergence of brown adipose tissue (BAT) as a potentially major player in our efforts to combat obesity and metabolic diseases in humans has stirred high hopes from the optimists and skepticism from the pessimists. Although for decades many had insisted, without success that adult humans have active BAT stores [1], the presence of BAT in humans was recognized in 2007 [2] and the scientific community unequivocally accepted the presence of metabolically active BAT in humans, only recently [3]. Since then, the epidemiology and physiology of human BAT is under intense investigation. Many sessions in scientific conferences have been devoted to BAT metabolism and at least two international research conferences focused specifically on BAT metabolism are scheduled for 2012. So, what is the significance of BAT in weight regulation and metabolic control in humans?

The very limited available data on BAT prevalence and metabolism in humans is presented in this issue of Current Opinion in Clinical Nutrition and Metabolic Care in the very elegant reviews by van Marken Lichtenbelt and Wu. The most common method to activate BAT is by exposing humans to cold. Mildly cold temperatures, above the shivering threshold, are apparently sufficient to induce BAT activation. However, the studies so far indicate that although active BAT appears to be present in most adult humans, older adults (>65 years) and the morbidly obese do not consistently show BAT activity when exposed to mild cold for 2–3 h. Are these groups lacking BAT tissue or are the protocols used not able to activate their BAT? Is the rise in middle-age obesity and insulin resistance a consequence of decline in BAT? Can we increase BAT mass in humans? Is adipose tissue adaptable in humans as it is in rats? Can white fat turn into brown fat? If yes, what does it take to achieve this conversion? How long does it stay brown before reverting back to the white phenotype? New, exciting evidence has started to emerge in the area of ‘browning’ of fat [4]. However, to date these questions remain unanswered.

The fact that activated BAT consumes energy, thereby increasing resting energy expenditure (REE), has led to claims that if we could activate BAT, with lifestyle, pharmacologic or other perturbations, this could, in the long-term, result in negative energy balance and weight loss. Therefore, BAT could play a significant role in our efforts to prevent and treat obesity. Although theoretically sound and proven in laboratory animals, this notion should be tested in real life situations in humans before we can claim, once again, that we have found the magic bullet to treat obesity. Many questions remain. How often and how long do we need to activate BAT in order to see a meaningful effect on energy balance? Will the increased REE lead to negative energy balance or increased appetite that cancels out any positive effects on weight? Remember that exercise, even though it burns calories, does not lead to significant weight loss. And even if we see a decrease in weight, will it be permanent or will weight go back up again, even if we keep activating BAT, as is the case with calorie restriction dietary regimens? Long-term clinical studies are needed to provide answers to these and many related questions.

Focusing on weight regulation may, once again, distract us from the real problem. For the past 40 years we have been trying to manage people’s weight and make everyone fit in the 20–25 BMI range. The fact that, after years of research and billions of dollars to combat obesity, our progress, still disappointing to say the least, should have forced us to refocus our attention to what really counts: the person’s health, not his looks. It is clear that obesity is not necessarily unhealthy and, on the contrary, being of ‘normal’ weight does make one immune to metabolic diseases. It is insulin resistance, dislipidemias, hypertension and fatty liver that should be the target of our efforts, irrespective of BMI. In this effort, BAT may have a role, as does...
exercise, even in the absence of any effect on weight. During cold exposure, glucose uptake by BAT increases 12-fold in humans [5]. In rats, cold exposure leads to increased triacylglycerol uptake by BAT [6]. Are these changes clinically significant? Can they improve insulin sensitivity and lipemia in humans? How often and for how long should BAT be activated to maintain these changes? How long do they last? Questions, questions, questions. We need to provide answers as soon as possible. Millions of people, obese and ‘normal,’ with metabolic syndrome are waiting.

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Conflicts of interest

There are no conflicts of interest.

REFERENCES