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EDITORIAL

Defining obesity as a disease

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Whether obesity should be declared as a disease is controversial.[1–4] Very recently, the World Obesity Federation argued that 'obesity was considered as a chronic, relapsing, progressive, disease process' that requires intervention.[4] By contrast, although the biological basis of obesity (e.g. so-called obesity genes, biology of fat cells) and pathological changes associated with the disease process have been characterized, obesity was not declared as a disease because there is no scientifically applicable definition of a disease.[3] Taking a public health point of view, many authorities again argued in favor of obesity as a non-communicable disease resulting from environmental drivers and host responses.[5] Finally, considering the benefits and harms arising from declaring obesity as a disease (i.e. taking an utilitarian point of view) also gave evidence to declare obesity as a disease.[3] It was assumed that the disease label would provide more benefits than harms to the general population, e.g. by the provision of more resources for novel and effective prevention and treatment of obesity.[4] There is however no evidence for the latter idea, i.e. the utilitarian approach is speculative.

Most of this scientific and public debate has been led in the US. Vallgarda *et al.* asked whether Europe should follow the US to declare obesity as a disease.[6] The authors addressed the utilitarian argument at two levels: (1) the legal and political level related to prevention and treatment of obesity; (2) the psychological and social level related to stigmatization and self-esteem. When compared with the US, health promotion, prevention and health care are already legal obligations of European states. Anyhow there is still a lack of attention to the obesity issue (e.g. by medical doctors and politicians) although obesity had been already included into the classification of diseases. At the psychological level, declaring obesity as a disease may affect the moral responsibility of obese patients; it may also give them a disability label and thus may add to discrimination. Taken together, the authors concluded that in a Western European welfare state a disease label will neither improve access to treatment and prevention nor provide a better protection of obese subjects. To summarize, the authors (i) questioned the utilitarian argument raised by the World Obesity Federation[4] and (ii) followed that obesity should be treated as a risk factor rather than a disease.

One of the major criticisms against defining obesity as a disease is its definition ad diagnosis. Obesity has been defined as an 'abnormal and excessive fat accumulation that may impair health'.[7] In practice, obesity is diagnosed by body mass index (BMI), which is taken as a surrogate of percentage fat mass. However, BMI has some obvious limitations related to the assessment of fat mass as well as the diagnosis of overweight- and obesity-related disturbances.[8–14] BMI was introduced into research and clinical practice on the basis of the association between BMI and mortality (which is U- or J-shaped, with minimal mortality toward the middle of the distribution), with a 'healthy' BMI range associated with the lowest mortality, which is in the range between 18.5 and 25 kg/m^2. This range varies, for example, by age, ethnicity and chronic diseases. Then, BMI values exceeding 25 kg/m^2, i.e. between 25 and 29.9 kg/m^2 and above 30 kg/m^2, were defined as overweight and obese, respectively. Obviously, obesity is defined based on statistical criteria, which may not have a biological meaning.

BMI is a score rather than objectively measured fat mass (or fat mass-related mechanical and metabolic disturbances). Neither is it biologically sound nor does it reflect a suitable phenotype worthwhile to study.[12] In fact, detailed analyses revealed considerable inter-individual variances in the associations between BMI and either subcutaneous adipose tissue (SAT) or visceral adipose tissue (VAT) or skeletal muscle mass or biomarkers of insulin resistance and inflammation or the adipocyte secretory activity (Figure 1). It is obvious that BMI can define neither 'excessive fat accumulation' nor functional impairments related to it. Consequently, Sharma *et al.* proposed a re-definition of obesity based on the health status of the individual.[14] The latter is characterized by clinical assessment, laboratory and endocrine testing, as well as detailed body composition analysis. As Sharma *et al.* noted themselves,[14] it still remains to be proven that even in the case of a detailed assessment possible health deficits identified have to be related to excess fat to provide a rationale of specific obesity treatment strategies.

Taken together, the issue whether obesity should be declared a disease is not trivial at all. US advocates mostly refer to the utilitarian argument.[2,4] Taking a European point of view, the authors of the present paper[6] question the disease label. This position is in line with evidence from public health research suggesting that 'obesity is the result of people responding normally to the obesogenic environments they find themselves in'.[5] Thus, obesity cannot be declared as a disease. Finally, the definition of obesity based on BMI alone is weak. Faced with the public health issue of obesity as well as the high activities in biomedical research on genetics, molecular and cellular biology, metabolism, endocrinology, microbiology and psychology of obesity, we should be open minded that a weak definition of obesity does not only question its status as a disease but also adds to explain the limited success rates in research, treatment and prevention of obesity. Since obesity research still provides more speculations rather than solutions, there is an obvious need of a self-critical discourse within our scientific community.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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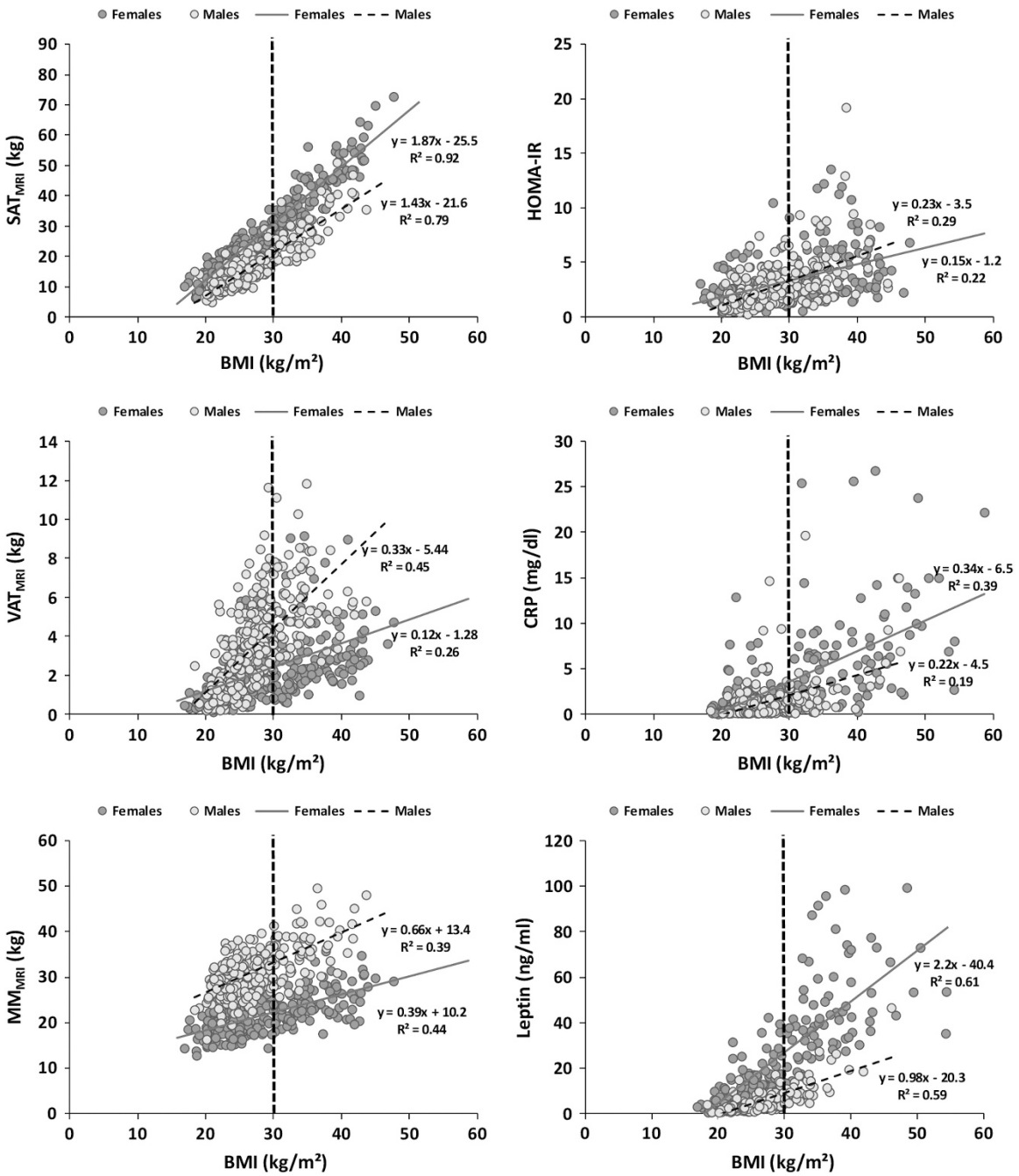

Figure 1. Associations between BMI (x-axis) and subcutaneous adipose tissue (SAT), visceral adipose tissue (VAT), skeletal muscle mass (MM), insulin resistance (as assessed by HOMA-IR), plasma levels of high sensitive CRP (as a marker of inflammation) and leptin (as a marker of adipocyte secretory activity). Detailed body composition was performed by whole-body magnetic resonance imaging (MRI) in 764 healthy Caucasian subjects (53% females; mean age 40 years with a range from 18 to 82 years). For reference and further details of the protocol, see Geisler *et al.*[15,16]

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