rects excess fat primarily to central depots, and in addition increases the turnover of these depots. This is amplified by a parallel activation of the main trigger of lipolysis in humans, the sympathetic nervous system. The result will be increased concentrations of NEFA, providing substrate to local tissue pools, where excess fat oxidation is interfering with insulin effects. In this way the since long established association between obesity and insulin-resistance diabetes seems to be understandable.

The semantic question posed by Sharir and Raz [1], requires a final comment. It is very difficult to change an established name of a prevalent disease, a name that has been with us for a long time. Nevertheless, their suggestion is logical. The original name, diabetes mellitus was probably also selected to separate this condition from diabetes incipidus, a problem of minor importance. A suggestion here might be to just skip the "mellitus" and call the condition "diabetes", divided into Type 1 or 2, or whatever new types of diabetes might be discovered.

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Author's reply

To the Editor: Dr. Per Björntorp rightly confirms our thesis that the basic metabolic derangements leading to diabetes originate in aberrations of lipid rather than carbohydrate metabolism and the augmented fat oxidation may be the key to understanding the development of diabetes.

He emphasises that obesity is the dominant factor leading to diabetes, maintaining that central rather than peripheral obesity is more closely associated with insulin resistance. Rightly so, this factor, often neglected in the understanding of the pathogenesis of Type 2 diabetes, most probably induces an increased mobilisation of fatty acids from central adipose tissue and accelerates fat oxidation by central viscera. However, even if the lipid supply to skeletal muscles may be a sequel of central obesity, the problem of hormonal regulation leading to preferential central fat deposition and release has to be fully resolved. The very fact of the increased size of abdominal adipocytes and the increased mass of the abdominal fat depot seems to enhance the fatty acid turnover along with their release. The activity of several lipid mobilising substances secreted from central adipose tissue can differ from those of peripheral adipose tissue and would be of major interest to know how to counteract their action and use this knowledge for the treatment of diabetes.

As explained in our article, we stress that the ectopic intramyocellular fat deposition is committed to eliciting insulin resistance, not necessarily directly related to obesity. It has yet

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to be determined whether the traffic of newly ingested fat is directed to muscles rather than to peripheral or central adipose tissues, or if it is following the growth of adipose tissues. Also, it is well known that in many cases obesity is secondary to insulin resistance rather than preceding it and muscle fat increment is likely to occur prior to obesity.

The accumulation of diacylglycerol in muscles as a result of increased triglyceride and fatty acyl-CoA turnover (during augmented local triglyceride synthesis and breakdown) leads to a negative loop in insulin signal transduction. This is due to PKC activation and alteration in components of the insulin signalling pathway by multisite serine phosphorylation. It remains to be fully elucidated how the muscle fat deposition is related to obesity, particularly abdominal fat accumulation, or how it is brought about by other hormonal factors and cytokines, including those secreted by adipose tissue itself. It is also important to establish what factors are responsible for the ineffective arrest of lipolysis in the central, compared with peripheral depots. It should also be remembered that long-term insulin resistance, associated with glucose intolerance, can involve fat inflow into beta cells with resultant secretory impairment.

With regard to the revision in diabetes nomenclature, we welcome the support of Dr. Björntorp for dropping the adjective "mellitus" from the name of diabetes as a first step. It is sufficient for now that we diagnose Type 1 and Type 2 diabetes without additional labels. These are markedly different diseases and require an exact definition. However, in the case of Type 2 diabetes, the development of which is overtly associated with deviations in lipid metabolism, the adjective "lipidus" appears to be justified. This should remain a subject of current debate and decision by international diabetes institutions.

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