In insulin resistance and obesity in a mouse model of systemic lupus erythematosus

To the Editor:

We have read with great interest the article by Ryan et al. We agree with the authors that the NZBWF1 strain may be an important model to study the effects of obesity and insulin resistance on systemic lupus erythematosus (SLE)-associated hypertension. The authors have observed that this hypertensive mouse model of SLE has an increased body weight, central adiposity, plasma leptin, and insulin resistance. The relationship between SLE and insulin resistance is not clear. As suggested by the authors, the autoimmune pathogenesis of insulin resistance in subjects with SLE is rare. Lagana et al. have shown that an autonomic dysfunction characterizes SLE patients with and without microvascular disease. We suggest a possible role for mechanisms as the activation of the autonomic nervous system in the development of hypertension and insulin resistance and in the change in body composition in SLE patients. The autonomic nervous system modulates glucose and fat metabolism. Recently, a prospective cohort study revealed a high relative risk to develop type 2 diabetes if autonomic dysfunction is present in healthy subjects independent from other risk factors. Kreier et al. propose an unbalanced and arrhythmic autonomic nervous system as a major cause of the metabolic syndrome, and, in a previous study, we propose that an impairment in the autonomic nervous system activity could precede the development of insulin resistance and type 2 diabetes mellitus. In conclusion, we hypothesize that the SLE is characterized by an autonomic dysfunction that could affect the glucose metabolism, and it could cause insulin resistance and change in body composition in these patients. We suggest that the authors test this hypothesis.

Disclosures

None.

Antonio Perciaccante
Alessandra Fiorentini
Luigi Tubani
Department of Clinical Medicine
University "La Sapienza"
Rome, Italy

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Antonio Perciaccante, Alessandra Fiorentini and Luigi Tubani

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