CLINICS

READER'S OPINION

Differences in the mechanisms that induce obesity and metabolic syndrome in experimental animal models and humans may cause treatment failure

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Dear Editor,

We recently read the article ''Monosodium glutamate neonatal treatment induces cardiovascular autonomic function changes in rodents'' by Signora Peres Konrad et al. with great interest(1). These authors concluded that obesity induced by neonatal monosodium glutamate treatment impairs cardiac autonomic function and most likely contributes to increased arterial pressure and insulin resistance in rats. The authors are to be commended for contributing such a well-designed and successfully presented study. We believe that these findings will guide further study of obesity and metabolic syndrome (MS).

Obesity is a growing public health problem, and metabolic syndrome is an important risk factor for cardiovascular diseases (2,3). Although it is correct that this study should be emphasized in terms of these factors, we believe that obesity and MS induced in rats with monosodium glutamate may differ from any hereditary or acquired obesity and from MS in humans, i.e., that the enzymatic pathways utilized by and the physiologic effects resulting from these two entities may be different. These differences may affect treatment success, and we believe that the study results should be assessed with this perspective in mind.

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