Reply to Dr. Götz and colleagues

Luca Mascitelli · Francesca Pezzetta ·
Mark R. Goldstein

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We agree with Götz and colleagues that a similar role for iron in type 2 diabetes mellitus and Alzheimer's disease could also extend to a general phenomenon of altered biometal stasis. Indeed, it has been shown that copper, iron and zinc are increased in senile plaques in AD [1]. In view of reports of increased aggregation of amyloid β peptide in vitro in the presence of these elements, it is possible that they play a role in the increased aggregation of amyloid β peptide in senile plaque formation in AD brain, thus enhancing the genesis of senile plaques. The increase in iron and copper could also serve as a catalyst for increased free radical formation which might be important in the toxicity of amyloid β peptide.

However, among the strategies aimed at biometals depletion, phlebotomy, which effectively reduces body iron stores, is the safest and easiest way to reduce a biometal [2].

References

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L. Mascitelli (✉)
Medical Service, Comando Brigata alpina "Julia", Udine, Italy
e-mail: lumasci@libero.it

F. Pezzetta
Cardiology Service, Ospedale di Tolmezzo, Tolmezzo, Italy

M. R. Goldstein
Fountain Medical Court, Bonita Springs, FL, USA