Serum Neuron-Specific Enolase in Lead-Exposed Individuals

Dear Editor,

The article by Ravibabu, et al., provides important new data in the field of biomarkers to monitor exposures to lead within the workplace, as in the lead acid battery manufacturing. They found that the serum neuron-specific enolase (NSE) levels are not elevated in association with concentrations of blood lead levels among workers in the lead acid battery manufacturing plant. In our (unpublished) cases we also found that serum NSE levels did not correlate with changes in lead concentrations in whole blood among individuals with potential exposure to lead. However, we would like to clarify some of the issues raised by Ravibabu and colleagues. In the introduction section, they state that “the levels of serum NSE were associated with exposure to solvents, chromium, 1-bromopropane, 2,5-hexanedione...” However, if we are correct, serum NSE levels is likely to be a more effective biomarker for mercury toxicity than chemical substances that the authors cite (ie, solvents, chromium, 1-bromopropane, 2,5-hexanedione). The authors acknowledge this point in their Discussion of the clinical evaluation of serum NSE levels as a marker of traumatic brain injury, silicosis, mercury intoxication, pneumoconiosis, and in chromium-exposed as well as carbon monoxide-exposed persons. We were surprised, however, that there is no mention in their Introduction, or in the Discussion section, of the role of serum NSE as a biomarker of effects associated with mercury poisoning that was first reported and suggested by Benz, et al., and our report on four patients with mercury overexposure, subsequently published, shows that there is an association between mercury exposure and levels of serum NSE, which is a good indicator of alterations in the central as well as the peripheral nervous system cellular structure and biochemical processes. Serum NSE seems to be a promising biomarker of effects for mercury exposure in humans. Future studies evaluating the effects of mercury exposure on serum NSE are needed.

Conflicts of Interest: None declared.

Funding Source: None.

PD Pigatto¹, A Ronchi², M Pontillo³, G Guzzi⁴*

¹Department of Biomedical, Surgical and Dental Sciences, Unit of Oral Pathology and Medicine, IRCCS Galeazzi Hospital, University of Milan, Milan, Italy
²Pavia Poison Control Center and National Toxicology Information Centre, Toxicology Unit, IRCCS Maugeri Foundation and University of Pavia, Italy
³Laboratory Medicine, “San Raffaele” Hospital Scientific Institute, Milano, Italy
⁴Italian Association for Metals and Biocompatibility Research – A.I.R.M.E.B., Milan, Italy
E-mail: gianpaolo_guzzi@fastwebnet.it

Cite this article as: Pigatto PD, Ronchi A, Pontillo M, et al. Serum neuron-specific enolase in lead-exposed individuals. Int J Occup Environ Med 2016;7:58-60.
Dear Editor,

We sincerely appreciate efforts by Pigatto, et al., for their comments on our article\(^1\) and for validating our study findings regarding non-elevation of serum neuron-specific enolase (NSE) levels in association with blood lead levels among the lead acid storage battery plant workers. To the best of our knowledge, studies on combined chemical exposures and serum NSE were not reported in the literature. Furthermore, in our work we primarily examined the relationship between occupational exposure to lead and NSE and not exposure to other chemicals/metals. Therefore, we had only limited consideration of the possibility of NSE as a biomarker of exposure to other chemicals. The referenced articles\(^2-4\) have been published very recently and thus were not reviewed in the Introduction and Discussion of our article.

Regarding the Pigatto and colleagues’ statement that “serum NSE seems to be a promising biomarker of effects for mercury exposure in humans,” the number of systematically conducted studies supporting this view point is very limited, however there are few early studies pointing towards this direction. Study conducted by Yilmaz, et al.,\(^5\) reported elevated levels of serum NSE in children exposed to elemental mercury presented with neurological symptoms compared to asymptomatic children. However, the findings of the study were not significantly different among the subgroups. Benz, et al.,\(^6\) in a case report presented brain MRI findings in a child exposed to mercury but could not find any association between mercury and serum NSE. Furthermore, an editorial by Costa, et al.,\(^7\) on the above article just mentions about the authors anecdotal experience and not a systematically conducted research, thus limiting its scientific validity. Well-designed studies are required to establish the serum NSE as a potential biomarker of neurotoxic effects of occupational mercury exposure in humans.

Conflicts of interest: None declared.

K Ravibabu

Scientist-B (Biochemistry)
Regional Occupational Health Centr (southern)
Indian Council of Medical Research
ICMR Complex, Devanahalli, Bangalore-562110
Karnataka, India
E-mail: kalahasthi20012002@yahoo.co.in

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