A reply to the commentary of Lehmann et al.

Avital Schurr and Benjamin M. Rigor

Department of Anesthesiology, University of Louisville School of Medicine, Louisville, KY 40292, USA

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In their commentary, Lehmann et al. tried to refute our hypothesis regarding the role of taurine in protecting cerebral tissue against ischemic/hypoxic damage [1]. They claim that in their own studies taurine did not protect immature rat hippocampal slices from such damage. They also point out our misinterpretation of their own results [2], namely, that brain taurine levels are remarkably stable during and after ischemia.

We agree with Lehmann et al. that there is no evidence regarding a net increase in taurine synthesis during cerebral ischemia and thus, its questionable role in free radical scavenging. However, these investigators themselves showed that extracellular taurine increased manifold upon cerebral ischemia, by changing the intra- to extracellular distribution [2]. We believe that their results are in complete harmony with our hypothesis; an increase in the extracellular level of taurine should provide the necessary protection against ischemic damage by attenuating Ca^{2+} influx. It is difficult to comment on their study (in preparation), although it is possible that the immature rat hippocampal slices they used were already enriched with high levels of taurine, such that an exogenous supplement of taurine made no difference. In a recent study, Hagberg et al. [3] showed an excessive release of taurine in the cortex and basal ganglia of fetal lambs during hypoxia-ischemia. In this paper they suggest that such extracellular overflow of taurine, on one hand and the attenuation of both ischemia- and excitotoxin-induced Ca-entry, on the other, account for the higher resistance of immature brain to hypoxic insults. Recently, Oja and Kontro [4] reported on the K^+- and Ca^{2+}-stimulated release of taurine, a release which could be antagonized by Mg^{2+} [4] and the calcium blocker verapamil [5]. Walz and Allen [6] also reported on the osmoregulatory function of taurine in brain cells and Hanretta and Lombardini in a recent review [7] concluded that taurine is both a Ca^{2+} modulator and a membrane stabilizer.

Hence, we cannot agree with the conclusion of Lehmann et al. about our hypothesis. The established facts, including their own findings, surely suggest a protective role for endogenous taurine against cerebral ischemic damage. As to the higher resistance of immature mammals to ischemia/hypoxia, our hypothesis associating this phenomenon with the very high levels of taurine in their brains is valid until proved otherwise.

Correspondence address: A. Schurr, Department of Anesthesiology, University of Louisville School of Medicine, Louisville, KY 40292, USA

REFERENCES

[1] Lehmann, A., Hagberg, H., Andinè, P. and Ellrèn, K. (1988) FEBS Lett. 233, 000–000.
[2] Hagberg, H., Lehmann, A., Sandberg, M., Nystrom, B., Jacobson, I. and Hamberger, A. (1985) J. Cereb. Blood Flow Metab. 5, 413–419.
[3] Hagberg, H., Andersson, P., Kjellmer, I., Thiringer, K. and Thordstein, M. (1987) Neurosci. Lett. 78, 311–317.
[4] Oja, S.S. and Kontro, P. (1987) J. Neurosci. Res. 17, 302–311.
[5] Schramm, M. and Towart, R. (1985) Life Sci. 37, 1843–1860.
[6] Walz, W. and Allen, A.F. (1987) Exp. Brain Res. 68, 290–298.
[7] Hanretta, A.T. and Lombardini, J.B. (1987) Brain Res. Dev. 12, 167–201.

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