Review of PhD thesis
Extracellular space diffusion parameters and metabolism in the rat somatosensory cortex during recovery from transient global ischemia and hypoxia
by Norbert Zoremba

PhD thesis "Extracellular space diffusion parameters and metabolism in the rat somatosensory cortex during recovery from transient global ischemia and hypoxia" is based on four full-length papers in prestigious international journals (sum of impact factors of these four papers is 15.183) what means that the data already passed thoroughful peer review process. Data presented in these PhD thesis are very important for pathophysiology of ischemia and hypoxia and it shows directions for further studies in this field.
Thesis have classical chapters – Introduction (18 pages), Methods (9 pages), Results (19 pages), Discussion (14 pages) and Conclusions (2 pages). References are numerous, appendix is formed by reprints of the original papers. The proportion of indiv idual parts of the thesis is well equilibrated and the thesis is very clearly written and easy to read.
Introduction presents all necessary data about measurement of extracellular space and details of all calculations. Methods and results have the right si ze to be fully informed. Discussion exactly corresponds to the style of the whole thesis.

I have three questions and some minor points:

1. What is the significance of the late changes? There are many examples of these changes clearly seen in Figures: Overshoot of alpha after the end of ischemia -hypoxia (unilateral clamping of a .carotis) – Fig.6; lactate and glutamate in Fig.7 (even 90 min after the end of hypoxia are their levels higher than before insult) ; see also Fig.8, 15-min ischemia.
2. How to explain the lack of correlation between DC level and extracellular potassium activity during reperfusion in Fig.10, 15-min ischemia (DC potential is at first more negative, then there is an overshoot to values more positive than preischemic ones) .
3. I am missing a correlation of data with EEG activity in the model of epileptic seizures. EEG was recorded in the paper in Experimental Neurology. The time of normalization of bioelectric activity is important therefore I would like to ask for how long time was it possible to see pathological EEG activity.

Minor points:
P.5, l.7: Angati et al. – right: Agnati
P.23, ll.9-7 from bottom: Depth 1200-1500 µm probably represents layer V, not IV and V

P.26, figure 1B: Both abscissa and ordinate are not sufficiently described
P.34, ll.12-13: The difference between control and hypoxic animals was ***extremely*** significant. The difference either is or is not significant; the word “extremely” is in this connection a nonsense.
P.36, para 3.2. A decrease in oxygen supply should be menti oned (as in methods).
P.40, para 3.3. Again a decrease in oxygen supply should be mentioned (as in methods).
P.54, l.7: Sykova et al. 2005 is not in References .
P.54, l.8: It is not epilepsy, but pharmacologically induced epileptic activity (single epileptic seizure, probably SE).
Statistical significant differences are not marked in all figures, it is only exceptional .
One formal but important comment: References are listed in an uncommon manner. If one author is in question (e.g. Sykova in this thesis) nearly all journals are asking to put at first papers with one author, then papers with two authors and at least papers with more than two authors. Chronological order is secondary in these different parts.

My comments do not decrease the value of this thesis. It is from both factographic and formal points of view fulfilling all conditions for PhD thesis and I propose to give the author (after successful defense) a title Ph.D. according to the laws of the Czech Republic.

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