CEFTRIAXONE ACTIVATES BRAIN GLUCOSE TRANSPORT THROUGH INCREASE OF GLUTAMATE UPTAKE

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Introduction: Glucose constitutes the major source of energy in the brain, with its utilization rate correlating with neuronal activity, however, contributions of glial cells to neuroenergetics have been the focus of extensive debate. For over 30 years, brain [^{18}F]FDG PET uptake has been viewed as a proxy of neuronal activity, and despite widespread use in both clinical settings and basic research, the identity of the cell type(s) contributing to the [^{18}F]FDG PET signal, as well as the mechanisms regulating its variations, remain highly controversial.

Methodology: we conducted a micro-PET study using [^{18}F]FDG to assess whether ceftriaxone, a known stimulator of astrocytic glutamate transport via GLT-1, was capable of modulating cerebral [^{18}F]FDG consumption in awake adult rats. We also tested this hypothesis in adult astrocyte cultures (30059).

Results: Here we provide positron emission tomography and cells culture evidence that activation of astrocytic glutamate transport via the excitatory amino acid transporter GLT-1 triggers widespread but graded glucose uptake in the rodent brain.

Conclusions: Our results highlight the need for a reevaluation of the interpretation of [^{18}F]FDG positron emission tomography data, whereby astrocytes would be recognized as contributing to the [^{18}F]FDG signal.

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