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Sommario/riassunto	One research field that early recognized the importance of intercellular interactions was endocrinology, initially in processes involved in lactation, pubertal maturation and regulation of the female ovarian cycle and later in appetite regulation. These interactions included, but were not restricted to neuronal-astrocytic interactions. The importance of glutamatergic and GABAergic signaling during all of these events is now realized. At the same time huge advances have been made in i) determination of metabolic rates in the human and rodent brain in vivo, including oxidative metabolism rates in astrocytes which per volume are at par with those in neurons; ii) understanding the unique ability of astrocytes, but not neurons to synthetize tricarboxylic acid intermediates necessary for net synthesis of glutamate and thereby also GABA; iii) determination of the rates at which such synthesis occurs, and iv) the two-fold higher rates at which glutamate and GABA are cycled between astrocytes and neurons in the brain in vivo. This

quantitative difference reflects that most transmitter uptake, especially that of glutamate, occurs in astrocytes and that on average two thirds of astrocytically accumulated neuronal transmitters are recycled to neurons, whereas the last one third is oxidatively degraded, mainly or exclusively in astrocytes. The progress in these areas puts emphasis on i) firmly establishing whether or not aralar, a necessary component of the aspartate/glutamate exchanger in the malate-aspartate cycle is expressed in astrocytes, and ii) the detailed processes occurring in astrocytes and in neurons during the formation and subsequent oxidative degradation of transmitter glutamate and GABA. Initial observations by different groups showed no astrocytic aralar expression in mature brain. However, a recent paper by Pardo et al. (J. Cereb Blood Flow & Met.) used improved cytochemical techniques and showed some protein expression in astrocytes in mature brain; Hertz (same journal) calculated that the amount would be sufficient for normal oxidative degradation. However, there are indications that the astrocytic-neuronal-astrocytic interactions in formation, transfer and re-oxidation of transmitter glutamate and GABA may repeatedely require additional MAS function. Equal expression of aralar mRNA has been shown by the Nedergaard group in neurons and astrocytes obtained by fluorescence-activated cell sorting of brain cells from mice co-expressing astrocytic and neuronal markers with different fluorescent signals. This has recently been confirmed and also shown to be the case for aralar protein (J. Neurochem, under revision).