Glutamate immunoreactivity of insular cortex afferents to the nucleus tractus solitarius in the rat: A quantitative electron microscopic study

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Abstract

Corticosolitary axons and their terminals were labeled by the anterograde transport of wheat germ agglutinin conjugated to horseradish peroxidase, after injections into the rat insular cortex. The ultrastructure of these cortical afferents was analysed in the medial and commissural subnuclei of the nucleus tractus solitarius. Cortical terminals had a mean area of 0.36 μm^2, and were among the smallest terminals in the nucleus. They made single, asymmetric synaptic contacts with thin dendritic stems or with spines. The average diameter of the dendrites postsynaptic to cortical axons was 0.59 µm, and significantly smaller ($P < 0.01$, Kolmogorov-Smirnov test) than the mean (0.87 µm) of the population of dendrites in the same region of the nucleus tractus solitarius. Cortical boutons contained closely packed round and clear synaptic vesicles of diameter ca. 28 nm, a few mitochondria, and no dense core vesicles. Postembedding immunogold analysis showed that the anterogradely labeled cortical axon terminals were immunoreactive to glutamate, but not to GABA. Cortical afferents had no average four times the glutamate immunoreactivity (assessed by gold particle density) than local dendrites or terminals making symmetric synaptic contacts. Similarly, most of the unlabeled axon terminals participating in asymmetric synaptic contacts were highly enriched in glutamate immunoreactivity, suggesting that glutamate may be a most prevalent transmitter in the nucleus tractus solitarius. Terminals immunoreactive to GABA always made symmetric synapses, mostly with dendritic shafts and perikarya.

We concluded that insular cortex axons made single, asymmetric synaptic contacts with thin, probably distal dendrites in the nucleus tractus solitarius. Cortical terminals are immunoreactive to glutamate, and morphologically different from primary afferents and from terminals immunoreactive to GABA.