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IMMUNOCYTOCHEMICAL LOCALIZATION OF GLUTAMATE DECARBOXYLASE (GAD) IN OLFACTORY BULB

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Immunocytochemical localization of glutamate
decarboxylase (GAD) in the olfactory bulb.

Glutamate decarboxylase (GAD), the enzyme that synthesizes the neurotransmitter, γ -aminobutyric acid (GABA), has been localized in the olfactory bulb by immunocytochemical methods at the light and electron microscopic levels. The light microscopic results demonstrated GAD positive puncta throughout all layers of the olfactory bulb with the greatest concentration in the external plexiform layer. In addition, the cytoplasm of many neuronal somata in the granule and glomerular cell layers was GAD positive but not the cytoplasm of mitral cell somata. The GAD staining of these presumed granule and periglomerular neuronal somata also extended into their dendrites for many microns. This somal and dendritic staining has not been previously described for neurons such as the basket, stellate and Purkinje cells of the cerebellum, even though their axon terminals are known to be GAD positive.

The electron microscopic observations confirmed the presence of GAD positive reaction product within the cytoplasm of granule and periglomerular neurons. Also, in the external plexiform layer, reaction product filled many of the granule cell gemmules which form reciprocal dendrodendritic synapses with mitral cell dendrites. When these gemmules were seen in continuity with their granule cell dendrites, the GAD positive reaction product stained the dendrites and the gemmule stalks, but to a lesser degree than that of the gemmules themselves. GAD positive cellbodies have also been observed in other CNS regions where dendrodendritic synapses are known to occur such as the lateral and medial geniculate nuclei and the superior colliculus. For this reason, it is suggested that somal and dendritic GAD may be detected by current immunocytochemical methods only within neurons that have presynaptic dendrites. The presence of GAD within granule and periglomerular cells provides further evidence that these inhibitory interneurons use GABA as their neurotransmitter. (Supported by USPHS grants #NS-12116 and #NS-1615)