Summary

This thesis investigates odor processing in the mushroom body (MB) of the honeybee *Apis mellifera carnica*. Using Ca^{2+} imaging of selectively labeled neurons, fundamental differences in odor coding between the MB and the antennal lobe (AL) were found, and evidence for associative plasticity in the MB intrinsic Kenyon cells (KC) was discovered. These results allow conclusions about the interplay of odor coding and neural plasticity in the context of honeybees' odor discrimination and learning capabilities.

Chapter I addresses the function of the MB in odor coding. Odor-evoked network activity was characterized at three consecutive neural compartments: first, in the dendrites of the projection neurons (PN) that connect the AL with the MB, next, in the presynaptic terminals of these PNs (boutons), and finally, in their postsynaptic partners, the clawed KCs (cKC). Odors evoked combinatorial activity patterns at all three processing stages, but the spatial patterns became progressively sparser along this path. PN dendrites and boutons showed similar response profiles, but PN boutons were more narrowly tuned to odors. The transmission from PN boutons to cKCs, was accompanied by a further sparsening of the population code. Activated cKCs were highly odor specific, distributed over the lip and not grouped into functional subunits as is the case for PNs in the AL glomeruli. Furthermore, cKCs integrated PN activity only within 200 ms and transformed complex temporal patterns into brief phasic responses. Thus, two types of transformations occurred:

- Population sparsening, depending on pre- and postsynaptic processing within the MB microcircuits.
- Temporal sharpening of postsynaptic cKC responses, probably involving a broader loop of inhibitory recurrent neurons.

Chapter II addresses the role of cKCs in odor learning by combining differential conditioning with Ca^{2+} imaging of cKC dendrites in the MB input region. Pairing an odor with sucrose induced a pronounced prolongation of and/or increase in odor responses without changing the ensemble of activated cKCs. 15 minutes after

training, the responses to the rewarded odor were enhanced, while the pattern of activated cKCs remained stable. The results demonstrate that cKCs are sensitive to the coincidence of odor and reward. Furthermore, they indicate that the formation of odor memories depends on the modulation of the cKC spiking activity, presumably caused by octopamine release from the VUMmx1 neuron, which represents the reinforcing function in appetitive odor learning.

The results from Chapters I and II were integrated in a functional model of the MB, which illustrates how the sparseness of the cKC code could be exploited by a learning mechanism.

Chapter III presents an *in vivo* preparation of the honeybee that allows the study of odor evoked activity in the MB with 2-photon laser scanning microscopy (2PLSM). This approach overcomes the limitations imposed by conventional fluorescence microscopy. The data presented show that 2PLSM can be used to study anatomy and neural activity at high temporal and spatial resolution in the honeybee brain.