

Proceedings of the Symposium on Brain Mechanisms of Taste Recognition Memory and Neural Plasticity Held at the Ninth European Congress of Psychology on 6 July 2005

Milagros Gallo

Institute of Neurosciences F. Oloriz, Department of Experimental Psychology and Physiology of Behavior, University of Granada, Granada 18071, Spain

Correspondence to be sent to: Milagros Gallo, Institute of Neurosciences F. Oloriz, Department of Experimental Psychology and Physiology of Behavior, University of Granada, Granada 18071, Spain. e-mail: mgallo@ugr.es

Research on the neurobiological substrates of taste-related learning and memory is a broad and rapidly evolving multidisciplinary field. Historically, research on taste aversion learning has contributed critically both to the behavioral and the neurobiological assessment of learning systems. Since the discovery by Garcia et al. (1955) of conditioned taste aversion and its peculiar features, such as one-trial and long-delay learning, taste aversion learning represented a challenge for learning theorists and contributed decisively to the development of modern learning theories. Also, the search for the brain circuit responsible of taste–illness association has revealed a complex interaction among a variety of areas located at different brain levels from brain stem to cortex. During the ninth European Congress of Psychology, a symposium was held where 5 speakers discussed the recent developments in the brain mechanisms of taste memory and neural plasticity. Their talks reflected how the research field has widened both in the types of processes and brain areas studied as well as levels of analysis aimed and technical approaches applied. First, an increased interest in the study of the neurobiological mechanisms not only of taste aversion but also of the safe taste memory was evident because the effect of nonrewarded taste exposure on habituation of neophobia and latent inhibition were issues of interest for the speakers. This is consistent with wider theoretical approaches to taste memory, such as that proposed by Bermúdez-Rattoni (2004). Also, from the speakers' talks, it became evident that the study of brain areas additional to those involved in taste and visceral processing may be necessary for a complete understanding of the neural plasticity required for taste memory. Second, research aimed not only at the system but also at the cellular and molecular levels of analysis was presented. Third, the range of approaches covered by the speakers included data from lesion, electrophysiological recording, immunoreactivity mapping, neurochemical stimulation, and blockade, as well as developmental approaches.

Federico Bermúdez-Rattoni opened the symposium offering a comprehensive review of the brain mechanisms underlying the neural representation of safe and aversive taste memory. He presented data supporting a role of cortical glutamatergic transmission and metabotropic glutamate receptors on taste aversion, taste neophobia, and attenuation of neophobia. His data suggest that the formation of the safe taste memory needs the temporal inactivation of the glutamatergic system during the first period of time after taste consumption.

Ilene Bernstein reviewed studies assessing gene expression in a brain-distributed system underlying taste, visceral, and associative processing. Differential gene expression as a function of taste novelty throughout the circuit subserving conditioned taste aversion was discussed. Based on her studies that examined the induction of fos-like immunoreactivity in response to taste preexposure, Ilene Bernstein proposed the synthesis and subsequent degradation of an immediate early gene such as *c-fos* as a potential molecular substrate for changes in conditioning strength over a range of intervals and pointed to central amygdala as a potential site for taste–visceral association relevant for the formation of conditioned taste aversions.

The talk by Takashi Yamamoto emphasized the separated amygdala substrates underlying the sensory and hedonic changes induced by taste aversion learning. The data presented suggest that the enhanced activation induced by the aversive taste originates in the central amygdala and the hedonic shift from appetitive to aversive originates in the basolateral amygdala. By reviewing electrophysiological and recent *c-fos* immunohistochemical studies, he presented a distributed neural system underlying conditioned taste aversion expression that includes the parabrachial nucleus, amygdala, insular cortex, supramammillary nucleus, nucleus accumbens, and ventral pallidum.

Milagros Gallo added a developmental approach to the study of taste memory. After reviewing data showing the

complex aging impact in a variety of taste learning phenomena, evidence on the role of the hippocampus in modulating some complex taste memories was presented. It was proposed that the aged hippocampus may be unable to support certain types of taste memory modulation because aged rats showed similar deficits to those seen in young-adult hippocampal-lesioned rats regarding the blocking effect and the contextual dependency of latent inhibition. However, an intact latent inhibition as well as an enhancement of taste aversion may indicate that aging induces a rather complex reorganization of the brain systems involved in taste memory, not exclusively explained by the hippocampal function decay.

Finally, Barbro Johansson reviewed recent research showing the effect of stimulation by both environmental enrichment and learning behavioral procedures on the hippocampal and olfactory system plasticity, either in intact or lesioned brains. She showed that a stimulating environment modifies cell proliferation, migration, and differentiation

patterns. The talk evidenced that the olfactory and taste systems may be excellent models for the study of neural plasticity underlying memory.

The present proceedings encompass 4 manuscripts covering the presentations on taste memory. This series of manuscripts present a comprehensive review of recent advances in the understanding of the taste memory systems. They show that the field is very active and it faces exciting challenges, being taste learning memory a privileged assay for neurobiological assessment.

References

- Bermúdez-Rattoni F. 2004. Molecular mechanisms of taste-recognition memory. *Nat Rev Neurosci.* 5: 209–217.
- Garcia J, Kimeldorf J, Koelling R. 1955. Conditioned aversion to saccharin resulting from exposure to gamma radiation. *Science.* 122: 157–159.

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