Discussion. As evidenced by Tm profiles shown in the Figure, fertilization results in a greater proportion of single stranded DNA. This phenomenon is accompanied by significant increase in free GA-like activity. In the light of the reports of FELLENBERG[10], BAMBERGER[11] and KESSLER and SNIR[12] that in vitro GA causes DNA 'opening', it is postulated that in the maize kernel the DNA Tm profile changes following fertilization are GA triggered. The observed effect may be the result either of direct binding of GA to DNA as reported above or, alternatively, by GA-induced enhanced membrane permeability which possibly provides 'unwinding' enzymatic agents greater accessibility to the DNA.

Comparison of endogenous GA-like activity, RNA and protein levels in *Zea mays cv* Jubilee kernels at successive developmental stages

Developmental stage	Free GA [a]	Glycosidic bound GA [a]	RNA content [b]	Protein content [c]
I	0.260	3.860	0.255	1.550
II	0.430	3.260	0.322	2.060
LSD $p < 0.05$	0.110	0.140	0.059	0.380

I and II – as in the Figure. Figures presented are 4 replicate means expressed as O.D. units at [a]560 nm, [b]259 nm and [c]660 nm as determined on a recording Unicam UV-spectoronic photometer Model SP 800 B.

The GA performing this function may stem from conversion of bound to free GA. The Table indicates that, with a decrease of the former, an increase of the latter is manifested. A further GA source, not necessarily excluding the above, may be the pollen, since BARENDSE et al.[13] have reported that pollen grains contain considerable amounts of plant growth hormones including GA. It thus seems that in the maize kernel the partial 'unwinding' of DNA may result in greater template activity and this may account for the observed increase in RNA and protein levels (Table).

These effects are primarily in the kernel tissue excluding the egg cell(s) and the ovule sac which are the site of fertilization, since preliminary determinations indicated that DNA content of the embryo (at either stage) was negligible as compared to that of whole kernels. Furthermore it has been shown[14] that during the initial stages following fertilization the developing ovule contains only a small number of nuclei. This implies a general stimulatory effect of fertilization taking place in a comparitively minute locus of generative tissue upon metabolism of somatic tissue.

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Effects of Denervation and Decentralization upon Taste Buds

J. A. DONOSO[1] and P. ZAPATA[2]

Departamento de Neurobiología, Universidad Católica de Chile, Casilla 114-D, Santiago (Chile), 25 August 1975.

Summary. Denervation of vallate papillae results in failure of tactile and gustatory reception at a time when impulse conduction in the distal stump of the glossopharyngeal nerve is still unimpaired; delay of receptor deficit depends on axon length between receptor and axotomy sites; taste buds disappear by 10 days. Decentralization, through intracranial rhizotomy, does not modify lingual receptors structure or function.

Taste buds differentiation and maintenance depend on their connection with gustatory nerves. Those buds of the vallate papillae are innervated by the glossopharyngeal nerves, and they degenerate shortly after nerve severance[3,4]. The present work was a study of some structural and electrophysiological changes in taste receptors after neural damage at different levels.

Experiments were performed on adult cats under pentobarbital anaesthesia. One or both glossopharyngeal nerves were transected either: a) at its immergence into the pharyngeal wall, b) at its exit from the jugular foramen, c) at the emergence of its roots from the brain stem, approached through a craniotomy of posterior fossa (Figure 1). Levels *a* and *b*, separated by ca. 20 mm, are peripheral to the petrossal and superior ganglia, and thus severance at these sites produce 'denervation' of taste buds, while damage at level *c*, central to sensory ganglia, produces 'decentralization' of receptor organs. Electrophysiological recordings of peripheral stumps of both glossopharyngeal nerves (severed at different levels or one intact as control) were performed between 15 h and 84 days after the initial operation, and followed by fixation and staining of the vallate papillae for histological studies.

Glossopharyngeal nerve activity was elicited by mechanical (rubbing the posterior third of the tongue with a plastic probe) and chemical (jets of citric acid, quinine or NaCl solutions directed towards the vallate papillae) stimuli. While impulses in mechanosensory fibres are generated directly by deformation of nerve terminals, sapid substances act on epithelioid receptor cells which in turn excite synaptically chemosensory fibres.

Tactile and gustatory responses disappeared between 22 and 30 h after denervation (transection at levels *a* or *b*), tactile responses usually persisting for 1 or 2 h after chemical stimuli were no longer effective. This confirms an earlier report[5] that generator activity in nerve terminals fails at a time when conduction of nerve impulses electrically-elicited is still preserved. Indeed, we found that the compound potential and conduction velocity in the peripheral stump of transected glossopharyngeal nerves were normal at least up to 36 h after operation. The early failure of receptor function may be correlated

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with ultrastructural alterations of nerve endings on taste receptor cells observable as soon as 12 h after denervation[6,7].

In a series of cats in which both glossopharyngeal nerves had been transected, one close to the mucosa (level a) and the other about 20 mm farther (level b), bioelectrical activity always persisted for approximately 2 h longer in those nerves in which the peripheral stump was longer. It is interesting to note that in catfish barbels, taste buds near to the site of axotomy degenerate before those located farther away[8]. In a similar way, the time for neuromuscular failure depends on the distance between the lesion of the motor nerve and the endplates[9].

Our observations (see Figure 2) showed that the number of taste buds per trench wall–analized according to the procedure of GUTH[4] – is reduced to 82% at the 5th day of denervation, to 41% at the 8th day, to 12% at the 13th and to ca. 5% between 20 and 84 days after denervation (in animals reoperated at 42 and 84 days, McKenzie silver clips had been applied to the central stumps to prevent reinnervation of taste buds). These results are clearly different from those obtained after transection of glossopharyngeal nerve roots: the decentralized vallate papillae showed apparent normality, and the number of taste buds counted at 4, 10, 36, 45 and 83 days after decentralization was within normal limits. Otherwise the electrophysiological responses to mechanical and chemical stimulation of the posterior third of the tongue were similar in these decentralized nerves, as compared with the contralateral normal nerves.

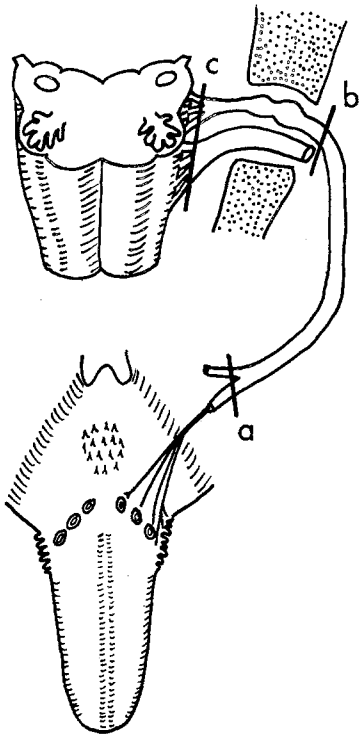

Fig. 1. Glossopharyngeal nerve severance toproduce decentralization (c) or denervation of vallate papillae with long (b) or short peripheral axon stump (a).

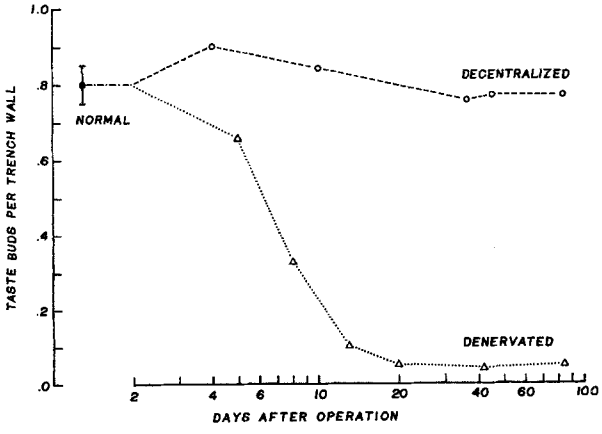

Fig. 2. Number of taste buds in normal, decentralized and denervated vallate papillae, analyzed following GUTH's procedure[4].

In conclusion, while denervation of circumvallate papillae results in a rapid failure of taste reception and subsequent disappearance of taste buds, decentralization does not interfere with the function and structure of these receptors. The continuity of gustatory nerve terminals with corresponding sensory ganglia is essential for integrity of taste buds, but this trophic influence can still be exerted by neurons disconnected from the central nervous system (see also[10–12]).

The observation that receptor function of nerve fibres fails later when there is a longer distance between the site of axotomy and the receptor organs, suggests that the function of nerve terminals can still be maintained by substances supplied by axoplasmic transport in the peripheral stump of the nerve transected (see[13,14]); the fast transport system still functions in axons disconnected from their perikarya[15].

As to the nature of the trophic factor here implicated, it appears to be specific of 'gustatory' sensory neurons since reappearance of taste buds occurs after reinnervation by glossopharyngeal, chorda tympani and vagus nerves, but it is absent after reinnervation by motor or non-gustatory sensory nerves[4,16]. The substance(s) is most probably synthetized in the sensory ganglia and then transported both towards the central and peripheral endings, since taste buds regeneration can be achieved with both peripheral or central processes of the nodose ganglion[17]. Furthermore, blockade of axoplasmic transport by colchicine applied to the glossopharyngeal nerve induced the disappearance of taste buds in an analogous manner to that observed after nerve transection[18]. It has been shown that labelled amino acids are incorporated by the petrossal ganglion, migrate along the glossopharyngeal nerve (the front of radioactivity advancing faster than 1 cm/h) and become concentrated at the level of sensory endings in contact with carotid body and vallate papillae chemoreceptor cells[19].

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