Morphological alterations of tongue epithelium in zinc-deficient rats

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[Accepted for publication: July 20, 1983]

Key words: zinc-deficiency / hyperparakeratosis / taste disorder / SEM

It is known that deficiency of dietary zinc has caused hyperparakeratosis in orthokeratinized epithelia of skin and oral mucosa as well as growth retardation in several experimental animals. Since the demonstration by McConnell and Henkin (1974) that a zinc-deficient diet caused hypogeusia in rat, interest has developed on the possible relationship between gustatory disorder and zinc. This communication describes briefly the morphological changes of the tongue, especially the gustatory area, in zinc-deficient rats together with a discussion concerning the pathogenesis of the gustatory disorder.

Materials and Methods

Fifty male weanling rats were separated into experimental groups of 30 animals and 20 controls of matching body weights. The experimental animals were fed a zinc-deficient diet containing 1.3 ppm zinc and the control animals were placed on the same diet supplemented by zinc carbonate at 40 ppm. The experimental animals were supplied with distilled water and the controls with tap water. After four weeks on these diets, the rats were perfused under anesthesia through the left ventricles with 2.5% glutaraldehyde. The tongue tips were immersed in isooamyl acetate after dehydration in ethanol, the critical point dried and coated with gold palladium to be examined under a JSM-25 scanning electron microscope (SEM). Some tissues were embedded in paraffin for light microscopic examination.

Results and Discussion

All of the zinc-deficient rats showed marked retardation of growth, loss of hair and skin changes (Fig. 1). On gross examination, the dorsal surface of the tongue of the experimental animal was shrived, often fissured and discolored whitely. Under examination by light microscopy, the dorsal epithelium of the zinc-deficient rat tongue remarkably increased in thickness and indicated hyperparakeratosis (Figs. 2, 3, 4 and 5). These hyperparakeratotic changes were conspicuous in the valleys between the filiform papillae. The fungiform papilla along with their taste pore was covered with keratinized cells (Fig. 5). Microabscess and cell death were found occasionally in the epithelium of the spinosus layer (Fig. 6).

When observed by SEM, remarkable changes were seen on the dorsal surface of the tongue. In zinc-deficient rats, no fungiform papilla could be found on the dorsal surface, even on the tongue tips where the greatest accumulation of the fungiform papillae was seen. A large number of keratinized cells and cell debris were piled up between the tips of the filiform papillae.

Several mechanisms on the pathogenesis of the hyperparakeratosis in zinc-deficiency have been discussed and are summarized by Chen (1980) as follows: 1) accelerated cell proliferation, 2) reduced desquamation rate, and 3) failure of enzymatic breakdown of nuclei and organelles in the keratinizing cells. It is known that zinc plays an important role in the taste sensation of humans.
Some hypogeusia in humans is improved after administration of zinc\textsuperscript{10,11}). In addition, it has been reported that hypogeusia occurred in rat fed with a zinc-deficient diet\textsuperscript{4-7}). The pathogenesis of hypogeusia in zinc-deficient rat remains to be elucidated. In the present study, the fungiform papilla of the zinc-deficient rat was buried under heaps of keratinized cells and could not be found on the dorsal surface of the tongue even by the use of SEM. Since the taste hairs in the taste pore were considered to be the only site of contact with the tastants\textsuperscript{12)}, our results suggest that one of the causes of hypogeusia in the zinc-deficient rat may be related to obstruction of the taste pore by keratinized cells.

Other plausible causes for the occurrence of taste disorder in zinc-deficiency can also be speculated. Alvares and Meyer (1973)\textsuperscript{1)} showed that parakeratosis of the zinc-deficient rat buccal epithelium might be due to the accelerated cell proliferation by autoradiographic technique. It is of interest to speculate that the life span of the taste bud cells of zinc-deficient rats may be accelerated similar to the situation of the buccal epithelial cells. Beidler and Smallman (1965)\textsuperscript{13)} estimated the average life span of the taste bud cells at about 250±50 hrs. This means that the nerve to taste cell contact is limited in time. Assuming that there may be accelerated senescence of taste bud cell due to zinc-deficiency, it is quite plausible that the contact period between nerve and taste cell may be shortened. Further studies including ultrastructural and autoradiographic analysis are needed.

References

Explanation of Figures

Fig. 1 Zinc-deficient rats show marked growth retardation, loss of hair and skin changes. C: control rat.

Fig. 2 Light micrograph of a control dorsal surface of tongue. The epithelium is orthokeratinized. ×70.

Fig. 3 Light micrograph of a fungiform papilla. A taste bud contacts directly with oral environment through a taste pore (arrow). ×150.

Fig. 4 Light micrograph of a zinc-deficient ventral surface of tongue. Hyperparakeratosis is conspicuous in the valleys between filiform papillae. ×60.

Fig. 5 Light micrograph of a zinc-deficient fungiform papilla. A taste pore (arrow) is obstructed by keratinized cells. ×140.

Fig. 6 Microabscess is occasionally seen in zinc-deficient dorsum of tongue. ×60.

Fig. 7 SEM photograph of a control rat tongue. Fungiform papillae are scattered among the filiform papillae. ×60.

Fig. 8 SEM view of a zinc-deficient dorsum of tongue in the same area as Fig. 7. No fungiform papilla is seen among the filiform papillae. ×80.

Fig. 9 SEM photograph showing the tongue tip of a zinc-deficient rat. The keratinized cells are piled up between the apexes of the filiform papillae and no fungiform papilla is observed. ×125.