The experimental evidence at present indicates that the main cause of infections atrophic rhinitis (AR) in Bordetella bronchiseptica; young pigs inoculated intranasally with this organism develop characteristic deformities and reduction in volume of the nasal turbinates.

Ultrastructural investigations have shown that the decreased bone formation of the nasal turbinates appeared to be due to a disturbance in the protein metabolism of the osteoblasts. Non-specific alkaline phosphatase (AP) and acid phosphatase have been reported as playing a role in osteogenesis. A correlation of the increase in AP activity with the morphological changes in the nasal turbinates of pigs with AR has been suggested.

Fifty conventional pigs were used in this investigation. Thirty piglets were given intranasal inoculations of Bordetella bronchiseptica, at 21 days of age. Pigs were killed at 2, 3, 4, 5, and 6 weeks post-inoculation (PI). Biochemical estimations of AP in the turbinates were performed and procedures for histochemical investigation of AP and acid phosphatase and for ultrastructural changes were carried out. Turinate hypoplasia, at the gross level, was graded according to Dorem's scale.

Turinate atrophy was recorded in all pigs killed at 2, 3, 4, 5, and 6 weeks PI with grades of atrophy varying from 1 to 4. Biochemical investigation of AP showed a double increase in enzymatic activity in turbinates of pigs killed at 2 weeks PI and a threshold rise in those killed at 4 weeks PI. The AP levels obtained from pigs killed at 5 and 6 weeks PI were similar to those at 4 weeks PI. Histochemically an intense and widespread enzymatic response was also observed in the periosteum of turbinates of pigs killed at all stages. A slight increase in acid phosphatase activity was noted in turbinates of pigs killed from 2 to 6 weeks PI. The localization of the reaction product in the osteoblasts was predominantly cytoplasmic. Ultrastructural observations showed that severe and progressive degenerative changes occurred in osteoblasts of turbinates in pigs killed at all stages.

The network of the endoplasmic reticulum contained many lacunae which showed from moderate distention to severe dilatation with occasional rupture. A fine fibrillar material was a constant presence within the cisternae. Degenerative changes, from moderate to severe, were also observed in the Golgi complex.

The results show that the infection with Bordetella bronchiseptica produces turinate atrophy up to 6 weeks PI. The levels of AP activity increased up to 4 weeks PI, thus confirming previous observations made by Silveira, Edington & Smith. The levels of AP did not have any significant rise after 4 weeks PI.

Biochemically, acid phosphatase activity has shown a slight increase at all stages. Considering the localization of the reaction-product for acid phosphatase, it is possible to suggest that the increase in enzymatic activity may be correlated with the disturbance in protein synthesis or in protein transport by the Golgi complex. The ultrastructural findings confirm previous observations made by Patten and Cappen and Silveira, Edington & Smith.

Conclusions:
1) Bordetella bronchiseptica inoculated into 21-day-old piglets produced an increase in AP activity up to 4 weeks PI which remained at the same level up to 6 weeks PI.
2) Acid phosphatase was slightly increased from 2 to 6 weeks PI suggesting a possible correlation with the metabolic changes in the osteoblasts.
3) Confirming previous observations, the turinate hypoplasia produced by Bordetella bronchiseptica is mainly a result of a reduced bone matrix due to a disturbance in the protein metabolism of the osteoblasts.

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