ectodermal N-acetyl-β-glucosaminidase is a lysosomal enzyme related to the ability of thecate hydroids to break down the perisarc14. The results with the method for β-D-glucosidase were entirely negative.

The results give strong indication that the perisarc of C. flexuosa is hardened and darkened by quinone tanning. A single cell type has been shown to contain primary

catecholamine, phenol oxidase and copper.

In addition to the histochemical evidence for quinone tanning, I have identified dopamine (a primary catecholamine with a free para position) in alcoholic extracts of whole colonies using column, paper and thin-layer chromatography and paper electrophoresis. The dopamine in extracts is thought to come from the tanning cells.

The positive results given by the desmocytes may indicate that these cells arise from tanning cells by the in situ tanning of their own proteins. Although cells morphologically similar to tanning cells have been described before in Campanularia flexuosa and other thecate hydroids, no evidence of their function in quinone tanning of the perisarc has been previously presented.

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Serum Thiocyanate Concentrations in Tobacco Amblyopia

THERE has recently been interest in the suggestion that tobacco amblyopia may be a manifestation of chronic cyanide toxicity, the cyanide radical being derived from tobacco smoke¹⁻³. Although it is known that cyanide in the body is chiefly detoxified to thiocyanate and that smokers show higher serum thiocyanate concentrations than non-smokers, no study of serum thiocyanate concentrations in those affected by tobacco amblyopia has been reported.

Serum thiocyanate determinations by Bowler's method⁵ were made on three groups of subjects. The first group consisted of forty patients suffering from tobacco amblyopia, diagnosed according to the criteria of Heaton et al.6. In group two were thirty-seven non-amblyopic smoking subjects drawn from a hospital population. three contained sixty non-smoking hospital patients. Those in groups two and three were elderly patients attending hospital with conditions unrelated to tobacco amblyopia. The age composition of all three groups was similar.

Patients in the three groups were classified at each of three levels of serum thiocyanate concentration as shown in Fig. 1. Sixty-one per cent of the non-smokers had a thiocyanate concentration of 0.5 mg per cent or less, only 6.5 per cent having a concentration of 1.5 mg per cent or more. In contrast, a serum thiocyanate concentration of 0.5 mg per cent or less was found in 38 per cent of nonamblyopic smokers, whereas 32 per cent had concentrations of 1.5 mg per cent or more. The distribution of serum thiocyanate concentrations in these two groups is significantly different ($\chi^2 = 11.76$, n = 2, P < 0.01), con-

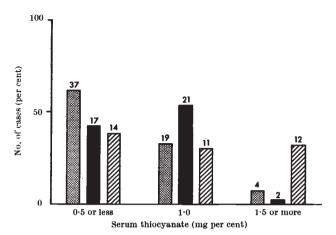


Fig. 1. Serum thiocyanate concentrations in three groups of subjects: forty patients with tobacco amblyopia (black columns), thirty-seven non-amblyopic smoking subjects (hatched columns) and sixty non-smoking hospital patients (stippled columns).

firming the findings of Stoa4 that serum thiocyanate concentrations are higher in smoking than in non-smoking subjects.

As might be expected, the mean tobacco consumption of the group of patients suffering from tobacco amblyopia (2.95 ± 1.24) ounces per week) was higher than that of the non-amblyopic smokers (1.85 ± 1.19) ounces per week) (t =14.39, n = 75, P < 0.001). In spite of this, the group of amblyopic patients in general showed much lower serum thiocyanate concentrations than did the group of nonamblyopic smokers, the distribution of thiocyanate contrations being significantly different in the two groups $(\chi^2 = 10.47, n = 2, P < 0.01)$.

Somewhat surprisingly, in spite of their heavy consumption of tobacco, the distribution of thiocyanate concentrations in this group of amblyopic patients resembled that of the non-smokers and in fact the distribution of thiocyanate concentrations in these two groups was not significantly different ($\chi^2 = 3.5$, n = 1, P > 0.05).

These relatively reduced concentrations of serum thiocyanate in tobacco amblyopes as compared with healthy smokers suggest that if cyanide derived from tobacco smoke is indeed a factor of aetiological importance in the development of tobacco amblyopia, the biochemical defect in these cases may be a failure of conversion of cyanide to thiocyanate, its chief detoxification product, as suggested by Smith and Duckett7.

The role of B₁₂ deficiency in this disease is difficult to determine and it will be interesting to see the effect on the serum cyanide and the thiocyanate concentrations of treatment of these amblyopic patients with hydroxocobalamin, a study we are at present carrying out, using the method described by Aldridge⁸ for determination of thiocyanate and cyanide. This method was not available to us for the present study.

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