For complete inhibition at saturating light intensity, the molar ratio of adsorbed CMU to chlorophyll is of the order of 1/200 (ref. 4) or of 1/280–1/700 in *Euglena*[14], i.e. about one CMU molecule per Emerson center. However, our data show that the photochemical reaction is affected at concentrations three or four orders of magnitude smaller than that required to produce full inhibition at saturating light intensity. This high affinity of CMU for the photochemical reaction can be explained by two alternative hypotheses: either it competes with a photo product for an enzyme or it acts as a trap at the energy-transfer level.

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Received October 20th, 1962

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Biochim. Biophys. Acta, 69 (1963) 438–440

SN 1209

Biosynthesis of ascorbic acid by rats fed a high level of dietary tyrosine under toxic and adapted conditions

That a high level of dietary tyrosine is toxic to rats is well established[1–3], though the nature of this toxicity is not clear. It has also been reported[4] that rats can adapt to a high tyrosine intake. This adaptation is indicated by a return to lower excretion levels of intermediary metabolites of tyrosine and is correlated with an increase in growth rate. However, little is known about the mechanism of this adaptation. Since ascorbic acid is known to have a role in tyrosine metabolism[5], it seemed worthwhile to examine whether the biosynthesis of ascorbic acid by the liver tissues of rats is affected during feeding toxic amounts of tyrosine and after adaptation to these high amounts of tyrosine.

In our experiments, albino rats of either sex, weighing 40–50 g were used. They were fed a basal diet containing 9% casein prepared according to BENTON *et al.*[1],

Tyrosine at 5 % level was mixed with the diet at the expense of an equivalent amount of sucrose. The tyrosine-toxic rats were sacrificed simultaneously with control rats within 3–4 weeks of the experimental period when they developed acute toxic symptoms, as described by earlier workers[1–3]. The tyrosine-fed adapted rats, on the other hand, were sacrificed simultaneously with control rats within 9–10 weeks of the experimental period by which time they had recovered from the toxic symptoms. The synthesis of ascorbic acid by the liver tissues of these rats were studied *in vitro*, by following the method of CHATTERJEE *et al.*[6] using D-glucuronolactone as substrate.

TABLE I

SYNTHESIS OF ASCORBIC ACID *in vitro* BY LIVER TISSUES OF TYROSINE-TOXIC, TYROSINE-FED ADAPTED AND CONTROL RATS

The test system contained 0.02 M phosphate buffer (pH 7.4), 0.025 M D-glucuronolactone, 0.05 M KCN and 0.63 ml of enzyme preparation* (equivalent to 157 mg wet tissue) in a total volume of 2.5 ml. Incubated for 1.5 h at 37°, in air (6).

	*μmole of ascorbic acid synthesized***
Tyrosine toxic (8)**	0.13 ± 0.01
Control (7)	0.34 ± 0.02
Tyrosine-fed adapted (6)	0.33 ± 0.03
Control (6)	0.34 ± 0.02

* A 25 % liver homogenate was prepared in 0.25 M sucrose solution. The homogenate was centrifuged at 9000 × *g* for 40 min at 0°. The resultant supernatant was used for the assay.
** Figures in the parentheses represent the number of animals assayed in each group.
*** Values represent mean ± standard error of mean.

There was a marked decrease in the synthesis of ascorbic acid by the liver tissues of the tyrosine-toxic rats as compared to the controls but in the case of the adapted rats this synthesis was much increased and was equal to that of the controls (Table I). Furthermore, sex difference was found to have no effect on the relative synthesis of ascorbic acid by rat-liver tissues under these conditions.

From these observations it would appear that tyrosine toxicosis in rats might be related with the reduced biosynthesis of ascorbic acid by the liver tissues. It is also apparent that biosynthesis of ascorbic acid plays a part in the adaptation to high tyrosine intake.

However, the results do not indicate the mechanism by which ascorbic acid biosynthesis is inhibited in tyrosine-toxic conditions. It might be that tyrosine or some of its metabolites is acting as inhibitor. Studies on the mechanism of this inhibition in tyrosine-toxic rats and the ascorbic acid concentration of various tissues of rats under tyrosine-toxic and tyrosine-fed adapted conditions are in progress.

We should like to express thanks to Dr. J. J. GHOSH and Dr. N. C. GHOSH for their suggestion during this work and also to the Indian Council of Medical Research for financing this work.

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Received November 8th, 1962

Biochim. Biophys. Acta, 69 (1963) 440–442

PN 1102

A new variant of abnormal methaemoglobin: Hb M$_{Boston}$

The ability of haemoglobin to bind reversibly molecular oxygen depends on the specific native structure of this haemoprotein. It is therefore to be expected that many different structural alterations of the haemoglobin molecule, of denaturational as well as genetic origin, result in a loss of the respiratory properties of haemoglobin and lead to spontaneous oxidation of the haemoglobin iron (formation of methaemoglobin) in presence of oxygen. About ten genetic variants of haemoglobin possessing abnormal α or β chains[1,2] and occurring in methaemoglobin form (known as M haemoglobins) are known at present. A variant with different spectrochemical properties then those of the known Hb M types is described below.

The haemoglobin under study has been found in four cyanotic but otherwise symptomless subjects from three generations of a Polish family. The absorption spectrum of destromatized haemolysate treated with $K_3Fe(CN)_6$ showed in these cases an elevation characteristic for Hb M instead of a minimum at 600 mμ; addition of cyanide resulted in a normal cyanmethaemoglobin spectrum (Fig. 1). When the

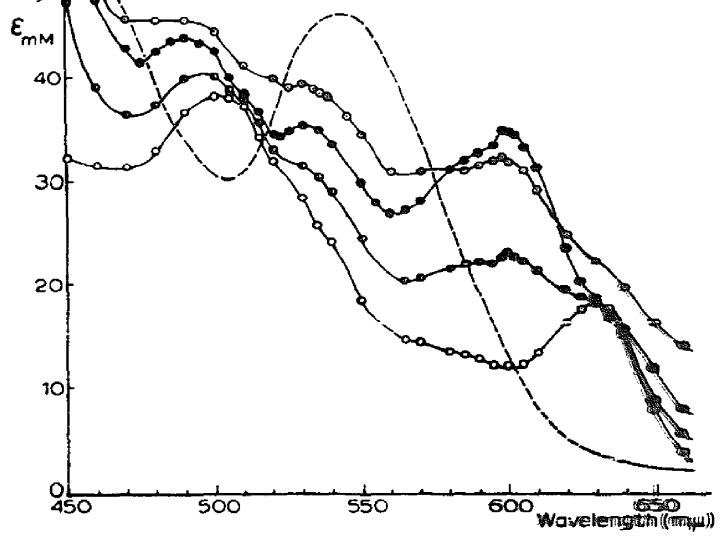

Fig. 1. Absorption spectra (0.0167 M phosphate buffer, pH 6.6) of methaemoglobins: ○, normal; ◐, patient; ●, calculated abnormal; ⊙, abnormal isolated from starch gel; ——, cyanmethaemoglobin (identical for patient and normal control).