Table 2. Hydrocarbons of blue-green algae (percentage of composition). The letters tr indicate trace, that is, less than 5 percent.

Organism*	15:0 †	16:0	17:0	17:1	br18 ‡	18:0	19:1	19:2
Trico	tr	2	95	1		1		
Ana. n.	21	5	68	5				
Mev	9	tr	91	tr		tr		
TX-20	6	2	90	2		tr		
BA-1	2	4	92			2		
Mont	1	4	86	1	8			
NM	1	2	83	1	10			
17A				1		1	85	13
PR-6							92	7
BG-1							98	2
SP 31	6	3	90	1		tr		
Mat §	2	2	36	4	11	1	8	

* See Table 1 for species names. † The numbers before the colon indicate the numbers of carbon atoms; the numbers after the colon, the numbers of double bonds. ‡ The identifications of the branched C_{18} compound(s) (br18) are tentative, based only on estimated gas-liquid chromatography retention times and hydrogenation data. § The mat was more complex than the pure cultures. In addition to small amounts of br18 molecules it contained 32 percent of C(17:2), a molecule not found in the pure cultures, and 1 percent of C(19:0).

C_{18} range either saturated or with one double bond. *Nostoc* and *Lyngbya* contain significant amounts of a C_{18}, branched-chain hydrocarbon. Similar amounts of a branched alkane were reported for *Nostoc* sp. and tentatively identified as 7,9-dimethylhexadecane (*2*). Our pattern for *Anacystis* agrees with that reported recently (*2, 3*). The olefins of high molecular weight (more than C_{20}) reported (*4*) for *Anacystis montana* were not found in any of our samples. A special effort was made to detect hydrocarbons in the C_9 range in the case of *Trichodesmium*. The fatty acids of this bloom of *Trichodesmium* consisted of 50 percent C_{10} chains with no unsaturation (10:0). The fact that no C_9 or C_{10} hydrocarbon was detected suggests that fatty acids are not the precursors of hydrocarbons either by decarboxylation or reduction.

The comparative data on the hydrocarbons and fatty acids (*1*) define the role of blue-green algae as source material for the organic matter found in ancient sediments. The blue-green algae surveyed could supply only very simple (C_{12} to C_{20}) hydrocarbons and fatty acids to sediments.

Neither the biological function nor the significance of the restricted pattern of biosynthesis of hydrocarbons in blue-green algae are understood.

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Adrenal Tyrosine Hydroxylase: Compensatory Increase in Activity after Chemical Sympathectomy

Abstract. *Destruction of peripheral sympathetic nerve endings with 6-hydroxydopamine causes a disappearance of cardiac tyrosine hydroxylase, accompanied by a twofold increase in adrenal tyrosine hydroxylase and a small increase in phenylethanolamine-N-methyl transferase. No change in adrenal catecholamine content occurs under these conditions.*

6-Hydroxydopamine (6-OH-DA) selectively destroys postganglionic sympathetic nerve endings in various species (*1, 2*). These morphological changes are accompanied by a long-lasting depletion of norepinephrine in sympathetically innervated organs and by an impaired uptake of H^3-norepinephrine (*2*).

The rate of synthesis of the sympathetic neurotransmitter norepinephrine is determined mainly by the activity of tyrosine hydroxylase (*3*). This enzyme hydroxylates tyrosine to dihydroxyphenylalanine (*4*), which is subsequently decarboxylated to dopamine, the immediate precursor of norepinephrine. In the adrenal gland a further step occurs—norepinephrine is methylated to epinephrine by phenylethanolamine-*N*-methyl transferase (PNMT).

Administration of 6-OH-DA almost completely destroys adrenergic nerve endings in the rat heart, but appears to leave the adrenal medulla intact (*2*). Therefore the activity of tyrosine hydroxylase in these two organs was examined after chemical sympathectomy with 6-OH-DA. We report that after this treatment the tyrosine hydroxylase activity in the rat heart disappears and that this enzyme in the adrenal gland is markedly increased.

Male Sprague-Dawley rats (100 to 125 g) were given two intravenous injections of 6-OH-DA (100 mg/kg in 0.01*N* HCl) at 8-hour intervals, and were killed 16 or 40 hours after the second injection by a blow on the head. The hearts and adrenal glands were rapidly removed and chilled on cracked ice. The adrenal glands were homogenized in 2.0 ml of 0.25*M* sucrose and centrifuged at 27,000*g* for 10 minutes. The hearts were blotted dry, minced with an Arbor tissue press, and centrifuged at 105,000*g* for 20 minutes. To remove compounds that interfere with the determination of tyrosine hydroxylase activity in the rat heart, we passed samples of the supernatant over Sephadex G-10 columns (3 cm long) equilibrated with 0.25*M* sucrose. Portions of the effluent from the heart or supernatant fraction of the adrenal gland were used to estimate protein (*5*) and tyrosine hydroxylase (*6*); PNMT was determined in the adrenal gland supernatant fraction by a previously described method (see *7*). Epinephrine and norepinephrine were determined by the procedure of Anton and Sayre (see *8*).

Sixteen hours after the second injection of 6-OH-DA cardiac tyrosine hydroxylase activity had decreased to almost 15 percent of control values (Fig. 1), whereas the enzyme activity in the adrenals was markedly increased, whether expressed as activity per protein, unit weight, or pair of adrenals (Fig. 2). At this time cardiac catecholamines were no longer detectable (con-

trol, 0.88 ± 0.07 μg/g). Forty hours after administration of 6-OH-DA cardiac tyrosine hydroxylase was not measurable and the adrenal enzyme activity had increased further to more than double the control value. At this later time there was also a small but significant ($P < .05$) increase in PNMT (Fig. 2). At neither time was the concentration of adrenal norepinephrine and epinephrine altered.

The coincident destruction of cardiac sympathetic nerve endings and the disappearance of tyrosine hydroxylase after administration of 6-OH-DA are consistent with the view that this enzyme is located within the nerve (*9*). The disappearance of norepinephrine 16 hours before the enzyme had completely disappeared may be due to a displacement of norepinephrine by 6-OH-DA or one of its metabolites, or by both, while the destruction of the nerve terminals is not yet complete (see *1, 2*).

In contrast to that in most peripheral tissues, such as the heart, vas deferens, and spleen, the norepinephrine and epinephrine content of the adrenal is unchanged after this dose of 6-OH-DA.

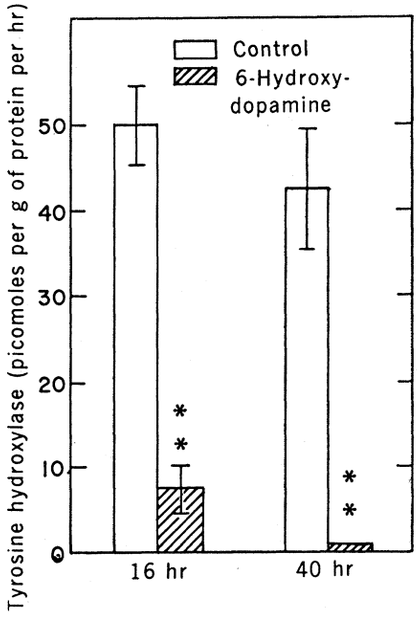

Fig. 1. Cardiac tyrosine hydroxylase after administration of 6-OH-DA. Individual hearts of rats (four or five per group) that had received the second dose of 6-OH-DA 16 or 40 hours previously, or their corresponding controls, were assayed for enzyme activity. Results are expressed as mean ± standard error of the mean (brackets). The P values (** $< .01$) were obtained by a t-test for differences between groups.

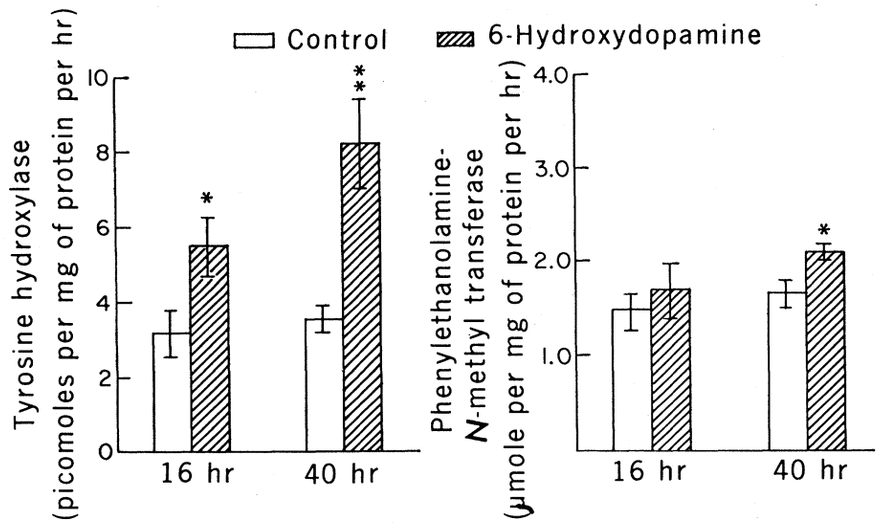

Fig. 2. Adrenal tyrosine hydroxylase and PNMT after administration of 6-OH-DA. Pairs of adrenals from groups of rats (four or five per group) that had been treated with 6-OH-DA 16 or 40 hours previously, or their corresponding controls, were assayed for enzyme activity. Results are expressed as mean ± standard error of the mean (brackets). The P values (* $P < .05$; ** $P < .01$) were obtained by a t-test for differences between groups.

In addition, the activity of tyrosine hydroxylase, the enzyme critical in the regulation of catecholamine biosynthesis, is considerably increased (*3*). Thus as a consequence of the destruction of the apparatus for synthesis and storage of the physiological sympathetic neurotransmitter in the rat heart and other peripheral tissues, the adrenal gland appears to compensate by increasing the activity of tyrosine hydroxylase. The unchanged concentration of catecholamines, together with a marked elevation of tyrosine hydroxylase, suggests an increase in catecholamine turnover.

Preliminary studies indicate that the rate of synthesis of adrenal catecholamines at this time is also accelerated. The virtually complete destruction of the peripheral sympathetic nerves and the increased capacity, by the adrenal gland, to synthesize catecholamines would indicate that this latter organ serves to supply the peripheral tissues with the depleted neurotransmitter. In addition, the physiologic effect of the catecholamines released from the adrenal gland is increased, since chemical sympathectomy leads to a supersensitivity to these pressor agents (*10*).

There are several mechanisms that may be responsible for the increase in tyrosine hydroxylase in the adrenal gland. One might be a blood-borne factor, as in the case of the induction of PNMT by adrenal corticoids (*11*). Another might be a consequence of the hypotension produced by 6-OH-DA (*12*), which might lead to an increase in the activity of splanchnic nerves supplying the adrenal by a baroreceptor reflex. In either case the increase in tyrosine hydroxylase activity might be the result of synthesis of new enzyme protein or activation of the preexisting enzymes.

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