annatto among the richest vegetable sources of vitamin A thus far reported.

It is unknown at this stage of the investigation whether we are dealing with vitamin A as such, carotin or some related pigment, or a new substance which can function as vitamin A in the animal body.

A preliminary experiment of feeding bixin obtained from annatto that had had the active resinous coloring material removed by alcoholic extractions confirmed Euler's finding in that it did not cause growth in rats on an A-free diet. There is some indication that bixin may exert a toxic action on the organism.

Further work is in progress on this interesting development.

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A NOTE ON THE EFFECT OF ULTRA-VIOLET LIGHT ON THE VITAMIN A OF BUTTER

ZILVA[1] has shown that vitamin A is destroyed by ultra-violet light when exposed in air but not in a carbon dioxide atmosphere. Spinka[2] found that ultra-violet radiations did not destroy vitamin A, but that toxic materials were formed which were sufficient to cause death in rats. His animals succumbed before the controls on a vitamin A free diet developed the typical symptoms of vitamin A deficiency.

The fact that radiations other than ultra-violet light[3] destroy the vitamin A of butter suggested that the mechanism might be of a photochemical nature.

Butter fat was exposed in large flat dishes to the rays of the quartz mercury arc at a distance of 15 cm. for 6 hours. At the end of 3 hours all the color of the material had disappeared. Another portion of the fat was exposed under similar conditions in an atmosphere of nitrogen which had previously been purified to free it from oxygen. No change in color was observed. These materials and the untreated butter fat were fed to rats depleted of their vitamin A stores, as follows: (1) The untreated butter fat; (2) butter fat exposed to ultra-violet light in air; (3) butter fat exposed to ultra-violet light in a nitrogen atmosphere; (4) the untreated material mixed in equal parts with the butter fat exposed to ultra-violet light in the air. Control animals received no source of vitamin A. The results are presented in Fig. 1.

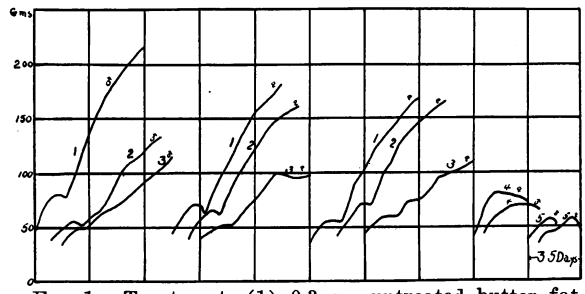

FIG. 1. Treatment. (1) 0.3 gm untreated butter fat, daily. (2) 0.3 gm butter fat irradiated in nitrogen atmosphere, daily. (3) 0.3 gm untreated butter fat plus 0.3 gm butter fat irradiated in air, daily. (4) 0.3 gm butter fat irradiated in air, daily. (5) No source of vitamin A.

RESULTS

Practically complete destruction of vitamin A potency and loss of color in butter fat was obtained when the material was exposed in air. The material exposed in a nitrogen atmosphere did not fade. Its antiophthalmic properties were not reduced in any detectable degree, although some reduction in its growth-promoting power was found.

When butter fat exposed in the air was mixed with the untreated material a definite slowing of growth was obtained as compared to that produced by untreated butter fat fed in an equivalent amount.

Negative controls and animals receiving butter fat exposed in the air succumbed at about the same time. Ophthalmia developed in both groups.

These experiments indicate that the change that takes place in vitamin A potency when butter fat fades is not due to a direct effect of ultra-violet light. Oxidation, indirectly produced by ultra-violet radiations, is at least one mode of destruction of the vitamin A of butter fat. It appears that irradiation in air and to some extent in a nitrogen atmosphere produces a principle that retards growth of rats. This principle was not of sufficient strength to cause death before the onset of vitamin A deficiency symptoms.

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