Cigarette Smoking and Vitamin C

Recently adduced facts indicate the embattled cigarette has yet another count against it.

Cigarette smoking apparently adversely affects the body's utilization of vitamin C.

By OMER PELLETIER, Ph.D.

ne thing is certain about cigarette smokers: They are more prone to cardiovascular and pulmonary disorders than nonsmokers. There are other adverse effects, too. What these sequelae of smoking are really caused by is not known. But whatever it is, cigarette smoking produces a variety of biochemical abnormalities-some expected, others surprising. Among the latter is the way inhalation of eigarette smoke affects the body's use of vitamin C. It is now evident the cigarette smoker needs more vitamin C each day than the nonsmoker.

One is tempted to pass off the eigarette's adverse effect on vitamin C levels to the generally depressing effect smoking has on appetite. But the effect in this case is both more direct and more complicated. That a factor other thanthe general curtailment of food intake is responsible has been suspected for some time. Now we have discovered clues to the biochemical phenomena at work. These clues should throw new j light on nutrition as a whole.

As early as 1939, Drs. L. H. Strauss and P. Scheer of the University of Cologne studied the acute effect of smoking eigarettes on the urinary exerction of vitamin C. They reported a drop in vitamin C exerction due to smoking, they related this effect to the inhalation of nicotine, which caused hyperactivity of the thyroid and adrenal glands, and enhanced the level of genal oxidative activity. Some investiga-

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tors have since found the acute and chronic effects of cigarette smoking depress vitamin C levels. Others were unable to confirm these findings. Contradictory results, the lack of suitable analytical methods and proper control, and failure to relate vitamin C levels to vitamin C intakes made it impossible to arrive at a conclusion. However, it appeared some oxidation reactions arising from metabolism of inhaled constituents of tobacco, or from modifications of the general metabolism affected by these constituents, possibly might cause vitamin C oxidation and degradation.

We found that lower levels of vitamin C in smokers are not caused by more rapid utilization or by a lower intake of vitamin C. The lower levels are due to less vitamin C available for utilization and storage reflecting the lower amount absorbed.

Over a prolonged period, a negative balance of vitamin C available for storage would result in the development of lower stores and reduced utilization to conserve these stores.

Less efficient absorption of vitamin C by eigarette smokers is not necessarily the result of general malabsorption. Solutions of vitamin C are easily oxidized in vitro; thus, one should look for its oxidation in the gastrointestinal tract. This is not easy to demonstrate experimentally in humans, but the following hypothesis appears possible: Since nicotine is known to release serotenin (5-hydroxytryptamine) from the small intestine, and since serotonin is known to be oxidized by enzymes, one can visualize increased activity of these

enzymes (nonspecific) which would cause oxidation of vitamin C.

It is well known that humans must take vitamin C (I-ascorbic acid) to prevent scurvy. Nor is scurvy entirely a disease of the past, since occasional cases are still to be seen. The characteristics of scurvy-loosening of the teeth, swelling of the joints, and various types of hemorrhages (petechial, subcutaneous, in joints and muscles, and intestinal]-can be attributed to collagen defects. Collagen is synthesized by specialized cells-fibroblasts, osteoblasts, and odontoblasts. This substance, which accounts for about thirty percent of all our body proteins, is the main supportive protein of skin, tendon, bone, cartilage, and connective tissue. The integrity of cellular structure depends on it. In the absence of vitamin C, the three peptide chains which form the collagen fibrils are synthesized, but do not acquire the fibrous properties characteristic of connective tissue. This is due to the lack of hydroxylation of proline and lysine by a specific enzyme which requires vitamin C for its activity.

Most vital organs of the human body contain much higher concentrations of vitamin C than do musele and connective and supporting tissues. Although there are indications vitamin C may be involved in biochemical functions other than collagen synthesis, the data supporting such theories are in most a cases too limited or too controversial to N permit a definite conclusion. Similarly, reports of the association of certain diseases with hypovitaminosis C are not well enough documented to conclude

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