
**Abstract**

The effect of ascorbic acid deficiency on adrenal hydroxylation of cholesterol and deoxycorticosterone in guinea pigs was studied by using mitochondria and isolated cytochrome P-450 fractions. The effects obtained were compared with the effects of long-term treatment with ACTH.

Advanced scurvy as well as treatment with ACTH resulted in an increase in the weight of the adrenals, the total amount of cytochrome P-450, the cholesterol side-chain cleavage activity, the cortisol level in plasma, and the excretion of unconjugated cortisol in urine. Total 1p- and 18-hydroxylation of deoxycorticosterone were not stimulated or were stimulated only to a small extent. It is suggested that the major effects observed in advanced scurvy are due to ACTH, the level of which was significantly increased, most probably as a consequence of the stress. In animals kept on a scorbutogenic diet for 2-4 weeks or, with a small dose of ascorbate added, for several weeks, changes were observed that could not be fully explained as effects of ACTH on normal adrenals. Although the plasma levels of ACTH and cortisol were increased only to a small extent and excretion of unconjugated cortisol in urine was unaffected, there was a significant increase in the total capacity of adrenal mitochondria to hydroxylate exogenous cholesterol. It is concluded that the level of ascorbate in the adrenals might be of some importance for the capacity to convert cholesterol into pregnenolone. The normal feed-back regulation is, however, intact in moderate ascorbate deficiency and the plasma level of cortisol is kept within normal limits.