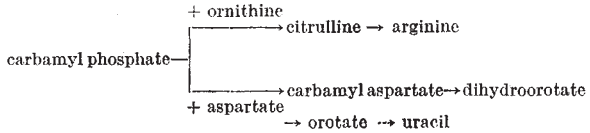
requirement was found to be due to changes in the level of arginine in the pre-incubation medium. Exhaustion of arginine relieved the cells from the 2-thiouracil requirement.

The 2-thiouracil requirement was also obtained when the mutant was grown with 2-thiouracil in the presence of citrulline. However, when the mutant was grown with 2-thiouracil in the presence of ornithine, inhibition of growth was less marked and neither loss of viability nor a 2-thiouracil requirement was observed when these cells were plated on to minimal medium.

Wild-type *E. coli W* cells are only slightly inhibited by 2-thiouracil. However, when arginine was added to the growth medium, 2-thiouracil exerted a marked inhibitory effect and also showed the 2-thiouracil requirement.

In all the experiments described, the 2-thiouracil requirement occurred when the synthesis of ornithine transcarbamylase, which catalyses the conversion of ornithine to citrulline, was repressed. This repression occurs in *E. coli W* and its mutant with arginine or citrulline but not with ornithine. In *E. coli B*, a non-repressible strain[3], no 2-thiouracil requirement was observed under the conditions described.

These results suggest a mechanism based on the metabolic relationship existing between arginine and uracil :



It has been suggested that these syntheses are to some extent competitive[4] and regulated by negative feed-back mechanisms[4,5].

Preliminary results show that 2-thiouracil mimics the feed-back action of uracil. Experiments are now in progress to elucidate the mechanism of the 2-thiouracil effect described.

We are indebted to Dr. B. D. Davis for strain *M*-39 *A*-23 used in this investigation.

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[1] Smith, L. H., and Sullivan, M., *Biochim. Biophys. Acta*, **39**, 554 (1960).
[2] Yates, R. A., and Pardee, A. B., *J. Biol. Chem.*, **227**, 677 (1957).
[3] Gorini, L., *Proc. U.S. Nat. Acad. Sci.*, **46**, 682 (1960).
[4] Gorini, L., and Maas, W. K., in McElroy, W. D., and Glass, H. B., *The Chemical Basis of Development*, 469 (Johns Hopkins Press, Baltimore, Md., 1958).
[5] Yates, R. A., and Pardee, A. B., *J. Biol. Chem.*, **221**, 757 (1956).

A Fatty Acid / Protein Complex in Human Hair

In an earlier publication[1] a new sensitive method for the detection of small amounts of damage to human hair was reported. The method involves counting the numbers of cuticular and cortical cells in the liquid after treating the hair under standard conditions with papain/bisulphite solution. When the ratio of cuticle cells to cortical cells is low, the hair is assumed to have been damaged in some way. Soxhlet extraction of the hair with ethanol increases the rate of breakdown, but ether, benzene and many other non-polar solvents do not affect the ratio. It was suggested that a protective substance soluble in alcohol but not in non-polar solvents exists in the hair which maintains it intact during papain digestion. This protective substance has now been isolated from ethanol extracts and identified as a fatty acid/protein complex.

Isolation by solvent fractionation was impracticable because the materials denatured at liquid/liquid interfaces. Silica fractionation by the method of Hanahan[2] was also unsatisfactory as the material was irreversibly adsorbed on the silica. Successful isolation was achieved by electrophoresis on polyurethane sponge in dilute acetic acid at *p*H 2·5. Under these conditions fatty acids and neutral fatty materials do not move, but the complex moves towards the negative electrode. Analysis of the isolated complex shows that it consists of 20–30 per cent fatty acid and 60–70 per cent protein, which yield substantial amounts of lysine but very little cystine. Methyl esters of the fatty acids are not volatile under normal GLC conditions, but the mass spectrometer indicates there is considerable variation between samples. Treatment of alcohol-extracted hair with an alcoholic solution of the complex reduces the rate of breakdown of hair to the normal level. This is not due to enzyme inactivation, since the degradation of casein by papain is not affected by the presence of the complex. The complex is positively charged in acid solutions and the rate of migration on paper electrophoresis decreases as the *p*H is made more alkaline until no movement is observed at *p*H 6·0. The complex then remains at the origin at *p*H values up to 9·5. This, combined with the high level of lysine, suggests that the fatty acid is combined through the amino-groups of the lysine so that the effective isoelectric point is in the range for normal proteins. In alkaline solution, negatively charged fatty acid residues are removed and this maintains the electrical neutrality of the protein.

I wish to thank Unilever, Ltd., for permission to publish this communication.

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[1] Holmes, A. W., *J. Text. Inst.*, **50**, 422 (1959).
[2] Barron, E. J., and Hanahan, D. J., *J. Biol. Chem.*, **231**, 493 (1958).

PHYSIOLOGY

Distribution of Acetylcholine and Cholinesterase in the Heart of White Rats

The opinion has been advanced that acetylcholine acts in the heart not only as the mediator of the vagus nerve but also as the so-called local hormone in local extraneural regulation of heart activity and that in this last function it is closely associated with heart automation[1]. If this view is correct, it can be assumed that the most rapid turnover of acetylcholine will occur in those parts of the heart which possess the highest degree of automation. In order to verify the correctness of this assumption, experiments were carried out in which the distribution of acetylcholine and cholinesterase in different parts of white rat heart was examined.

White rats of either sex were used. The animals weighed 190–365 gm. On account of the small