appears to be located in that part of the pathway between acetyl coenzyme A and mevalonic acid. The earlier part of this pathway, leading to the formation of β-hydroxy-β-methyl-glutaryl co-enzyme A, is common to both cholesterol synthesis and acetoacetate formation. Since it has previously been shown that in rats treated 6 hr. previously with growth hormone the rate of hepatic acetoacetate output is much reduced[5], it would appear that the increased rate of formation of cholesterol is due to a diversion of the β-hydroxy-β-methyl-glutaryl co-enzyme A from acetoacetate formation, and an increased rate of its reduction to mevalonic acid by its reductase. The hormone appears to have no effect on the subsequent condensation of the mevalonate molecules to cholesterol. This finding is in keeping with the conclusion of Bucher, Overath and Lynen[6], who have suggested that the decreased rate of formation of cholesterol in starving animals is due to a reduction in the activity of the β-hydroxy-β-methyl-glutaryl co-enzyme A reductase.

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Blockage of Transformation of Tryptophan to Nicotinic Acid by Experimental Hepatitis in Mice

The existence of biochemical lesions in the complicated path from amino-acid to vitamin in man, and in experimental animals suffering from various diseases, has been identified by a number of authors analysing urinary metabolites of tryptophan by means of paper and column chromatography[1–6]. These analyses have shown an accumulation in the urine of metabolites proximal to the lesion.

In the present work we have studied the chromatographic picture of urinary metabolites of the tryptophan–nicotinic acid cycle both in normal mice and in mice with experimental hepatitis induced by mouse hepatitis virus strain 3 (*MHV*-3) at different stages of multiplication, in an attempt to identify the level of a possible biochemical lesion. Since the spontaneous elimination of urinary metabolites of tryptophan is small, we have preferred to follow them after a dose of L-tryptophan.

Mice of Swiss stock, weighing about 15 gm. and fed on Voght and Moller's diet were used. The programme of one of our experiments was as follows: 100 mice were divided into 5 (*A*, *B*, *C*, *D*, *E*) groups of twenty animals. Ten mgm. of L-tryptophan were injected subcutaneously into each animal in order to maintain the normal excretory pattern. After five days, mice from groups *A*, *B*, *C* and *D* were inoculated by the intraperitoneal route with 100 LD_{50} virus *MHV*-3.

To study the excretory pattern of metabolites of the biological degradation of tryptophan in different phases of the multiplication of the virus, a second dose of tryptophan was given to groups *A*, *B*, *C* and *D* at 12, 24, 48 and 72 hr. respectively. The mice in group *E*, not inoculated with the virus, were also given a second dose of tryptophan simultaneously with group *A*.

In the 24 hr. following the injection of tryptophan, the urine from each group of mice was collected in opaque glass vessels containing 2 ml. of 50 per cent glacial acetic acid (1: 1), and was diluted with water to a volume of 5 ml. The urine was then centrifuged. Dalgliesh's[7] method of chromatographic analysis for urinary metabolites in the tryptophan–nicotinic acid cycle was used, but omitting the absorption on carbon and the subsequent treatment with phenol.

The analysis of the urine of the control mice (*E*) injected with L-tryptophan showed the appearance of conjugates of the kynurenines (R_F 0·1–0·15) of kynurenic and xanthurenic acids, of acetylkynurenine and sometimes of free anthranilic acid.

In contrast, the urine of the mice inoculated with the virus showed the presence of all these metabolites (except anthranilic acid) in greater and more constant quantities, and in addition, the presence of kynurenine and of 3-hydroxykynurenine in notable quantities. The chromatographic picture also showed the progressive growth of the metabolic lesion paralleling the aggravation of the viral hepatic lesion.

Other metabolites, such as 3-hydroxyanthranilic acid, have never been observed on normal mice or in mice inoculated with the virus.

From the results obtained, we conclude that in mice with experimental hepatitis caused by the *MHV*-3 virus a biochemical lesion occurs at the level of enzymic transformation of 3-hydroxykynurenine to 3-hydroxyanthranilic acid.

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Sucrases in *Phaseolus vulgaris*

During work on the oligosaccharides of *Phaseolus vulgaris*, L. (namely, verbascose, stachyose, raffinose and sucrose[1–3]—the raffinose family of oligosaccharides[1]), an examination was made of the enzymes catalysing the hydrolysis of these saccharides. α-Galactosidase and sucrase activities were found in the ungerminated and germinated seeds.

The α-galactosidase activity proved to be typical of that described by Courtois *et al.*[4–7] in extracts of coffee and haricot beans and by Pridham[8] in extracts of *Vicia faba*, since it removed galactose from verbascose, stachyose and raffinose *in vitro* to give sucrose. From the higher homologues, verbascose and stachyose, the galactose was removed stepwise