Table 2. Effect of Treatment of Concentrate and Extract with Di-isopropyl-phospho-fluoridate on the Stability of AHF in the Concentrate and Mixtures of the Extract and Residue

Sample	Percentage AHF remaining after 7 hr. at 25°	Fibrinogen life (hr. at 37°)
Original concentrate	74	130
Residue	97	76
Extract	—	> 130
Residue + extract	70	—
Original concentrate treated with DFP	96	—
Residue + DFP-treated extract	94	—

See footnotes to Table 1.

These results suggest that the instability of human antihæmophilic factor in solutions of concentrates is largely due to the action of a DFP-sensitive component other than plasmin; the nature of this component is under investigation. The enhanced stability of antihæmophilic factor in DFP-treated concentrates should facilitate further purification.

Fuller details of this work, for the support of which we are indebted to the Medical Research Council, will be published elsewhere.

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HISTOCHEMISTRY

Effect of 3-Methylcholanthrene Crystals and Solutions on the Chorioallantoic Membrane of the Developing Chick Embryo

Metaplastic changes of the chorioallantoic membrane of the developing chick embryo, such as keratinization and epidermoid pearl formation, have been reported by several authors[1-4].

We have subjected the chorioallantoic membrane of chick embryos of various ages to single topical applications of crystals of 3-methylcholanthrene as well as to various concentrations of the carcinogen dissolved in acetone or polyethylene glycol. In order to determine the effects of carcinogen *per se*, the vehicles alone, as well as particles of egg-shell, have also been tested.

The results so far have shown that the response depends in part on the age of the embryo. After initial necrosis and inflammation, the most impressive reaction of the chorionic ectoderm was metaplasia, such as stratification of epithelium and of prickle cell, keratin and epithelial pearl formation. An additional reaction seen in 10-day chorioallantoic membranes subjected to methylcholanthrene crystals was the presence of cords, columns and clusters of epithelial cells deep in the mesenchyme. Such accelerated response under similar conditions of embryonic age was not noticed with the application of the non-carcinogenic irritant.

The egg-shell powder produced a characteristic foreign-body giant cell response.

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PATHOLOGY

Urinary Phenolic Acids in Infective Hepatitis

An abnormal urinary excretion of aromatic metabolites of tyrosine (that is, tyrosyluria) is well known to occur in scurvy. Recently, Robinson and Smith[1,2] reported tyrosyluria in severe stress (spinal shock, burns, etc.) and various pathological conditions, in patients receiving an adequate dietary intake of ascorbic acid.

Since tyrosine is metabolized in the liver, it seemed worth while examining the urines of patients suffering from parenchymatous liver damage for tyrosine metabolites.

Urine specimens were collected over a period of 24 hr. from 6 patients suffering from infective hepatitis. All the patients had increased serum glutamic-oxalacetic transaminase activities, reduced serum albumin concentrations and raised thymol turbidities. Their dietary intake of ascorbic acid was adequate.

The urinary phenolic acids were extracted by the method of Hill *et al.*[3]. A quantity of extract equivalent to the volume of urine passed in 1 min. was subjected to two-dimensional paper chromatography using isopropanol–ammonia followed by *n*-butanol–pyridine–water[2]. After drying, the chromatograms were sprayed with 10 per cent sodium carbonate solution, dried again and resprayed with diazotized sulphanilic acid. Hydrolysed urine samples were not examined.

Using this technique, *p*-hydroxyphenylacetic acid is almost invariably found in small quantities in normal urine, but *p*-hydroxyphenyllactic acid is rarely detected.

All six patients suffering from infective hepatitis excreted increased amounts of both *p*-hydroxyphenylacetic and *p*-hydroxyphenyllactic acids. A seventh patient with hepatic cirrhosis also excreted increased amounts of these phenolic acids. None of the patients excreted significantly increased amounts of 4-hydroxy-3-methoxymandelic acid.

It seems probable that parenchymatous liver damage can be the commonest cause of tyrosyluria. The question arises whether the tyrosyluria which Robinson and Smith[1,2] observed in stress and in various pathological conditions was due to some degree of liver dysfunction. This is being investigated. Preliminary results show that in myocardial infarction this is probably true.

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