action of sulfanilamide, its effect on rats fed diets containing sulfaguanidine was explored. The action of yeast was also tested. At the time of the completion of this work, Black *et al.*[4] reported that liver extract, and to a lesser degree p-amino benzoic acid, prevented the growth-inhibiting effect of sulfaguanidine in rats fed a purified ration. Our results on growth are in general agreement with theirs.

We wish to report the extensive alterations observed in the thyroids of rats fed sulfaguanidine[5] in a diet containing synthetic B vitamins[6] and p-amino benzoic acid, or in a diet containing yeast.

The basal ration was composed of purified casein 200, sucrose 600, lard 40, salts 60, 2-methyl-1.4-napthoquinone 0.005, and 13 drops of haliver oil fortified with viosterol. To this mixture was added either 100 parts of dried yeast, or 5 mg each of thiamin, riboflavin and pyridoxin, 15 mg of calcium pantothenate, 250 mg of choline, and 500 mg of cystine, with or without 2.5 gm of p-amino benzoic acid. When added, sulfaguanidine was incorporated at a 1 or 2 per cent. level. The distilled drinking water contained 20 mg of iodine and 40 mg of potassium iodide per liter one day a week.

Rats from our stock colony were placed on these diets at 21 to 23 days of age. Animals receiving sulfaguanidine were sacrificed at periods varying from 6 to 16 weeks. Without exception their thyroids were hypertrophied and hyperemic. The glands were 3 to 8 times larger than those of the control animals receiving the same diets without sulfaguanidine. Rats on the diets containing synthetic B vitamins (without p-amino benzoic acid) plus sulfaguanidine developed bleeding from the anterior corner of the eye, which later involved the whole eye. This symptom was prevented by p-amino-benzoic acid in rats receiving 1 per cent. of sulfaguanidine, but not in those receiving 2 per cent. It was always prevented by yeast.

A second experiment was conducted in which rats on the yeast diet plus 1 or 2 per cent. of sulfaguanidine were killed at the end of 4 weeks and their thyroids removed for sectioning. The glands were hyperemic and 3 to 4 times larger than those of the control animals on the yeast diet without sulfaguanidine. Histologically, the thyroids of the 2 per cent. sulfaguanidine rats showed marked hyperplasia. The epithelium was distinctly columnar, and in most follicles so increased and invaginated as to nearly extinguish the lumen. But few of the lumina contained colloid, and where present it was vacuolated and shredded. The connective tissue was not appreciably increased, but the glands were very vascular. In the rats receiving 1 per cent. sulfaguanidine, the thyroids contained a little more colloid; and the columnar epithelium was not so invaginated, otherwise the picture was the same. The thyroids of the control rats were normal. They contained an abundance of colloid and the epithelium was of the cuboidal type. Histological examination of the kidneys of these sulfaguanidine animals revealed no abnormalities. The bladders and ureters contained no visible calculi. The growth of the rats on both levels of the drug equaled that of the controls during the 4-week experimental period, and no gross symptoms were observed. (After the fourth week there is a retardation in the rate of growth.) It is of interest to note that Richter and Campbell[7] have very recently reported similar thyroid changes in rats fed phenylthiocarbamide.

At present we are investigating the effect of increasing the iodine intake at the beginning of the experiment and after the thyroid has hypertrophied. We are also testing the action of other "sulfa" drugs, sulfanilic acid, guanidine and thiourea on the thyroid in several species. The results of these studies together with a detailed account of the above observations will be published elsewhere in the near future.

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EFFECT OF ULTRAVIOLET LIGHT ON POLYCYCLIC HYDROCARBONS IN STEROL SURFACE FILM SYSTEMS

IN the course of a detailed study of the interaction of carcinogenic and other polycyclic hydrocarbons with sterols and other cellular constituents in mixed films at the air-water interface,[1] the conditions under which such hydrocarbons undergo ultraviolet decomposition have been investigated. Since these experiments may have some bearing on (*a*) the mechanism of detoxification and disposal of hydrocarbons subject to carcinogenic experiments,[2] and (*b*) the intense photodynamic effects exhibited by polycyclic hydrocarbons on bacteria[3] and other cells,[4] a preliminary statement of the results is presented here.

In such mixed films with sterols, certain polycyclic

[4] S. Black, J. M. McKibbin and C. A. Elvehjem, *Proc. Soc. Exp. Biol. and Med.*, 47: 308, 1941.

[5] We are indebted to Lederle Laboratories, Inc., for the sulfaguanidine used in this experiment.

[6] We are indebted to Merck and Company, Inc., for supplies of the synthetic vitamins.

[7] C. P. Richter and K. H. Campbell, *Arch. Path.* (in press).

[1] W. W. Davis, M. E. Krahl and G. H. A. Clowes, *Jour. Am. Chem. Soc.*, 62: 3080, 1940.

[2] L. Velluz, *Compt. rend. Acad. Sci.*, 206: 1514, 1938.

[3] A. Hollaender, P. A. Cole and F. S. Brackett, *Am. Jour. Cancer*, 37: 265, 1939.

[4] I. Doniach and J. C. Mottram, *Nature*, 145: 748, 1940.

hydrocarbons can be held in two-dimensional solution or in two-dimensional molecular association with the sterols.[1] By application of pressure to the mixed films, the hydrocarbon molecules can be displaced from their area-determining positions in the film, passing into an excess phase outside of the film, but in close proximity to it. The formation of the excess phase is characterized by the appearance of light-scattering particles which display Brownian movement; when the pressure is released, those hydrocarbons which are capable of solution type interaction with sterols can rapidly re-enter the films with the complete disappearance of the excess phase.

The present experiments show that, when in this excess phase, certain hydrocarbons are extremely sensitive to ultraviolet photodecomposition; on the other hand, when held between sterol molecules in the area-determining film phase, the same hydrocarbons are not subject to photodecomposition at the intensity of ultraviolet light employed.

A 15-watt G. E. germicidal lamp, supplied with an aluminum reflector, was mounted over the previously described[1] film tray at a vertical distance of 8 inches from the surface. The predominant radiation was at 2,537 A., but the principal mercury emission lines up to 4,338 A. were also prominent. The mixed films were put under pressures which gave the desired proportion of the hydrocarbon in the excess phase, irradiated for 15 minutes, and the force-area characteristics of the resulting film were then studied.

Typical results with 10-amyl-1,2-benzanthracene in mixed films with cholestanol may be cited as a convenient example. This hydrocarbon, when in the excess phase, was converted by irradiation into a surface active substance which was adsorbed, in addition to the sterol, at the water surface; this surface active substance could be detected and characterized by its surface film behavior. The conversion of the hydrocarbon was partially blocked by inclusion of a reducing agent, such as pyrogallol, in the water upon which the mixed film was originally spread; this indicated that the excess phase was in the water beneath the film and that the photodecomposition of the hydrocarbon took place there. This point of view was supported by two other observations: (1) when an aqueous suspension of 10-amyl-1,2-benzanthracene was introduced into the water phase immediately under a sterol film, the hydrocarbon molecules entered the film in much the same manner as hydrocarbons enter the film from the excess phase; (2) when an aqueous suspension of 10-amyl-1,2-benzanthracene was irradiated in bulk in the presence of dissolved oxygen, the substance formed had exactly the same surface properties as the material produced by irradiation of the 10-amyl-1,2-benzanthracene in the excess phase of the mixed films.

Similar experiments were performed with a number of other polycyclic hydrocarbons, including 9,10-dimethyl-1,2-benzanthracene, numerous mono-alkyl-1,2-benzanthracenes, and several mono- and di-alkyl-chrysenes. In each case where photodecomposition occurred in the film experiments, irradiation of the same hydrocarbon in bulk suspension caused loss of its characteristic ultraviolet absorption at wavelengths greater than 2500 A.

The photodecomposition product isolated after irradiation of the bulk aqueous suspension of 9,10-dimethyl-1,2-benzanthracene corresponded, in its analysis for C and H and in its lack of the characteristic absorption spectra of 1,2-benzanthracene derivatives, to the photo-oxide produced from 9,10-dimethyl-1,2-benzanthracene in CS_2 solution according to the method of Cook and Martin.[5] The relative ease of photodecomposition of the hydrocarbons in the excess phase and in bulk aqueous suspension was found to be the same as the relative photooxidizability in CS_2 as observed by Cook and Martin.

In contrast to the parent hydrocarbons, the photo-oxidation products of 10-amyl-1,2-benzanthracene and other alkyl-1,2-benzanthracenes were found to exhibit no interaction with sterols in surface films. The photo-oxides which were prepared from carcinogenic hydrocarbons by Cook and Martin[5] were found by them to be non-carcinogenic.

SUMMARY

Certain alkyl-1,2-benzanthracenes and other polycyclic hydrocarbons, when irradiated either in bulk aqueous suspensions or in the comparable excess phase under mixed surface films, were converted rapidly by ultraviolet light to photo-oxides. When held in two-dimensional solution or molecular association with sterols in mixed surface films at the air-water interface, the hydrocarbons were protected from such photodecomposition. In the one case where the comparison could be made photo-oxidation was accompanied by a loss of the ability of the hydrocarbon to interact with sterol films.

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A PHYTOPATHOGENIC BACTERIUM FATAL TO LABORATORY ANIMALS

Phytomonas polycolor (Clara) Bergey *et al.*, the causal agent of a bacterial leafspot disease of tobacco, was first isolated and described by Clara[1] in the

[5] J. W. Cook and R. H. Martin, *Jour. Chem. Soc.*, 1940, 1125.

[1] F. M. Clara, *Phytopath.*, 20: 691, 1930.