thetic detergent sodium dodecyl sulfate[1] in 0.5 per cent. solution slowly splits the large tobacco mosaic virus protein into smaller molecules and separates the nucleic acid from the protein, but does not denature the protein as shown by solubility and digestibility tests. Unfortunately the tests for denaturation were not carried out in an altogether satisfactory way. In any case, tobacco mosaic virus is much more resistant to sodium dodecyl sulfate than hemoglobin or egg albumin.

Hemoglobin is denatured by extraordinarily small amounts of Duponol PC. The addition of only 2 mg of Duponol PC to 10 mg of beef methemoglobin dissolved in 10 cc of pH 6.8 phosphate buffer solution suffices to denature the protein. This denaturation of methemoglobin in dilute solution can be followed optically. Native methemoglobin is a brown compound with a band in the red. Denatured methemoglobin in neutral Duponol solution is a red compound with no band in the red.

If 0.5 gm of Duponol PC is added to 10 cc of 2.5 per cent. hemoglobin and the excess Duponol is then removed by dialysis against water, the hemoglobin remains in solution after the dialysis and retains the color and spectrum of denatured methemoglobin. This result shows the great affinity of Duponol PC for denatured methemoglobin. The dialyzed hemoglobin is precipitated by 0.1 saturated ammonium sulfate, which does not precipitate native methemoglobin. It is digested by trypsin, which does not digest native methemoglobin. Duponol PC in sufficiently high concentration can prevent the precipitation by 0.1 saturated ammonium sulfate and can inactivate trypsin. This is why the excess Duponol is removed by dialysis before the solubility and digestibility tests.

It takes about 10 times as much of the bile salt, sodium glycholate, as of Duponol PC to denature hemoglobin.

0.2 N trichloracetic acid does not precipitate hemoglobin in 1 per cent. solution if 1.3 per cent. Duponol PC is present.

Hemoglobin can be estimated colorimetrically as denatured methemoglobin in 1 per cent. Duponol PC solution. This colorimetric procedure has two advantages over the usual acid hematin procedure. The amount of light adsorbed does not change with time and it is the same whether the Duponol is added to oxyhemoglobin or to methemoglobin.

Bacteria do not grow in the hemoglobin solution containing 1 per cent. Duponol, not even at 37° C.

The synthetic detergents and the bile salts all have the same type of hydrophobic-hydrophilic structure. Each synthetic detergent and bile salt consists of a large hydrophobic part with a small hydrophilic part attached to it. In actual chemical structure, the detergents and bile salts vary considerably. The hydrophilic group may be an acid group, such as OSO_3^-, or a basic nitrogen group. The hydrophobic part may consist of a long straight fatty acid chain, or it may contain a naphthalene or other ring structure.

The denaturation of protein and the solution of denatured protein by detergents and bile salts which differ widely in chemical composition must be attributed to the one property all the detergents and bile salts have in common, their general hydrophobic-hydrophilic character.

Bile salts have been used to extract the photosensitive protein pigment of the eye[3] and to extract a chlorophyll compound from the chloroplasts of spinach.[4] Chlorophyll, I have found, can be extracted from spinach by Duponol PC much more effectively than by bile salts. It is now clear, however, that a protein extracted by bile salts or detergents may no longer be in its original native undissociated form.

Physiologically, the bile salts emulsify fats, activate lipase and promote the absorption of various substances. The possibility must now be considered that the physiological reactions of the bile salts, like their reactions with proteins, depend not on their specific structures, but on their general hydrophobic-hydrophilic character, and that other substances with the same general hydrophobic-hydrophilic character can act as physiological substitutes for the bile salts.

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THE CONFIGURATION OF THE GLUTAMIC ACID OF ADENOCARCINOMA PROTEIN

RECENTLY, Kögl and Erxleben[1] have reported that the glutamic acid of malignant tumor proteins is present in partly racemized form. Chibnall, Rees, Tristram, Williams and Boyland,[2] using the Foreman procedure, found only l(+) glutamic acid in Crocker mouse sarcoma and in bronchial carcinoma. Kögl and Erxleben[3] have offered evidence to show that the Foreman procedure is unsuited for the isolation of the unnatural glutamic acid isomer (d(−) glumatic acid). It is reported in a recent issue of *Science News Letter* (36:37, 1939) that the results of Kögl and Erxleben have been confirmed by Dr. E. Schroeder, of the Biochemical Research Foundation of the Franklin Institute.

[3] W. Kühne in L. Hermann, "Handbuch der Physiologie," Leipzig, 1879, 3: 264.
[4] E. L. Smith, SCIENCE, 88: 170, 1938.
[1] Kögl and H. Erxleben, *Zeit. f. physiol. Chem.*, 258: 57, 1939.
[2] A. C. Chibnall, M. W. Rees, G. R. Tristram, E. F. Williams and E. Boyland, *Nature*, 144: 71, 1939.
[3] F. Kögl and H. Erxleben, *Nature*, 144: 111, 1939.

Using the method employed by Kögl and Erxleben, we have isolated glumatic acid from two adenocarcinomas of the large intestine, and from a sample of normal intestinal tissue adjacent to one of the carcinomas. Our results indicate that approximately 41 per cent. of the glumatic acid in the first carcinoma, and approximately 26 per cent. of the glumatic acid in the second, were present as d(−) glutamic acid. These findings confirm the report of Kögl and Erxleben. Our experimental data are summarized below:

(1) *Adenocarcinoma of the sigmoid colon.* Initial weight, 97.5 g. Weight of dried combined protein, 14.0 g. Glutamic acid hydrochloride isolated, 97 mg. M. P., 205° C. $[\alpha]_D$, +5.5°.

(2) *Adenocarcinoma of the cecum.* Initial weight, 108.5 g. Weight of dried combined protein, 15 g. Glutamic acid hydrochloride isolated, 140 mg. M. P., 201° C. $[\alpha]_D$, +15.0°.

(3) *Normal tissue adjacent to adenocarcinoma of the cecum.* Initial weight, 150 g. Weight of dried combined protein, 19.1 g. Glutamic acid hydrochloride isolated, 19 mg. M. P., 205° C. $[\alpha]_D$, +31.0°.

It will be noticed that the yield of glumatic acid obtained from the adenocarcinoma of the cecum was considerably greater than that obtained from adjacent normal tissue. No conclusion can be drawn from this, however, since the insolation of the glumatic acid was not quantitative.

We are indebted to the departments of pathology and surgery of the University of Minnesota Medical School for giving us the tissues analyzed immediately after operation. Microscopic diagnoses of the tumors were made by Dr. Robert Hebbel.

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POLIOMYELITIC VIRUS IN SEWAGE

WITHIN the past two years it has become increasingly apparent that poliomyelitic virus may be readily isolated from the stools of human patients during acute and convalescent stages of this disease. Many reports now testify to the ease with which this can be accomplished.[1] Consequently, whenever poliomyelitis occurs within a city, there is at least an opportunity for this virus to enter the local sewage system. But actually poliomyelitic virus has never been demonstrated in sewage, and there is no information as to the possible amounts which may be there, nor the length of time which this virus might survive in this medium.

An opportunity to test this situation has recently occurred in the City of Charleston, South Carolina, where poliomyelitis assumed epidemic proportions during the months of May, June and July of this year. From this same South Carolina epidemic a strain of virus was isolated from a specimen of feces received in our laboratory on July first. The stool was from a child (B.Cr.) three years old, who had contracted paralytic poliomyelitis in a small up-state town, but who had been hospitalized in Charleston. Part of the stool suspension was inoculated intraperitoneally into one rhesus monkey (No. 1213) on July fourth, and typical experimental poliomyelitis resulted after an incubation period of five days. This strain has been successfully carried to its third passage (monkeys No. 1230 and 1242) and is characteristic of poliomyelitic virus.

Subsequently, through the kindness of Dr. L. Banov, health officer of the City of Charleston, samples of sewage were collected by two of us (J. R. P. and J. D. T.) from a few sites throughout the city during the period of July 7 to 14, 1939. Particular attention was paid to one pumping station in which the sewage came not only from the hospital where poliomyelitis patients were isolated, but also from that part of the city where most of the cases had arisen. One sample (C) of this sewage, amounting to about 8 liters in volume, was collected on July 11 in a tall glass bottle and allowed to stand for a period of twenty-four hours. Ice was packed about the base of the bottle for part of this time. After the first two hours, a sample of sediment (C-1) amounting to 200 cc was removed and to it 24 cc of ether were added for bactericidal purposes. This sample was then taken to New Haven, where, on July 12 and 13, 75 and 45 cc, respectively, were inoculated intraperitoneally into one monkey (No. 1227). Another sample (C-2) amounting to 700 cc of the sediment, was removed from the original 8-liter specimen on July 12; 70 cc of ether were added to it, and the specimen was sent to New Haven, where one monkey (No. 1232) was inoculated intraperitoneally with 125 cc on July 14. Both of these animals developed experimental poliomyelitis. The incubation period in monkey 1227 was eight days from the last inoculation, and in monkey 1232 it was seven days. After a brief febrile period both animals developed quadriplegia, and both were prostrate on the third day of their disease. Monkey 1232 died on this day; no. 1227 was sacrificed. Spinal cord lesions, demonstrable histologically, were found to be very extensive in both animals. It is unusual in our experience to see rhesus

[1] (*a*) P. H. Harmon, *Jour. Am. Med. Asn.*, 109: 1061, 1937; (*b*) J. D. Trask, A. J. Vignec and J. R. Paul, *Jour. Am. Med. Asn.*, 111: 6–11, 1938; (*c*) P. Lépine and P. Sédallian, *Compt. rend, Acad. d. sci.*, 208: 129–130, 1939; (*d*) S. D. Kramer, B. Hoskwith and L. H. Grossman, *Jour. Exp. Med.*, 69: 49–67, 1939; (*e*) C. Kling, G. Olin, J. H. Magnusson and S. Gard, *Bull. Acad. méd.*, 121: 826–831, 1939; (*f*) J. R. Paul, A. J. Vignec and J. D. Trask, *Trans. Asn. Am. Phys.* (to be published). Also personal communications from: G. Y. McClure, Division of Laboratories and Research, N. Y. State Department of Health; and from C. Armstrong, National Institute of Health, Washington, D. C.