observations were also made, on a warmed microscope stage, of the motility of the exudate cells and the myelocytes in the marrow. Thiouracil in 100 mg per cent. concentration did not appear to affect the motility of either type of cell.

Finally, 13 attempts to protect the marrow and exudate cells from the depressant action of thiouracil on respiration by adding 10 mg per cent. pyridoxine[7] *in vitro* all yielded negative results, as have 4 preliminary experiments with diluted liver extract.

A detailed account of these experiments will be published elsewhere.

SUMMARY AND CONCLUSIONS

Thiouracil in 100 mg per cent. concentration induces a small but significant inhibition of respiration of rabbit bone marrow cells, the effect upon the myeloid elements being the more striking. By comparing the results with those obtained with the polymorphonuclear cells of rabbit peritoneal exudates, it is inferred that the more immature marrow cells are more seriously affected. No effects on cell motility have been observed, and attempts to oppose the effect of thiouracil with pyridoxine have been unsuccessful. While caution must necessarily be exercised in relating the results of the present *in vitro* experiments to the known toxic effects of thiouracil in patients, the methods outlined above might be employed to test possible toxic effects of new therapeutic drugs, or the action of agents proposed to protect the marrow from harmful effects.

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ANTAGONISTIC ACTION OF A RED MOULD PIGMENT AGAINST BACTERIA OF THE TYPHOID-PARATYPHOID-DYSENTERY GROUP

A MOULD which produces a red pigment was isolated in our laboratory from human hair planted on Sabouraud's agar.

The mould grows rapidly at room temperature, spreading within 3 to 4 days over the whole surface of an agar plate. At first, the colony is fluffy and pure white; later, within 2 to 3 weeks, it becomes slightly yellowish. A striking characteristic of the mould is the production of a dark red pigment readily diffusing in the medium. No fruiting structures were observed regardless of medium or conditions of growth. The absence of these structures made impossible the identification of the mould.[1] However, it bears no resemblance to any of the known pathogenic fungi. Essential for the formation of the pigment is an organic source of nitrogen (peptone, proteose, casein, bran) and the presence of one of the sugars (dextrose, sucrose, maltose). No pigment is formed on media containing lactose or starch or on Czapek-Dox medium which is used for Penicillium cultures and contains inorganic nitrogen compound. The following medium gives a satisfactory production of pigment: Proteose 1.0, Dextrose 4.0, Agar 0.25, Water 100.0.

The medium is distributed in flasks or bottles in shallow layers 1.5 to 2 cm deep. Three or four days after inoculation, a compact white felt of mycelium develops on the surface of the medium. The formation of pigment which diffuses in the medium begins on the second or third day and attains its peak about the eighth to the tenth day. The pigment can be extracted from the medium by the method used in production of penicillin. After acidification with phosphoric or hydrochloric acid to pH2 the culture medium is shaken with an equal volume of an organic solvent, like ether, amylacetate, butyl alcohol, chloroform. From this solvent the pigment is reextracted by shaking with a phosphate buffer solution of pH7.

The activity of the pigment solution was tested by the Oxford cup method. When typhoid, paratyphoid or dysentery (strains of Shiga and Flexner) bacilli not susceptible to the action of penicillin were seeded on agar plates there always appeared a clear zone of complete inhibition of growth around the cup placed on the surface of agar and filled with the pigment solution. The diameter of the clear zone was 20 to 30 mm. On the other hand, gram-positive bacteria, sensitive to penicillin, such as staphylococci, streptococci, pneumococci and subtilis, were in no way affected by the pigment.

Cultures of the mould grown on media which did not produce pigment did not show antibiotic action—a finding which supports the assumption that the antibiotic properties are intimately connected with the pigment.

The pigment solution is stable and does not lose its antibiotic property after autoclaving, acidification (to pH2) or alkalinization (pH10).

After intravenous injection into a rabbit the pigment, within several minutes, appears in the urine from which it may be recovered in the usual way (acidification, extraction with organic solvent, reextraction with a buffer phosphate solution).

The pigment is not affected by the gastric secretion. When introduced by a tube into the stomach of a

[7] M. M. Cantor and J. W. Scott, SCIENCE, 100: 545, 1944.

[8] These studies were aided by a grant from the John and Mary R. Markle Foundation.

[1] This fact was confirmed by Dr. E. Muskatblit, of New York University, and by K. B. Raper, of the Northern Regional Research Laboratory, U. S. Department of Agriculture, to whom Dr. Muskatblit sent the culture for identification. To both I wish to express my gratitude.

rabbit the pigment within one hour can be demonstrated in the blood and urine.

Animal experiments are now under way to study the toxicity and effectiveness of this antibiotic.

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THE Rh AND Hr FACTORS IN CHIMPANZEES[1]

THE purpose of this paper is to report the results of tests for the Rh blood types and Hr factor on ten chimpanzees, three jungle-born and seven colony-born.

In the Rh and Hr tests,[2] the bloods of all ten chimpanzees behaved alike. In the tests for the Rh blood types, with sera anti-Rh_0, anti-Rh′ and anti-Rh″, the reactions were either negative or weak. When any agglutination occurred, this proved to be due to heteroagglutinins rather than specific Rh agglutinins, as was proved by absorption tests. Thus, absorption of the sera with chimpanzee blood removed the agglutinins for chimpanzee blood without affecting the reactivity of the serum for Rh-positive human blood; while absorption with human Rh-positive blood removed the agglutinins for human blood without affecting the reactions of the sera with chimpanzee blood. These results, therefore, indicate that all ten chimpanzees are Rh negative.

That this conclusion is correct was proved by tests for the Hr factor. In tests with an exceptionally potent anti-Hr serum it was found that the chimpanzee bloods were all agglutinated strongly and to the same titer of the serum (about 250) as human Rh-negative blood. Absorption with chimpanzee blood removed the agglutinin for human Rh-negative blood as well as the reaction for chimpanzee blood and, conversely, absorption with human Rh-negative blood destroyed the reactivity of the Hr serum for chimpanzee blood. Moreover, the anti-Hr agglutinin was absorbed equally well by equivalent volumes of chimpanzee and human Rh-negative red cells.

These investigations are being continued, and additional chimpanzees at the Yerkes Laboratories will be tested.* Most likely, the other chimpanzees will also give reactions corresponding to the human Rh-negative type. Perhaps this uniformity in the reactions of chimpanzee bloods is the final result of the selective action of isoimmunization in pregnancy, without the interference of racial crossing such as is apt to occur in man.[3]

[1] Aided by a grant from the United Hospital fund of New York City.

[2] For technique see: A. S. Wiener, J. P. Zepeda, E. B. Sonn and H. Polivka, *Jour. Exp. Med.*, 81: 559, 1945.

* After this article was submitted for publication, blood from five additional chimpanzees was tested, with similar results in the Rh and Hr tests.

[3] A. S. Wiener, SCIENCE, 96: 407, 1942.

In conclusion it should be mentioned that nine of the chimpanzees gave reactions corresponding to group A, while one gave reactions corresponding to group O. This agrees well with previous reports on a total of 92 chimpanzees, of which 81 belonged to group A and 11 to group 0.[4] The bloods of all ten chimpanzees reacted strongly with our anti-M serum, in conformity with the previous finding that all chimpanzees possess M-like agglutinogens.[5, 6] The anti-N serum which we had available did not agglutinate the chimpanzee bloods, but this does not necessarily contradict the conclusion from tests with other anti-N sera that chimpanzee blood also contains N-like agglutinogens.[6, 7]

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ACCUMULATION OF DDT IN THE BODY FAT AND ITS APPEARANCE IN THE MILK OF DOGS[1]

THE high lipoid-water distribution ratio of DDT suggested that it might be preferentially stored in the adipose tissues of mammals fed DDT. The toxicological behavior of this compound pointed also to possible deposition in body fat. Such a preferential distribution was first indicated by feeding the dibrom analogue of DDT, 2,2-bis(p-bromophenyl)-1,1,1-trichloroethane, to rats and rabbits and determining the increase in tissue levels of bromine. The rise in the bromine content of the fat was many times that in the liver, kidney, brain or blood. These analyses, however, did not show the exact nature of the stored compound. It was not until the specific colorimetric method of Schechter and Haller[2] became available that the material stored in the fat was shown to be the unchanged DDT. The extent to which DDT will accumulate in the fat of chronically fed animals

[4] A. S. Wiener, "Blood Groups and Transfusion," 3rd edition, chapter XIX, C. C Thomas, Springfield, Ill., 1943.

[5] K. Landsteiner and P. Levine, *Jour. Exp. Med.*, 47: 771, 1928.

[6] A. S. Wiener, *Jour. Immunol.*, 34: 11, 1938.

[7] A. S. Wiener, *Am. Nat.*, 75: 199, 1943.

[1] A portion of the funds used in this investigation was supplied by a transfer, recommended by the Committee on Medical Research, between the Office of Scientific Research and Development and the Division of Pharmacology of the Food and Drug Administration.

[2] M. S. Schechter and H. L. Haller, *Jour. Am. Chem. Soc.*, 66: 2129, 1944.