

FIG. 2. Visible absorption spectra of solutions of chlorophyll *b* at 230° K (-----) and at 75° K (——) for preparations I and II. Concentrations are different at the two temperatures.

possible impurity. Finally, with the cooperation of Professor Livingston, the purified chlorophyll *b* on the chromatographic adsorption column was removed in 3 fractions. The spectra of these fractions succeeded only in showing that the lowest fraction on the column consisted of considerably more chlorophyll *b'* than had been suspected, whereas the other 2 fractions gave the same spectra at each temperature. Neither of them furnished the shoulders and associate features as sharply and definitely as had been exhibited by a preparation of chlorophyll *b* made several months earlier. We are forced to conclude that there exists at least one other component in chlorophyll *b*, with a probably similar spectrum that has hitherto not been isolated by this process.

Insufficient work has been done to determine the nature of these isomers. The accepted chemical structures of the chlorophylls allow for a number of possible isomers: first, in the enol-keto forms and, second, in the different mutual configurations about the 3 asymmetric carbon atoms. The rate of transformation of one isomer of chlorophyll *b* into the other was rapid even at the lowest temperature 150° K at which the new isomer was first detected as the temperature was raised. At any fixed temperature no increase in

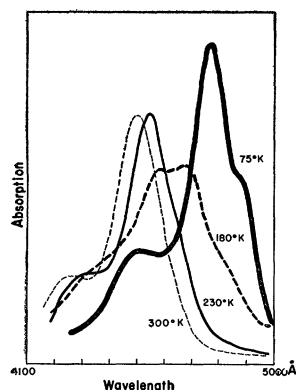


FIG. 3. Changes with temperature of the absorption spectra of the isomers of chlorophyll *b* in solution.

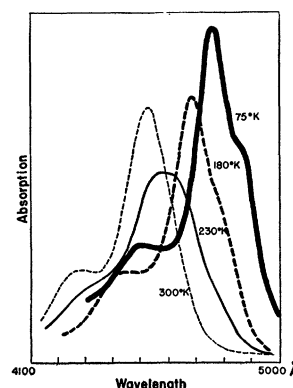


FIG. 4. Changes with temperature of the absorption spectra of the isomers of chlorophyll *b'* in solution.

its concentration was noted with time. However, a rise in temperature brought in at once an increase in the intensity of the spectrum of the high temperature form.

As the spectrum of the low temperature form begins to grow in, it modifies the over-all spectrum in a way that suggests the differences in appearance at room temperature of the spectra of chlorophylls in different solvents, especially in the 4,100 Å to 4,300 Å region (3). It is to be expected that the character of the solvent affects the energies of the isomeric transformations, especially if they are of the enol-keto type, and hence we are led to ask whether the difference in the spectra of the chlorophylls in different solvents may not be largely due to the change in the relative concentrations of the isomers present. Similarly, the well-known change in color of chlorophyll when it is adsorbed may to an appreciable degree be a shift in the equilibrium between the isomers induced by the adsorption process.

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The Pulmonary Circulation as a Source of Leucocytes and Platelets in Man

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The intravenous administration of epinephrine in man is followed by an immediate leucocytosis and thrombocytosis, the source of which has been variously attributed to the spleen and/or the bone marrow

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(1-3). However, leucocytosis and thrombocytosis following epinephrine have been observed in splenectomized patients (4), and it is doubtful that the bone marrow is capable of immediately delivering such large numbers of leucocytes and platelets into the circulation. As part of an investigation of the hematological role of the lung, 0.1-0.2 mg of epinephrine was administered intravenously to several patients with metastatic neoplastic diseases. By frequent sampling of blood from intravascular catheters placed in the right ventricle and an appropriate large artery, it was observed that the increase in number of leucocytes and platelets in the arterial samples preceded and exceeded that found in the venous blood by at least one to two circulation times (Fig. 1). The arterial-venous platelet difference was more marked and sustained than the leucocyte difference.

It would thus appear that the pulmonary circulation in man may act as an available source of leucocytes and platelets, which may be delivered rapidly into the peripheral circulation under the stimulus of intravenous epinephrine administration. The lung, therefore, must also be considered to contribute significantly to the leucocytosis and thrombocytosis following epinephrine in some patients under these conditions. Likewise, the pulmonary circulation warrants careful study in neutropenic and thrombopenic states that are not completely explained by current theories. These data do not prove that platelets are produced in

the human lung, as has been suggested by the studies of Howell and Donahue (5) in the dog, but merely illustrate that in some patients without panhematopenia, the lung may be stimulated to deliver platelets promptly into the circulation. The continued discrepancy between the arterial and venous platelet number suggests removal of some platelets in the peripheral circulation. Details of these studies will be published elsewhere.

References

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Effects of Male Hormone upon the Tail of the Slider Turtle, *Pseudemys scripta troostii*

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It is during the fourth or fifth year, usually, that the male slider turtle attains sexual maturity, as indicated by the presence of sperm in testes or vas deferens. At this time the tail grows rapidly and becomes notably longer than that of the female (1).

The manner in which the tail of the male is utilized in preliminary courtship (2) and in mating (3) has been described. The greater length of tail is necessary to consummate the mating process, since the length of the plastron of the male averages 13.5 cm, whereas that of the female averages 18.9 cm (based upon measurements of more than 800 specimens of each sex examined [4]). Measurements of the tails of skeletons of *Pseudemys* at the American Museum of Natural History also reveal that the tail of the male is definitely longer than that of the female, despite the fact that the same number of caudal vertebrae occurs in both sexes (24 ± 5).

It would thus appear that the greater length of the tail of the male slider represents a secondary sexual character and that it is subject to control by the male sex hormone. This is confirmed by the experiment to be described.

Two groups of juvenile sliders were secured for study. One averaged 5.5 cm plastron length, the other 3.5 cm. Ten of each group received pellets of testosterone propionate¹ (6.5 mg and 4.0 mg, respectively) in September 1948. Ten others of each group were retained as controls and were kept in separate aquaria. All specimens received similar care and food. Mortality averaged 20% among the larger specimens and 30% among the smaller, with no greater loss recorded among the treated turtles than the controls. The experiment was terminated in May 1950, when all animals then living were sacrificed.

¹ Generously supplied by Ciba Pharmaceutical Products.

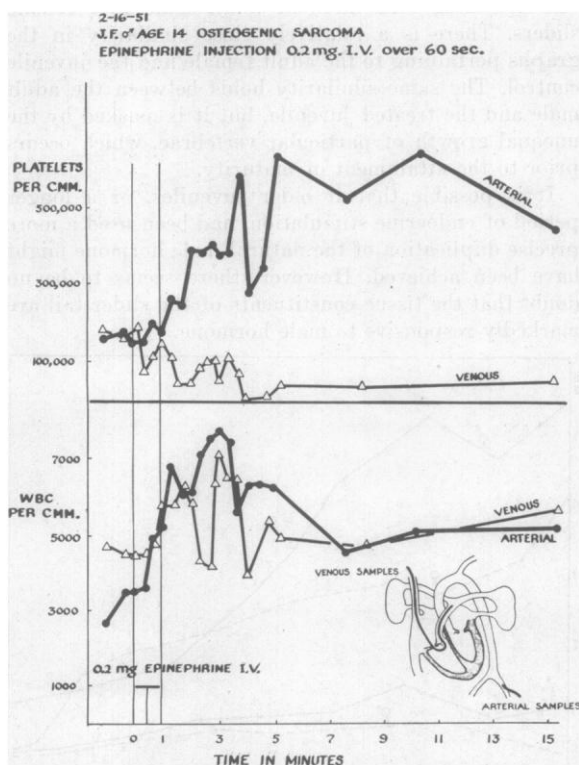


FIG. 1. Venous samples from the pulmonary conus; arterial blood from the femoral artery. There was no significant change in the red blood count in either arterial or venous blood throughout the period of study.