that about 11 percent of the total number of mitochondria is present in the nuclear fractions (*7*). The microsomal and supernatant fractions contain about equal amounts of activity. The intracellular distribution of acid and alkaline RNase activity of normal rat liver is similar to the distribution of acid RNase reported in normal mouse liver (*1*). The mitochondrial fraction activity, however, is lower in rat liver than in mouse liver.

It is of interest to mention that the alkaline RNase activity of the whole homogenate, when expressed in arbitrary units, is about twice that of the acid RNase activity. When the alkaline RNase activity is expressed per mg of substrate instead of per weight, it will be 2.5 times less than the acid activity. These results would suggest that acid and alkaline enzymes catalyze the hydrolysis of specific links in the RNA molecule. The possibilities are that the final substrate concentration for each of the acid and alkaline RNase would be an indirect measure of the concentration of chemical bonds specifically affected by each enzyme. Further work is being carried out in this laboratory to investigate the possible role of these enzymes as well as their intracellular distribution in normal and neoplastic tissues.

References

1. SCHNEIDER, W. C., and HOGEBOOM, G. H. *J. Biol. Chem.* **198**, 155 (1952).
2. DUVE, C., BERTHET, J., HERS, H. G., and DUPRET, L. *Bull. soc. chim. Biol.* **31**, 1242 (1949).
3. LASKOWSKI, M. *The Enzymes,* Vol. 1, Part 2, p. 959. New York: Academic Press, 1953.
4. MARTIN, A. J. P., and PORTER, R. R. *Biochem. J.* **49**, 215 (1951).
5. ZÖLLNER, N., and FELLIG, J. *J. Am. Phys.* **173**, 223 (1953).
6. SCHNEIDER, W. C., and HOGEBOOM, G. H. *J. Biol. Chem.* **183**, 123 (1950).
7. ALLARD, C., MATHIEU, R., DE LAMIRANDE, G., and CANTERO, A. *Cancer Research* **12**, 407 (1952).

Manuscript received January 11, 1954.

Leukocyte Counts in the Blood from the Tail and the Heart of the Mouse[1]

Horace Goldie, Arnold M. Jones, Herbert Ryan, and Melvin Simpson

Laboratory for Experimental Oncology, Meharry Medical College, Nashville, Tennessee

Law and Heston (*1*) found a marked difference in the total white count of heart and peripheral blood in albino mice. Similar findings were reported by other authors in the rat (*2–4*) and the guinea pig (*5*), but not in the dog (*6*). In order to find whether the concentration of leukocytes in the blood shows the same decreasing trend in various areas of the vascular system from the extreme periphery (tail tip) to the heart, we made counts in the same mouse from the tail tip, tail root, femoral vein, right heart, and left heart. The results from 15 C-57 mice were pooled and the average values as well as variation extremes are recorded in Table 1.

[1] This investigation was supported by a grant, C-2080, from the National Institutes of Health, U.S. Public Health Service, and partly by a grant from the Division of Biology and Medicine, Atomic Energy Commission.

TABLE 1. Blood leukocyte counts from peripheral and central areas of the vascular system in C57-6 mice (av. and variation extremes in 10 mice).

Tail tip	Tail root	Femoral vein	Right heart	Left heart
13,800 (8000–25,750)	6170 (5750–15,250)	4580 (2000–8000)	5720 (1500–8500)	2900 (1250–5500)

It appears from Table 1 that total leukocyte counts from the same vascular area showed considerable variations in different mice, but in no instance was the leukocyte count from the heart higher than the leukocyte count from any peripheral area of the same mouse. Thus, the leukocyte concentration in the blood decreased in the same mouse progressively during the passage from the capillaries to the left heart. This decrease could be estimated for each mouse by calculating the ratio T/H = count from the tail tip/count from the left heart. The average of this ratio and the extreme values for 10 CFW mice are recorded in Table 2. Moreover, this table indicates average T/H ratio and extremes for 4 series each of 10 mice treated by exposure to high temperature, by subcutaneous injection of anticoagulant (heparin, moranyl), and ether narcosis.

It appears from Table 1 that leukocyte concentration in the arterial blood of the mouse withdrawn from the left heart is lower than in any specimen from the venous system; that it increases to a maximum after passage through capillaries of the tail tip, but immediately after this passage (in the tail root) it decreases, and remains approximately on the same level until the blood reaches the right heart. Moreover, a new drop in the leukocyte concentration occurs after

TABLE 2. Leukocyte counts in CFW mice untreated and treated with anticoagulants, heat, or ether (av. and variation extremes in 10 mice).

	Tail tip	Left heart	Ratio T/H
Untreated	22,900 (18,750–33,750)	4230 (2250–6250)	5.7 (4.3–6.5)
Total body heating, 12 min at 94°	11,360 (3250–22,750)	5250 (2000–10,500)	2.1 (1.4–2.8)
Deep ether narcosis	13,790 (8500–20,500)	6500 (3250–11,500)	2.0 (1.1–4.0)
Heparin subcutaneously 100 units (1 mg)	14,750 (8000–22,500)	7900 (5000–10,750)	1.93 (1.15–2.3)
Moranyl (Fourneau 309 = Bayer 205) subcutaneously (5 mg)	31,500 (24,500–48,250)	20,715 (12,000–41,250)	1.7 (1.1–2.8)

the blood passage through the lungs into the left heart.

The usefulness and reliability of hematological work with blood specimens from the mouse tail is not affected by these data, as the ratio between leukocyte counts from the tail and the heart (T/H) was shown to be constant in all mice of the same (C57-6) strain (Table 1) and of another strain (CFW) (Table 2, line 1). Thus, reliable data on leukocyte concentration in the blood of the mouse can be obtained by taking blood from the same area of the tail or from the same section of the heart in all animals.

Table 2 shows that "equalization" of leucocyte counts was achieved as a decrease of tail counts without consistent and significant changes in the heart blood, thus indicating that the difference in counts was due to leukocyte concentration in the tail capillaries. It should be specified that equalization of counts by moranyl and heparin was observed only when their anticoagulant effect was well marked, thus indicating a relationship between these two effects. The blood of heated mice also showed considerable increase of clotting time in the specimens used for leucocyte counts. It follows that the "equalizing" effect of anticoagulants and perhaps also of total body heating on leukocyte counts cannot be attributed to reduction of resistance in peripheral vessels, (*2, 3*) and therefore the difference in tail and heart counts cannot be explained only by vascular phenomena as was suggested in experiments on rats (*2–4*). It may be presumed that some specific characteristics of fluid exchange between the vessels and the tissues in the tail of the mouse and the rat, but not of the dog (*6*), should be considered. The problem merits a special study on broader lines.

Summary. Leucocyte counts in blood specimens taken from various areas of the vascular system of the same mouse have shown that the leukocyte concentration decreased progressively during passage of the blood from tail capillaries through the femoral vein and right heart, to the left heart; red blood cell counts and leukocytic formula were found the same in all specimens. The ratio T/H (leukocyte number per centimeter in the tail and the left heart) was relatively constant (av., 5.7; variation extremes 4.3 and 6.5) for CFW and C57-6 mice. This ratio was decreased and thus the counts were equalized by the treatment of mice with total body heating, ether, or anticoagulants. For practical purposes, the relative constancy of the T/H ratio in all mice examined showed that reliable data on leukocyte concentration in the blood of the mouse can be obtained by taking blood from the same area of the tail or from the same section of the heart in all mice.

References

1. SNELL, G. D. *Biology of the Laboratory Mouse.* Philadelphia: Blakiston, 1941.
2. QUIMBY, F. H., SAXON, P. A., and GOFF, L. G. *Science* **107**, 447 (1948).
3. QUIMBY, F. H., and GOFF, L. G. *Am. J. Physiol.* **170**, 196 (1952).
4. ERSHOFF, B. H., and GARNES, H. G. *Science* **118**, 20 (1953).
5. ROOFE, P. G., LATIMER, N. B., MADISON, M., MOFFET, M., and WILKINSON, P. *Science,* **111**, 337 (1950).
6. AZARNOFF, D. L., BATTY, T. V., ROOFE, P. G., and MOFFET, M. *Science* **113**, 363 (1951).

Manuscript received January 29, 1954.

Communications

The White Pine Copper Deposit, Ontonagon County, Michigan

COPPER, typically as the mineral chalcocite, occurs over many square miles near White Pine, Ontonagon County, Mich., in the lower part of the Nonesuch shale. This copper deposit and the famous deposits of native copper in the Portage Lake lava series on the south shore of Lake Superior are distinctly separated, geographically by a distance of 45 to 70 mi and stratigraphically by the thickness of the Copper Harbor conglomerate.

The Nonesuch shale, of late Keweenawan age, is about 600 ft thick and is composed largely of gray siltstone. It overlies the 2300–5500 ft of red sandstones and conglomerates of the Copper Harbor conglomerate, which in turn overlies the middle Keweenawan Portage Lake lava series.

The copper-bearing zone at White Pine, mostly in the lower 20–25 ft of the Nonesuch shale, in local usage is divided into four stratigraphic units. These are, in ascending order, the lower sandstone (uppermost 4 or 5 ft of the Copper Harbor conglomerate), the parting shale, the upper sandstone, and the upper shale (lowermost beds of the Nonesuch shale). In the upper and parting shales the sequence of beds is almost identical and suggests cyclic sedimentation. This sequence and the distribution of sedimentary facies are attributed to two submergences, separated by an emergence, of a deltaic area.

Practically all the copper occurs in the upper and parting shales except in a small area near the White Pine fault, where it is abundant in the upper and lower sandstones. It is present in 5 different layers in amounts that average from 1 to 3 percent. The total amount of copper in each bed is generally higher where the bed is thick rather than thin, and thickness, in turn, seems to be greatest in areas that were hollows, away from the main channel or channels of the ancient delta. Copper content of the shale beds typically decreases as their sand content increases. The extent of individual copper-bearing beds 1–3 ft thick is measurable in square miles.

The local occurrence of copper in the upper and lower sandstone beds can be reasonably explained as the result of hydrothermal transportation from the White Pine fault up the dip of the relatively permeable sandstone to the crest of an adjacent anticline. Distribution of copper in the parting and upper shales, on the other hand, seems to be completely independent