ment of histamine sensitivity. The following experiments illustrate the results obtained.

The inability of quercitin to block the lethal action of histamine is demonstrated in the experiment outlined below. One hundred and fifty mice were immunized intraperitoneally with 2 billion cells of an antigenic *H. pertussis* vaccine, and 4 days later 100 of them received 1 mg quercitin intraperitoneally, the remainder serving as sensitized controls. Two groups of the treated mice were challenged with graded doses of histamine, using 10 mice/histamine dose, one group 2 hr after quercitin, the other 6 hr after quercitin. The LD_{50}, calculated by the method of Reed and Muench (*6*), was 0.08 mg for both groups, and for the 50 sensitized controls 0.07 mg. In this experiment quercitin exhibited no histamine-blocking properties. The possibility that these findings could be explained by the lack of absorption of quercitin seems unlikely in view of the following experiment, which can be explained only by the absorption of quercitin from the peritoneal cavity.

The experiment was for the purpose of demonstrating the ability of quercitin to inhibit the development of histamine hypersensitivity. Two groups of mice were immunized as before. The animals of Group I received daily intraperitoneal injections of 1 mg quercitin suspended in propylene glycol, and the animals of Group II received only propylene glycol and served as sensitized controls. After 4 days and approximately 17 hr after the last injection of quercitin, the mice of each group were challenged with graded doses of histamine. The LD_{50} for Group I was 1.2 mg of histamine, for Group II, 0.06 mg. Nonimmunized normal mice gave an LD_{50} of 12.5 mg. These results indicated that quercitin-treated mice would tolerate approximately twenty times more histamine than the immunized controls, although histamine hypersensitivity was not obliterated completely.

Chlorotrimeton, as an example of an antihistaminic, was tested both for its blocking properties and for its ability to inhibit the development of histamine sensitivity. These properties were demonstrated in the following manner: On the fourth day following immunization, half of a group of 100 immunized mice received an intraperitoneal injection of 0.3 mg Chlorotrimeton. Two and one-half hr later these mice and the immunized controls were challenged with graded doses of histamine. The LD_{50}s were 0.96 mg and 0.07 mg, respectively. When Chlorotrimeton was given in this dosage daily during the period of histamine-sensitivity development as in the quercitin experiment described above, it was found that histamine gave an LD_{50} of 0.12 mg in both the treated and the control groups. Thus, Chlorotrimeton, though capable of blocking hypersensitivity to histamine, would not prevent such hypersensitivity from developing.

Since previous workers (*1*) have shown that the ability to cause histamine hypersensitivity is a property of pertussis vaccines of demonstrable antigenicity, it was of interest to determine whether the inhibition of the development of histamine hypersensitivity would influence immunity. Mice were immunized as before and divided into two groups, one of which received daily injections of quercitin; the other group, serving as sensitized controls, received daily injections of the propylene glycol solvent. On the seventh day mice from each group were challenged with graded doses of histamine; it was found that in the quercitin-treated mice the LD_{50} was 1.02 mg, and in the controls it was 0.08 mg. The remaining mice from each group were challenged by intracerebral injections with approximately 100 LD_{50}s of pertussis organisms. Thirteen out of 15 (87%) survived in the quercitin-treated mice, and 19 out of 19 (100%) survived in the immunized controls. These results suggest that the suppression of the development of histamine sensitivity had not interfered seriously with immune phenomena. It should be noted that the mice under quercitin treatment were sickly and showed signs of toxicity, some dying before completion of the experiment. The toxicity was traced, in part, to the amount of propylene glycol used in the suspending menstruum and, in part, to the quercitin.

The data presented indicate the possibility that the diminution of hypersensitivity to histamine may be achieved by the inhibition of the development of such sensitivity rather than by the neutralization of the histamine reaction. It would appear further that this may be accomplished without serious interference with the development of immunity.

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The Life Span of Leucocytes in the Human[1]

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The life span of leucocytes has been measured by many techniques, most of which involved unphysiological experimental conditions. Using these methods, the life span has been estimated to vary from less than an hour to 3 weeks, depending upon the method used.

Shemin and Rittenberg (*1*), using a more physiological method, estimated the life span of red blood cells by measurement of the incorporation of glycine, labeled with N^{15}, into hemin. Because of the stability

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of the latter compound an accurate measurement of the red blood cell life span was obtained.

A similar method has been developed for estimation of the life span of leucocytes by measurement of the incorporation of P^{32} into the desoxypentose nucleic acids (DNA) of the leucocytes. The inertness of P^{32} in the DNA molecule after formation of the leucocytes has been found to be similar to the stability of N^{15} in hemin of the red blood cells, permitting a close estimation of the life span.

Hospitalized patients who had had a complete clinical work-up and were found to have normal total and differential white blood counts, who were free of diseases known to affect the white blood cells, and who could be studied for a prolonged period without receiving significant drug or other treatment were chosen for study. They primarily included patients with cardiovascular diseases.

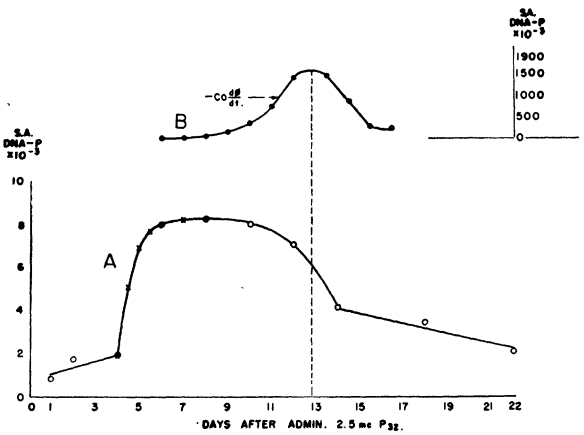

FIG. 1. Life span of leucocytes in the human. Curve *A*, 0 = specific activity of DNA (cpm/mg P) $\times 10^{-3}$; x = calculated points from equation $C(t) = Co\ (1 - e^{-\lambda t}) + 1900$, where Co = 6400 and $\lambda = 1.53$. Curve *B*, probability of cell survival time.

Each of 6 patients was given, by mouth, 2.5 mc of P^{32} as inorganic phosphorus. At 24 and 48 hr after administration, and every 2 days thereafter for periods up to 3 weeks, 50 ml of blood was drawn in a dry syringe and transferred immediately to a heparinized test tube. The blood was drawn at approximately the same hour in the morning. The red blood cells were settled by the addition of fibrinogen (*2*), the leucocytes were pipetted off, centrifuged, resuspended in their own plasma, and then centrifuged in a plastic straw. The contents of the straw were frozen, and a clean area of packed white blood cells was cut from the straw with a razor blade. The cells obtained were weighed, and the DNA fraction was precipitated by the method of McCarter and Steljes (*3*). The DNA was reprecipitated, dissolved, and then digested in nitric acid. Phosphorus was estimated by the method of King (*4*). For measurement of radioactivity the phosphorus was precipitated as ammonium molybdophosphate and filtered through sintered glass counting disks. The samples containing radioactivity were counted in a Tracerlab proportional flow counter to an accuracy of ±2 or 3%, except for a few with low activity, where the accuracy was ±5%.

In Fig. 1, curve *A*, the specific activity of the DNA-P^{32} (cpm/mg P) is plotted against time in days (open circles). Each point represents the average of determinations on 2–6 different individuals. The type of curve obtained, as has been pointed out by Shemin and Rittenberg, will result only if the uptake and release of isotope is not a random process, but depend on a selective factor such as the age of the cells. Osgood *et al.* (*5*) have recently shown that P^{32} is not incorporated into the DNA of mature leucocytes in tissue cultures.

By plotting the change in specific activity against time $\left(-\frac{dC}{dt}\right)$, an approximation of the life span of the white cells, based on the assumption that the release of labeled cells into the circulation is instantaneous, may be obtained. A correction to allow for the fact that the labeled cells are not discharged simultaneously may be calculated through the use of an equation to fit the early part of the curve, from the fourth to the eighth days. The equation $C(t) = Co(1 - e^{-\lambda t}) + 1900$, where $C(t)$ is the specific activity at time t, and Co and λ are constants whose values are 6,400 and 1.53, respectively, satisfies these conditions. The points obtained from this equation are shown on curve *A*, Fig. 1, as x's. Where $\phi(t)$ is the probability that a white cell will have a life span greater than t, it has been shown (*1*) that

$$Co\frac{d\phi}{dt} = \frac{1}{\lambda}\frac{d^2C}{dt^2} + \frac{dC}{dt}.$$

A plot of $-Co\frac{d\phi}{dt}$ against time gives curve *B*, Fig. 1, which yields an average life span of 12.8 days from the time of administration of the isotope and a value of 8.8 days for the time the labeled leucocytes are in the circulation in large numbers. The difference of 4 days may be interpreted as the time the bulk of the cells containing labeled DNA remained in the bone marrow. This is in accord with the observation that white cells continue to be released for 3–5 days following irradiation (*6*).

A life span of 12.8 days is considerably longer than the values that have appeared in recent literature (*6–8*). It should be borne in mind, however, that the shorter times were measurements, not of the life span of white cells, but of the time required for the replacement of cells that had been removed by cannulation or destroyed by irradiation. There is ample evidence (*9*) that discharge of stores of mature leucocytes from the bone marrow could satisfactorily explain the extremely short "life span" reported in these experiments.

The values reported here represent the average of all white blood cell types. If all these cell types persist in the circulation for days, it is impossible to explain the observed depression in lymphocyte and eosinophil levels after administration of ACTH and cortisone on the basis of inhibition of production of

these cells. Although it does not appear likely that the lymphocytes and eosinophils are completely different in their life span from the other leucocytes, final certainty on this point must await further experimentation.

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Significant Spatial Distribution Patterns of Minerals in the Coeur d'Alene District, Idaho

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In the Silver Belt of the Coeur d'Alene mining district, deeply buried high-grade oreshoots are found in carbonate-quartz veins. A plan map of the area has been published (*1*), and the geological problems involved in ore-search have been discussed (*2*).

A spatial distribution study has been made of the minerals of the area to determine which have distribution patterns indicative of the proximity of ore. Such minerals are called "indicators." The genesis of each mineral has been interpreted as an aid in the evaluation of the spatial distribution patterns.

Disseminated arsenopyrite forms envelopes around a number of the highest grade oreshoots. In horizontal or vertical cross sections, these envelopes have an average width of less than 15 ft; viewed normal to the plane of a vein, an envelope would appear as a halo of roughly 500-ft radius around an oreshoot. Similarly, late hydrothermal chlorite is considered to be an indicator; however, it is rather sporadically scattered in zones which tend to be umbrellalike in form above oreshoots, and which appear to extend as far as 3,000 ft above ore in some cases. Sericite and carbonates of prehydrothermal-vein origin have a negative significance as indicators; i.e., little ore has been found in areas where these minerals are concentrated. Also, since beds rich in detrital quartz are the best ore horizons, such quartz is considered to be an indicator of limited practicability.

In an attempt to clarify the origin of chlorite, six genetic types of chlorite are defined in the Coeur d'Alene District. These are chlorite in detrital biotite; diagenetic chlorite in certain strata; early, hydrothermal-vein chlorite; contact-metamorphic chlorite in and near the monzonitic intrusives; late, hydrothermal-vein chlorite; and chlorite resulting from the alteration of diabase and lamprophyre dikes.

The hydrothermal-vein history of the district is divided into three stages. In chronological order these are: the bleaching alteration stage, the carbonate-quartz stage, and the sulfide stage. The hydrothermal bleaching alteration of large areas of the country rock is largely the destruction of the rock pigments, and no strong sericitization appears to be involved, as previously believed. Regionally, the only exposed sedimentary rocks are those of the Algonkian Belt series; sericite is a major constituent of these rocks. If additional sericite is formed in the localized, bleached areas of the Algonkian rocks, it is a relatively negligible amount.

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DDT Resistance in Korean Body Lice[1]

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Contrary to expectation, routine application of 10% DDT powder to a large group of Korean military personnel during the winter and spring of 1951 resulted only in an increase of infestation with the body louse (*Pediculus humanus corporis* Deg.). The method used was essentially that employed by Soper *et al.* (*1*) in 1943, except that power dusters were used. The dust is applied without the removal of clothing. The DDT came from various sources, including a large stock of American manufacture that had been in storage for 5 or 6 years. Tests with mosquito larvae, however, demonstrated that it had retained its full insecticidal potency. The diluents most commonly used were talc and pyrophyllite.

The group of men treated increased rapidly in size during the first 3 months, then remained relatively stable. By the end of the second month it was possible to replace clothing worn by new arrivals with uninfested clothing. The number of layers of garments to be treated was thereby materially reduced. Living conditions were steadily improved so that bathing and clothes-washing facilities became readily available by the fourth month.

Despite these improvements, and the weekly application of DDT louse powder to all personnel, the percentage of infested persons increased steadily. During

[1] The opinions and assertions contained in this article are the private ones of the writers and are not to be construed as official or reflecting the views of the Navy Department or the Department of the Army.