mean monthly air temperatures for the first three months of 1942 were 46°, 49°, and 58° F., respectively.

Studies such as this, if carried on over an extended period of time to determine the degree of variation which undoubtedly exists in the length of the spawning periods, the periods of maximum decline in the ovary weight/body weight ratios, and the causes of such variation, would assist materially in providing a more scientific basis for the establishment of "closed seasons" if the evidence justified restrictive fishing measures.

Intercellular Surface Phagocytosis[1]

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Pathogenic microorganisms with protective capsules are ordinarily considered resistant to phagocytosis unless previously opsonized by specific antibody (*1*). Evidence has recently been presented that both encapsulated pneumococci (*3, 4*) and Friedlander's bacilli (*2*) are readily phagocyted in the absence of antibody by a mechanism referred to as surface phagocytosis. Direct visualization of this nonantibody form of phagocytosis in the lung has shown that the leucocytes phagocyte the encapsulated bacteria by trapping them against the surface of the tissue. Once taken into the cytoplasm of the phagocytic cells, the bacteria are rapidly killed (*2, 4*).

During experiments in which surface phagocytosis of Friedlander's bacilli in lung sections was being observed directly, phagocytosis was occasionally noted at some distance from the alveolar walls. The phagocytosis occurred whenever bacilli happened to get caught between the surfaces of two or more colliding leucocytes. On such occasions the surface of the adjacent leucocyte appeared to act in the same capacity as did the alveolar wall in the previously described form of surface phagocytosis. When the number of leucocytes in the phagocytic mixture was increased, the intercellular surface phagocytosis was noted frequently (see Fig. 1).

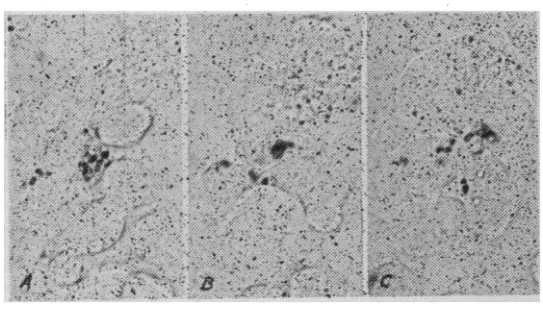

Fig. 1. Intercellular surface phagocytosis: (A) Leucocytes surrounding a group of encapsulated Friedlander's bacilli in a medium devoid of antibody (time, 2:10 P.M.); (B) Leucocytes have closed in on the bacilli so that they are trapped between the surfaces of the phagocytic cells (time, 2:23 P.M.); (C) The trapped bacilli have been phagocyted and can be seen in the cytoplasm of three of the leucocytes (time, 2:40 P.M.).

From these direct observations it appeared likely that intercellular surface phagocytosis would result whenever the concentration of leucocytes became such that the encapsulated bacteria could be readily trapped between two or more cells. To test this hypothesis, the following experiment was performed:

Friedlander's bacilli harvested from 2 ml. of a 4-hour broth culture (*2*) were suspended in gelatin-Locke's solution along with washed leucocytes obtained from the peritoneal cavities of 4 rats previously injected with starch aleuronat (*2*). The leucocyte-bacillus mixture was then concentrated by centrifugation (2,000 r.p.m. for 5 minutes), and as much supernatant fluid as possible was removed with a pipette and discarded. The concentrated mixture, which now contained a minimum of free fluid, was incubated for 30 minutes at 37° C., and smears of the mixture were stained with methylene blue. Examination of the smears revealed marked phagocytosis of the Friedlander's bacilli, whereas in control experiments, in which the leucocyte-bacillus mixture was not concentrated, little or no phagocytosis resulted.

Similar results were obtained with *Pneumococcus* Type I (A5 strain), *Pneumococcus* Type III (A66 strain)[2], and a mouse-virulent strain of *Staphylococcus aureus* (strain 235). Young cultures of each of these organisms resisted phagocytosis when incubated with leucocytes in dilute solution.

Intercellular surface phagocytosis of a number of pathogenic encapsulated organisms was thus brought about merely by diminishing the amount of fluid in the bacteria-leucocyte mixtures. Although the capsules protect the organisms against phagocytosis when the bacteria are floating freely in a fluid medium, they fail to protect them against surface phagocytosis when the latter occurs on a tissue surface or as a result of the intercellular mechanism just described. As previously stated, bacteria ingested by the nonantibody mechanism of surface phagocytosis are promptly killed by the phagocytic cells (*2, 4*).

The significance of intercellular surface phagocytosis in the mechanism of recovery in pneumococcal and Friedlander's bacillus pneumonias is indicated by the following considerations:

Although the earliest reaction of the lung to acute bacterial infection is the outpouring of edema fluid into the infected alveoli, this first stage is promptly followed by a rapid accumulation of leucocytes at the site of infection. The amount of edema fluid in the alveoli and bronchi diminishes, and the number of leucocytes present increases until the phagocytes are so numerous that they form the solid mass of cells characteristic of the advanced stages of pulmonary consolidation. Such a concentration of phagocytic cells eventually makes it virtually impossible for bacteria in the consolidated areas to escape surface phagocytosis. Those organisms that are not trapped against the tissue surfaces of the alveoli and bronchi are eventually phagocyted by being pinned between the surfaces of two or more of the crowded leucocytes. Thus, leucocytes in the lung may, through these two forms of surface phagocytosis, bring about destruction of even the most virulent encapsulated organisms, in the complete absence of immune bodies.

Conclusions: Encapsulated bacteria are phagocyted by leucocytes in the absence of opsonins, not only by being trapped against tissue surfaces but also by being caught between the surfaces of the phagocytic cells. Because large num-

[1] These studies were supported by the Commonwealth Fund.

[2] Kindly supplied by Colin M. MacLeod, of New York University.

bers of leucocytes accumulate in the lung during acute bacterial pneumonia, it is concluded that intercellular surface phagocytosis causes the destruction of many of the invading organisms, particularly during chemotherapy in the early stages of the disease when specific antibodies are not present to opsonize the bacteria.

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Use of 2,4-D Weed Killers on Woody Weeds in Cuba

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The problem of eradication of woody perennial weeds is much more serious in the tropics than in temperate zones. In Cuba the most serious weed is the legume *Dichrostachys nutans*, known as *Aroma marabu.* Since this plant regenerates vigorously from cut stumps, its eradication by continued cutting is almost impossible. In experiments carried out at the Atkins Garden and Research Laboratory of Harvard University, at Soledad, Cuba, several preparations and methods of spraying of 2,4-D have been tested on plots of large, well-established plants from cut stumps. Although delayed regeneration is still not entirely excluded, the following approximate figures for apparent kills in 4 weeks have been obtained:

TABLE 1

Treatment	Per cent apparently killed	Per cent probably alive
1. 2,4-D sodium salt, 0.2%	18	21
2. The same applied twice, 5 days apart	76	8
3. The same, 0.3%, applied once	62	8
4. The same, 0.3%, plus Carbowax 1500, 0.5%, applied once	87	1
5. 2,4-D ester, 0.3%, applied once	94	4

Since the plants die slowly, the percentage kill increases steadily with time, and a number of plants have to be classed as doubtful for a month or more after treatment. On balance, treatment No. 4 was considered the most effective, although No. 5 gave the most rapid defoliation. The volumes used were about 100 gallons/acre.

Plants in the shade were only slightly less affected than those in the sun. Spraying on the underside of the leaves did not increase the toxicity.

Another troublesome plant, *Comocladia dentata* (Guao), having a toxic action on the skin like that of poison ivy, was also studied. In this case the ester preparation ("Weedone") was definitely more effective than either the salt or the free acid, probably because of its better adherence to the vertical glossy leaves, from which aqueous sprays ran off rapidly. A concentration of 0.3 per cent gave about a 75 per cent kill as determined 6 weeks after spraying.

These experiments, which give promise of possible reclamation of land up to now considered virtually unavailable, are being continued.

Note on the Theory of Radiation-induced Lethals in *Drosophila*

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A rather complete theory of induced lethals in *Drosophila* which was proposed by Lea and Catcheside (*5*) has met with objections in private discussions. As this theory is now embodied in a comprehensive book (*4*) (reviewed in *Science*, April 25, p. 454), it may be advisable to publish some comments on the matter.

The frequency of x-ray–induced, sex-linked recessive lethals in *Drosophila* is experimentally known to be accurately proportional to the x-ray dose, at least up to about 5,000 r. The presence among these lethals of a large number which are located at points affected by chromosomal rearrangements creates a well-known difficulty for the interpretation of the dependence of frequency on dose (*2*). If it is simply assumed that this fraction of lethals is a by-product of the rearrangements, the total frequency of recessive lethals should increase faster than in proportion to the dose. Lea and Catcheside made the alternate assumption that the recessive lethals and the rearrangements result independently from a single type of primary effect. This primary lesion, the frequency of which is assumed to be proportional to the dose, may or may not, according to chance, lead to a recessive lethal and/or to a rearrangement. Such a common ancestry accounts for the observed coincidence of recessive lethals with rearrangements. It must also cause, however, a parallel frequent coincidence between the recessive lethals and those rearrangements (e.g. dicentrics) which are unviable ("dominant lethals") and thus escape detection. The latter rearrangements thus make an inroad in the observable recessive lethals, which should become increasingly important with increasing dosage; consequently, the frequency of recessives should increase slower than in proportion to the dose.

This nonlinear effect, embodied in Lea and Catcheside's detailed theory, leads to a very significant discrepancy between this theory and the experimental data. The authors merely discount the discrepancy because they reckon on having overestimated it by an undetermined amount (*5*). It is proposed here to give a minimum estimate of the importance of this discrepancy, using general arguments.

Lea and Catcheside classify the recessive lethals as class *A* (associated with no rearrangement), class *B* (associated with a minute rearrangement), and class *C* (associated with a viable gross rearrangement). This latter kind of rearrangement will be called *VGR*. We shall also consider class *D* lethals, *i.e.* those associated with an unviable gross rearrangement (*LGR*).

The various symbols will be used to indicate the corresponding frequencies, but it should be noted that the frequencies of *C*'s and *VGR*'s *among viable sperms* are, respec-