B. enteritides and of *B. prodigiosus* were injected intradermally in the right and left ears respectively of a series of mice. The injections were made on 2 successive days in some experiments, while in others 5 to 8 injections were given at intervals up to 2 weeks. From one to 3 weeks after the first injection extracts of the pooled right nodes and left nodes were titrated for agglutinins for both of the antigens as was also the pooled serum. Cross agglutination tests were negative. From 4 to 16 times as much agglutinin for *B. enteritides* was found in the node extract from the side injected with that bacterin, as in the extract from the other side; and from 3 to 10 times as much as in the serum. The findings for prodigiosus agglutinin were similar, that is to say, the titre for prodigiosus agglutinin was found far higher in the extract from the nodes of the side injected with *B. prodigiosus,* much less was present in the serum, and still less, or as much as in the serum in the node extract from the opposite side. The lymph glands on the 2 sides were invariably inflamed to about the same extent. The experiment was repeated 4 times with similar results.

The longer the time interval between injection of antigen and examination for antibody, up to 3 weeks, the greater was the concentration of the latter in both serum and nodes. In the experiments in which a single antigen was used, the concentration of antibody in serum and nodes became about equal within three weeks or less but the titre of the serum never exceeded that of the glands.

The findings will be fully reported elsewhere.

7307 P

Agglutinin Formation Within the Lymph Nodes of Resistant and Susceptible Mice.

STEPHEN S. HUDACK AND PHILIP D. McMASTER.

From the Rockefeller Institute for Medical Research, New York City.

An accompanying paper has reported the formation of agglutinins within lymph nodes following intradermal injection of killed cultures of bacteria in the ears of mice. Earlier work from this laboratory has shown such intradermal injections to be largely intralymphatic. After injections of killed bacteria into the ear of an animal a cervical lymph node draining the lymphatic capillaries enlarges and a week later agglutinins in high concentration can be found within it. At

this time the blood shows a lower concentration of agglutinin and the lymph nodes elsewhere in the body yield no demonstrable antibody. It is evident that the procedure engenders, in the early days of the immunizing process, a high concentration of agglutinin in a localized region.

We have experimented to find whether the lymph nodes of strains of generally susceptible and resistant mice developed by Webster[1, 2, 3, 4] differ in the ability to form agglutinins to certain bacteria to which they are susceptible and resistant respectively, and to other organisms against which their relative resistance is unknown.

Approximately 200 mice of each of the strains mentioned were obtained through the courtesy of Doctor Webster. A similar number of stock white mice of the ordinary laboratory mixed strain were employed for comparison. In each experiment groups of 12-20 mice from each strain were injected in the skin of both ears with killed cultures of various agglutinin forming bacteria, *B. enteritides, B. paratyphosus-B* or *B. prodigiosus,* using but one antigen in each experiment. The cervical lymph nodes draining the ears were removed after varying intervals up to 10 days following the last injection, and the animals were bled for serum. The individual nodes from the mice of each group were pooled and extracted and the titre of the extract was compared with that of the serum, and with that of the node extracts and sera of the other groups.

The concentration of specific agglutinin in the lymph node extracts of the generally susceptible strain of animals was found to be definitely greater than that of the resistant ones. So, too, with the serum titre. The control group yielded findings which were intermediate or else like those in the generally resistant strain of animals.

The mice of the strain manifesting a general lack of resistance seemed capable of forming more specific agglutinin than those of the resistant strain. For example, in a typical experiment the susceptible, resistant and mixed strains of mice received 5 intradermal injections of 0.02 cc. of a suspension of killed *B. enteritides* during a period of 15 days and 21 days after the first injection agglutination tests of the cervical lymph node extracts and sera were done, as described in the preceding paper.

The lymph node extracts of the susceptible, resistant and mixed strain mice showed positive agglutination at dilutions of 2048, 512,

1 Webster, L. T., *Harvey Lectures,* 1932, **27**, 154.
2 Webster, L. T., *J. Exp. Med.,* 1930, **52**, 931.
3 Webster, L. T., *ibid.,* 1933, **57**, 793.
4 Webster, L. T., *ibid.,* 1933, **57**, 819.

and 1024, respectively, and the sera at dilutions of 1024, 512, and 512, respectively. The findings will be published *in extenso* elsewhere.

7308 P

Experimental Hemosiderosis.*

VALY MENKIN.

From the Department of Pathology, Harvard Medical School.

Many pathologists have assumed that hemosiderin is a product in the partial degradation of hemoglobin. It is recognized mostly as phagocyted material of a yellowish-brown color within mononuclear phagocytes, liver cells, or cells of the kidney tubules. It yields a Prussian blue reaction when tissues containing it are treated with acidified potassium ferrocyanide. The substance is therefore iron-containing in distinction to hematoidin. Brown[1] pointed out a number of years ago that hemosiderin-like products can be obtained experimentally by the injection of hematin derivatives. These substances he termed *hemosideroid,* for he found that they were soluble in hydrogen peroxide or in potassium hydrate. True hemosiderin was found to be insoluble when treated with these agents. Sprunt[2] and more recently Whipple[3] have pointed out the possibility that hemosiderin may be the result of a change in the fundamental pigment metabolism of the organism rather than a product in the partial degradation of hemoglobin.

For the past few years the writer[4, 5, 6, 7] has demonstrated, in studies on inflammation and tuberculosis, that following intravenous injections of ferric chloride, the iron salt accumulates at the site of an acutely inflamed area; and in tuberculous rabbits, within the tubercles themselves, when the injections are given repeatedly. Concomitantly with this accumulation the life span of experimental rab-

* This investigation was aided by grants from the William W. Wellington Memorial Research Fund and by the DeLamar Mobile Research Fund.

1 Brown, W. H., *J. Exp. Med.*, 1911, **14**, 612.

2 Sprunt, T. P., *Arch. Int. Med.*, 1911, **8**, 75.

3 Whipple, G. H., and Bradford, W. L., *Am. J. Dis. Child.*, 1932, **44**, 336.

4 Menkin, V., *J. Exp. Med.*, 1930, **51**, 879

5 Menkin, V., and Menkin, M. F., *J. Exp. Med.*, 1931, **53**, 919.

6 Menkin, V., *J. Exp. Med.*, 1932, **55**, 101.

7 Menkin, V., *Am. J. Med. Sci.*, 1933, **185**, 40.