to keep this disease in check is to destroy all the currant and gooseberry bushes within a radius of many miles of any white pine—a drastic and difficult procedure, and only partially possible."

We are glad to inform readers of SCIENCE that the blister-rust-control situation is much more encouraging than Dr. Gager's comment would indicate. A number of years ago it was found that the sporidia produced on the leaves of currant and gooseberry plants (*Ribes*) are very delicate and normally do not cause commercial damage to pine under average forest conditions to a distance of more than 900 feet from the *Ribes* plant. Therefore, local control of the disease is accomplished by destroying all *Ribes* within 900 feet of white pines and keeping the *Ribes* suppressed on such areas. In addition, the European or cultivated black currant is eliminated throughout white pine regions as a general control measure, because it is more susceptible to the disease than other *Ribes* species and is the chief agent in the long-distance spread and establishment of the rust in uninfected areas. This is not a native plant, has not escaped from cultivation to any extent, and accordingly can be located without serious difficulty and eradicated.

Control of blister rust is entirely practicable and economical. Individual pine stands can be protected by the removal of *Ribes* in their immediate vicinity, and this measure is successful in controlling the disease locally, regardless of the possible neglect of such work in surrounding stands or the presence of centers of infection nearby. The protection of valuable stands of five-leafed pine has gone forward at a rapid rate, particularly since 1933, when emergency relief labor and labor from CCC camps became available for this work. Excellent progress has been made in the protection of all three of the commercially valuable species of susceptible pines in the United States, the northern white pine, *Pinus strobus,* extending from the Lake States to Maine and southward along the Appalachian Range to northern Georgia; the western white pine, *P. monticola,* occurring chiefly in Idaho, eastern Washington and western Montana; and the sugar pine, *P. lambertiana,* extending along the Cascade and Sierra Nevada Mountains from southern Oregon to the vicinity of Sequoia National Park in California.

These three species occur in stands sufficiently dense to be of commercial value on about 15,000,000 acres of land. The 900-foot-border zones increase the acreage of the control areas from which the *Ribes* must be removed to something over 26,000,000 acres. By the end of 1936, an aggregate of over 18,000,000 acres of these control areas had been given one working and the *Ribes* removed. From 12,000 to 20,000 men, largely from relief rolls and from CCC camps, have been employed on the work during each of the last four summers. Prior to that time the work was conducted on a much smaller scale for several years in cooperation with the states, counties and private owners concerned.

Control of white pine blister rust is carried on under the leadership of the Bureau of Entomology and Plant Quarantine, which is cooperating with the Forest Service, the National Park Service, the Indian Service, the Works Progress Administration and other federal, state and private agencies owning or administering forest land. This project has been in progress long enough so that it is possible, by comparison of protected areas with unprotected areas, to be sure that the control methods used are proving entirely effective. There is some regeneration of *Ribes* after eradication, and portions of the control areas must be gone over again in from three to ten or more years, depending on local forest and *Ribes* conditions. Present indications are that pine stands can be protected, from the seedling stage to maturity, by working the areas for *Ribes* from one to three times. The destruction of *Ribes* promptly stops the increase of infection in pine stands, regardless of the amount of infection that may have occurred before the work was begun, although such trees as are already fatally infected do not recover. Not all infected trees die, branches with blister rust cankers occasionally being shaded out before the canker reaches the trunk, but the disease is fatal when, as is usually the case, either trunk cankers or very numerous branch infections are present.

The extent to which relief labor will continue to be available for conservation work of this kind and the possibility of continuing operations to cover remaining unprotected pine stands can not, of course, be determined at this time, but in any event the protection already given to millions of acres of valuable white pine helps to conserve a forest resource of great value to the public for timber, recreational and water-supply purposes that would otherwise have suffered the fate of the chestnut.

S. B. FRACKER

BUREAU OF ENTOMOLOGY AND
PLANT QUARANTINE
U. S. DEPARTMENT OF AGRICULTURE

DOES THE VIRUS OF INFLUENZA CAUSE NEUROLOGICAL MANIFESTATIONS[1]

THERE has been a great deal of discussion in regard to the possible relationship between influenza and encephalitis or encephalo-myelitis. In this paper for the sake of brevity, the term "encephalitis" will be used with the understanding that it may refer either to a pure encephalitis or to an encephalo-myelitis. In this brief report no attempt will be made to cover the details of the various arguments that have been advanced.

[1] Preliminary report.

It may be well to point out that in Jordan's[2] study of the epidemiology of influenza, the conclusion was reached that any connection between these two diseases was probably fortuitous.

It is well recognized that, except in times of epidemic, the diagnosis of influenza is often loosely made. Since the discovery of the virus of influenza, it has been possible to make the diagnosis with a high degree of accuracy by the study of the neutralizing power of the serum after convalescence.

During the past several months, a certain number of cases have come to our attention in which an acute respiratory infection diagnosed as grippe or influenza has been followed within a short time by the development of various neurological conditions. In sixteen such instances the serum of the patients has been obtained within one to three or four months after the upper respiratory infection. These serums have been tested for influenzal antibodies according to the procedure of Francis and Magill.[3] In fourteen instances no protective antibodies were demonstrated in the serums. In one instance, the serum virus mixtures indicated slight protection and in one instance, a partial protection. In no instance was there complete protection. It, therefore, seems reasonable to assume that the original diagnosis of influenza or grippe had been incorrect in at least fifteen of the cases.

Further indications that the virus of influenza is not an etiological factor in cases of encephalitis or encephalo-myelitis is afforded by two facts: first, in a personal communication Francis has stated that in experimental animals the virus of influenza does not invade the central nervous system; second, Dr. L. S. Snegireff, of the New Jersey State Health Department, has given me permission to quote him to the effect that in investigating several New Jersey state institutions, where epidemics of influenza have occurred, there has been no instance in which encephalitis has developed during or following the outbreaks of influenza.

These data indicate that there is no etiological relationship between encephalitis and influenza. It is obvious that further work must be done to confirm or refute this opinion.

Two questions arise which await further investigation. When encephalitis or encephalo-myelitis follows an upper respiratory infection (diagnosed as grippe or influenza), are these early symptoms the first stage of the central nervous system infection or does an upper respiratory infection (not influenza) lower the barriers of resistance of the naso-pharyngeal mucosa to a virus either latent or acquired? This latter question presupposes that the virus or viruses of encephalitis may gain entrance through the naso-pharyngeal mucous membrane. On this point there is no definite information. However, it is quite generally believed that this is the portal of entry of the virus of poliomyelitis and of the St. Louis type of encephalitis.

We hope that this brief report will lead to further investigations by others, and we trust that we shall have the opportunity to carry on more work.

JOSEPHINE B. NEAL
HARRIET L. WILCOX

BUREAU OF LABORATORIES
NEW YORK CITY DEPARTMENT
OF HEALTH

[2] E. O. Jordan, "Epidemic Influenza," 1927; published by the American Medical Assn.

[3] Francis and Magill, *Jour. Exp. Med.*, 63: 5, 655–668, May 1, 1936.

THE OCCURRENCE OF A POSSIBLE MUTATION, CANCER TO NON-CANCER, IN THE HOUSE MOUSE

THE late Millard C. Marsh, in an attempt to demonstrate his theory that the irritation caused by intestinal nematodes (*Aspiculuris tetraptera*) was directly responsible for the appearance of mammary tumors in mice, developed a large colony of these animals in which the incidence of breast tumor was unusually high (80 to 100 per cent.). In support of his theory, he cited the behavior of these tumors when the experimental colony was freed from the parasites. In these mice he noticed that the tumor incidence was gradually becoming less and less.

Due to the fact that other workers, to whom he sent breeding stocks, of this strain, were unable to obtain the high tumor incidence recorded by Marsh an attempt has recently been made to determine the true incidence of cancer of the breast, in his stock.

In tabulating Marsh's data, it was noticed that this decline in cancer incidence among females of the worm-free colony was largely due to the inclusion, in the strain, of two families none of the members of which developed tumor. Tracing back the pedigrees of the first of these families, it was found that a mouse in his generation "Y," which itself had a mammary tumor and the female ancestors of which for 18 generations back had all been cancerous, gave rise to a line none of the members of which developed tumor. This line extended through twelve generations and contained 112 mice.

The female which gave rise to the second non-cancerous family was cancerous herself and had an ancestry of eight generations of tumor. All her descendants, which extended through eight generations and which numbered 28 females, were non-tumorous.

When these 140 animals are deducted from the data making up the incidence curve for the worm-free stock, the percentage of tumor remains remarkably constant in its range, for the different age periods (between 80 and 85 per cent.).

Unfortunately the stock, as it now exists in this