Chemical Society instructed the secretary to report at Cincinnati upon possible plans for providing retiring annuities for the staff of the society. This report was made and the council recommended action on the part of the directors. The directors have now voted that present employees of the society after twenty-five years of service, having hereafter reached the age of at least sixty years, shall on retirement be paid annually for life an amount equal to one per cent. of their average salary for the last five years of their service, multiplied by the number of years of service. It is further understood that this action shall not in any way affect the right of the society to discharge any employee. The directors further instructed the treasurer to set aside a reserve fund of $50,000 for employees' retirement, and recommended to future directors that the amount of the fund be adjusted as needed.

DISCUSSION

THE OCCURRENCE OF NATURAL AND ACQUIRED IMMUNITY TO INFECTIOUS MYXOMATOSIS OF RABBITS[1]

IN reporting investigations concerning the virus of infectious myxomatosis of rabbits, Moses (1911)[1] noted that the wild rabbit of Brazil was resistant to infection except in rare instances. In our work with the virus the common laboratory rabbit has proved susceptible in every case, over two hundred and fifty rabbits being used for various experiments during the last five years. Findlay (1929)[2] has reported the Belgian hare to be susceptible and we have found the Flemish Giant likewise susceptible. This would be expected since they are varieties of the common laboratory rabbit.

In an earlier paper (Hobbs, 1928),[3] it was suggested that the western jack rabbit and the varying hare or northern snowshoe rabbit might also be immune to this virus. Since then it has been possible to secure live specimens of *Lepus californicus* Gray, the black-tailed jack rabbit, *Lepus americanus* Erxleben, the varying hare, and *Sylvilagus transitionalis* Bangs, the common wild cottontail. Two animals of each species were obtained and all proved immune to skin inoculation with the infectious myxomatosis virus. As is true in certain of the other virus diseases, this natural immunity seems to be a tissue immunity since it has not been possible to demonstrate any virucidal property in serum from either the jack rabbit or the wild cottontail. Whether this immunity can be broken down by means of giving massive doses of virus or by using other routes of inoculation and whether virucidal antibodies can be produced in these wild hares and rabbits are among the several problems that suggest themselves.

In the large number of laboratory rabbits which have been used in our experimental work, only one individual has shown any signs of resistance to the virus and it was a member of a group that had received repeated injections of killed virus. It had a severe case but eventually recovered and has proved immune to subsequent inoculation. That its resistance was due to the previous injections of killed virus can not be claimed, of course, since Sanarelli (1898)[4] reported that two of his animals recovered spontaneously from the infection. Virucidal antibodies were demonstrable in its blood two months after its recovery and are still demonstrable, eighteen months after recovery by means of the following technique. A 5 per cent. suspension of virus was prepared by grinding freshly removed myxomatous tissue in normal saline in a mortar. The suspension was then centrifuged for fifteen minutes at low speed and 0.5 cc quantities of the supernatant added to 0.5 cc quantities of serum from the above rabbit, of normal rabbit serum and of normal saline. These mixtures were then incubated at 37° C., being shaken every thirty minutes. At the end of two hours, the mixtures were removed and inoculated into susceptible rabbits. The rabbits inoculated with the immune serum mixtures never showed any signs of myxoma at the sites of inoculation or elsewhere, while the control rabbits developed typical myxoma nodules at the sites of inoculation and died from the infection.

These experiments would suggest the existence of a genus immunity to infection as the laboratory rabbits all belong to the genus *Oryctolagus* (not *Sylvilagus* as stated in my earlier paper), while the wild hares are placed in the genus *Lepus* and the wild wood hares or cottontails of North and South America are placed in the genus *Sylvilagus* according to the classification of M. W. Lyon given in the Encyclopaedia Britannica, thirteenth edition, 1926. In this the wild rabbit of Brazil is called *Sylvilagus* (not *Lepus*)

[1] A. Moses, "O virus do mixoma dos coelhos," Mem. Inst. Oswaldo Cruz, 3, 46, 1911.

[2] G. M. Findlay, "Notes on Infectious Myxomatosis of Rabbits," *Brit. Jour. Exp. Path.*, 10, 214, 1929.

[3] J. R. Hobbs, "Studies on the Nature of Infectious Myxoma of Rabbits," *Amer. Jour. Hyg.*, 8, 800, 1928.

[4] G. Sanarelli, "Das myxomatogene Virus," *Centr. Bakt.*, Abt. 1, 30, 865, 1898.

braziliensis and is said to be very closely related to the wild cottontail of North America. Whether European species of *Lepus* and wild species of *Oryctolagus* would show a natural immunity to this virus is an interesting conjecture.

Our experiments are being continued and will be more fully reported at a later date.

JOSEPH R. HOBBS
DEPARTMENT OF COMPARATIVE PATHOLOGY,
HARVARD UNIVERSITY MEDICAL SCHOOL

NOTE ON THE CORN COMPONENT OF A RACHITOGENIC DIET

IN working with white rats and rickets the authors have found irregularity in the development of rickets on the Steenbock Diet 2965. The trouble has been traced apparently to the yellow corn component of the diet, and a satisfactory remedy was derived from the following experiment.

Thirty pounds of whole grain market yellow corn were equally divided. One half was immediately placed in a loosely covered earthenware crock, and the other half ground finely before storage in a similar container. Each lot was held at the variable room temperature of a dry laboratory for one year. At the end of this period of time the whole grain corn was ground in the same machine previously used, and twin batches of the above diet were compounded from these two samples of ground corn. The difference, obviously, was that one batch of meal was freshly ground from old corn, while the other batch had been aged as meal. A third lot of diet mixture employed corn-meal bought at a grocery.

The three diet mixtures were fed to animals selected equally from three litters of rats, and on the twenty-fourth day all animals were diagnosed by X-ray and line test, with the following result.

No. of animals	Peculiarity of diet	Positive rickets	Ave. gain in weight
6	Freshly ground corn	none	27 grams
6	Aged ground corn	6	19 "
6	Market corn-meal	6	8 "

We do not find in the literature the caution that whole market corn may contain an amount of anti-rachitic factor sufficient to interfere with desired development of rickets in white rats, if the corn be used freshly ground, but only the notation that storage of ground corn is attended with loss of growth-promoting factor. Since it is desirable to retain the growth-promoting vitaminic power but is imperative to avoid excess of antirachitic factor, one seems to be between the horns of a dilemma in respect to the corn component of this diet. A reasonable solution seems to be to store the ground corn-meal for six months and use it up in the next few months, and this procedure, for want of one more exact, serves as a successful expedient for insuring the development of a definite rachitic condition in white rats on a definite time schedule, with a simultaneous reasonable rate of gain in weight.

ROBERT S. HARRIS
JOHN W. M. BUNKER
MASSACHUSETTS INSTITUTE OF TECHNOLOGY

WHEAT MOSAIC IN EGYPT

IN 1927–1929 the writer began investigations on a "new" wheat disease in Egypt for the Egyptian Ministry of Agriculture. It seems advisable to present a preliminary note at this time; later a complete paper will be published on various aspects of the problem. This disease was called to the writer's attention in December, 1927. The crop of 1928 was severely attacked. It was present to a somewhat less degree in the wheat crop of 1929 and has been reported to the writer as occurring in the crop of 1930.

In December, 1927, many completely yellow or chlorotic plants were sent to the mycology laboratory of the Giza agricultural farm. The same condition appeared on certain varieties of wheat in the varietal plots at Giza. The characteristic yellow color or chlorotic condition of the early stages of growth (when the plants were from one to two months of age) suggested the tentative names "yellows disease" or "wheat chlorosis," names by which it became known in Egypt. This condition was said never to have been seen on wheat in Egypt prior to 1927, and many explanations for its cause were forthcoming from staff members of the several divisions of the Ministry of Agriculture as well as from laymen.

Circumstantial evidence as well as established facts convince the writer that this malady had been present in Egypt for several years prior to the outbreak of 1928 but had escaped notice.

Diseased plants were scattered in a field and were not restricted to definite areas. There was no definite relation with respect to soil conditions. Normal plants were found next to a group of diseased individuals. The Hindi variety, one of the standard wheats of Egypt, was especially susceptible. Many fields were observed that had from 40 to 60 per cent. of the plants badly affected. The yield in these fields was reduced 20 to 40 per cent. Badly diseased plants were completely yellow or chlorotic in the early stages of growth and as the tillers began to form. The root