capes into the maternal circulation, and in susceptible individuals the production of anti-Rh isoantibodies results. These antibodies filter through the placenta into the fetus and destroy its blood cells and in that way give rise to the disease.

At first sight, one might conclude that since only Rh-positive babies are affected, this mechanism operates in a selective manner so as to eliminate the Rh-positive type. As a matter of fact, all the affected infants are heterozygous, genotype *Rhrh*, so that equal numbers of *Rh* and *rh* genes are lost every generation. The effect of the loss of these genes over a period of many generations on the distribution of the Rh factor is readily computed as follows:

Let us assume that we are dealing with a population of constant size containing x *Rh* genes and y *rh* genes. The initial distribution of the genes would then be as follows:

$$Rh_0 = \frac{x}{x+y} \qquad rh_0 = \frac{y}{x+y}$$

If the number of fetuses and newborn that die from erythroblastosis during one generation is c, then the distribution of the genes during the second generation would be:

$$Rh_1 = \frac{x-c}{x+y-2c} \qquad rh_1 = \frac{y-c}{x+y-2c}$$

Accordingly, if at the onset the number of *Rh* genes is equal to the number of *rh* genes, this process would have no effect on the relative distributions of the genes. If the incidence of the two genes is unequal, however, the less frequent gene would be affected to a greater extent than the more common gene, so that eventually, other things being equal, over a period of thousands of generations, the incidence of the former would be substantially reduced and it might even be practically eliminated.

These results offer further evidence against the mutation theory as an explanation of the present distribution of the Rh factor in white individuals. Even assuming a rate of mutation from *Rh* to *rh* (or *vice versa*) higher than any so far recorded for *Drosophila* and man, this selective action of isoimmunization against the less frequent gene would effectively prevent a population originally completely Rh-positive from attaining as high an incidence of the Rh-negative type as 15 per cent. On the other hand, if one assumes the existence of populations in the past (and possibly still surviving at the present time) consisting almost exclusively of Rh-negative individuals, then from crosses with other populations consisting largely of Rh-positive persons (like the American Indians) a hybrid population could result with a serological composition resembling that of the white individuals of New York City.

In conclusion, it should be mentioned that, as Haldane[10] and Wyman and Boyd[11] have pointed out, if we go back to Paleolithic times when man was presumably a rare animal, chance probably played a large part in modifying gene frequencies. In large populations, however, chance has only a negligible effect, so that at least during post-glacial times racial mixture must have been the most important factor influencing the frequencies of the genes *Rh* and *rh*.

ALEXANDER S. WIENER

BROOKLYN, N. Y.

[10] J. B. S. Haldane, *Human Biology*, 12: 457, 1940.

[11] L. C. Wyman and W. C. Boyd, *Am. Anthrop.*, 37: 181, 1935.

VITAMIN A AND THE THYROID[1]

THERE is a theory that an antagonism exists between vitamin A and the thyroid. The evidence for this has been collected by Smith and Perman,[2] who have published evidence showing that in short experiments there is some counteraction of thyroxin by carotene. More recently Belasco and Murlin[3] in a somewhat similar experiment showed that vitamin A lowered the metabolic rate of hyperthyroid rats. No very logical reasons have been offered for such antagonism, if it exists, however, and close examination of results so far published reveal many discrepancies. It was felt, therefore, that further study was justified, and the following experiments were performed.

The sleeping metabolic rate was determined for 8 rats, 5 male and 3 female. After the range had been established, each rat was given 200,000 U. S. P. XI units of vitamin A[4] per kilogram daily by stomach tube. The concentrate was in oil solution, and contained negligible amounts of vitamin D. The volume of oil fed was between 0.2 cc and 0.6 cc daily. After 50 days of administration of the vitamin at this level (in one case after 34 days) desiccated thyroid powder, U. S. P. XI, was given in addition to the vitamin. The thyroid was given in amounts ranging from 0.25 to 0.35 gm per kilogram daily as a water suspension by stomach tube. The metabolic rate was determined at weekly intervals throughout. In no case did the vitamin A alone cause any significant alteration in the level of metabolic rate. When thyroid was fed in addition to the vitamin, the mean increase in metabolic rate was 25.5 per cent.

A second group of 4 rats, 3 females and 1 male, was given the same dose of thyroid powder after the range of metabolic rate had been determined. This dose caused a mean elevation of 58 per cent. in the metabolic rate. After this effect had been established, vitamin A was administered in addition to the thyroid,

[1] Part of the expenses of this investigation were borne by a grant from the Nutrition Research Laboratories.

[2] D. C. Smith and J. M. Perman, *Endocrinology*, 27: 110, 1940.

[3] I. J. Belasco and J. R. Murlin, *Jour. Nutr.*, 20: 577, 1940.

[4] Supplied by Atlantic Coast Fisheries.

and at the same level of dosage. Over periods of time ranging from 30 to 45 days from the beginning of vitamin administration there was no significant change in the metabolic rate, although there was a tendency to a lower level.

A third series of 6 rats, 4 males and 2 females, was thyroidectomized under ether anesthesia, and after recovery the level of metabolic rate was established. Vitamin A was fed to these animals at the same level as in the previous experiments. There was no significant change in metabolic rates over periods of time ranging from 45 to 60 days from the time vitamin A was started, although again there was some tendency to a lower level.

From these results it appears that the effects of vitamin A on the metabolic rate of rats, even in massive doses, are questionable.

R. F. SHEETS, JR.
H. C. STRUCK
UNIVERSITY OF ILLINOIS COLLEGE OF MEDICINE, CHICAGO

SCIENTIFIC APPARATUS AND LABORATORY METHODS

CONTROL OF BLUE MOLD OF TOBACCO BY A NEW SPRAY

BLUE mold or downy mildew of tobacco (caused by the fungus *Peronospora tabacina*) is one of the most difficult of all fungous diseases to control with fungicidal spray materials. During the five years that mildew has been prevalent in Connecticut the writer has tested many spray materials. None of them have been satisfactory; some failed to stop the disease, some caused plant injury, some were too complicated of preparation and the farmers would not use them.

On the other hand, fumigation of the seed beds with benzol or paradichlorobenzene has given excellent control, if properly manipulated in seed beds which are tightly constructed so that too much of the gas will not leak out during the night. But improper use of either chemical involves certain risks of plant injury. Both are expensive if continued through several weeks.

There exists, therefore, a definite need for a simple, safe, inexpensive but effective spray or dust for controlling mildew in the beds. In quest of such a material the writer has tried a long list of chemicals but only within the last six months has he found one which seems to fill all these requirements. This material is ferric dimethyl dithiocarbamate (under the trade name of "Fermate"). It was first suggested to the writer as a possible mildew remedy by Mr. Harry F. Dietz, of the Grasselli Chemical Department of E. I. du Pont de Nemours and Company, and we are indebted to him for a supply of the chemical and much helpful information on its use.

The first experiments were conducted in the greenhouse during the past winter. All experimental plots were artificially inoculated with spores and, as a result, 100 per cent. of infection on untreated crocks was the rule. Usually 100 per cent. of the unsprayed plants die from the severity of the attack and, therefore, any fungicide which will preserve the treated plants under these conditions must have real merit.

During the winter four crops of plants—eight or ten 10-inch crocks of 200 to 300 plants each—were grown to size suitable for setting in the field and were either kept sprayed during this time with "Fermate" or left unsprayed as checks. The detail of these and later experiments will be published elsewhere.

All unsprayed plants became infected and most of them died. The most successful dosage of "Fermate" was 1½ to 2 grams in a liter of water with the addition of an equal amount of lime. When the plants were sprayed twice a week this treatment gave 95 to 100 per cent. of disease-free plants and they remained healthy until grown to transplanting size. At times there was a small amount of spray injury evidenced by chlorotic areas on the leaves, but this never caused serious detriment to growth and was lacking entirely in most of the trials.

In April of this year the experiments were repeated in the seed beds. The results fully substantiate those in the greenhouse in giving excellent control of mildew.

The results of these experiments, conducted during one winter in the greenhouse and one spring in the seed beds, appear quite encouraging and lead us to believe that we have at last found a successful, simple inexpensive prevention for tobacco mildew. Before drawing final conclusions, however, this treatment should be repeated over several seasons and by practical growers in different sections.

P. J. ANDERSON
CONNECTICUT AGRICULTURAL EXPERIMENT STATION

AN ELECTRIC RECORDING MARKING COUNTER FOR THE CONSECUTIVE COUNTING OF SMALL OBJECTS

A NEW application of an electric counter has been devised for counting the projected cross sections of wool fibers. When magnified 500 times, the average diameter of wool fibers is seldom more than one inch and often is less than 0.5 inch. This counter shows possibilities of further applications in science and in-