

Alfalfa in Chick Rations

possible control of growth depressing effect of alfalfa indicated by addition of cholesterol to diet of chicks

D. W. Peterson

The following progress report is condensed from the full technical report to be published in a forthcoming issue of Journal of Biological Chemistry.

Evidence obtained in feeding tests indicates that the depressing effect of dehydrated alfalfa on the growth of chicks can be counteracted largely by the inclusion of cholesterol—a type of crystalline alcohol—in alfalfa-containing rations.

It appears that the growth depressing effect is due to a naturally occurring substance which in some unknown manner produces a toxic effect on chicks.

The deleterious effect of alfalfa meal is present in the fresh alfalfa—as determined by drying fresh frozen alfalfa and feeding it—but it is not due to the fiber present, nor to the mineral constituents.

Most of the inhibitory effect on growth can be removed by exhaustive extraction of the alfalfa meal with hot water. The residue so produced has little growth-depressing action, whereas the water extract produces marked inhibition of growth.

Precipitates—or fractions—from the extractions were prepared by chemical process for inclusion in growth tests.

Growth Tests

Biological tests employed to detect the presence or absence of the growth-inhibiting factor were growth tests on seven- to 14-day-old Single Comb White Leghorn

chicks that had been maintained previously on a standard stock diet.

The chicks were selected for uniformity of weight and rate of growth and were maintained on the experimental diets for periods varying from two to three weeks.

A basal ration was formulated from practical feedstuffs. Alfalfa meal at a 20% level was included in the ration and fed to one group of chicks. Other groups received the basal ration to which were added precipitates obtained by chemical fractionation of a water extract of alfalfa meal. All were compared with a group fed the basal ration containing no alfalfa meal and with a standard stock diet. One fraction, insoluble in an alcohol acetone solution, had marked growth depressing properties.

All inhibitory fractions foamed strongly. This characteristic suggested the presence of saponins—compounds present in many plants which have foam producing properties. Some of these compounds are toxic and this suggested an explanation of the growth inhibiting effect of alfalfa meal on chicks.

Saponins react with sterols—crystalline alcohol compounds—to form addition compounds which have lost a property peculiar to saponins—the ability to hemolyze—dissolve—red blood cells.

To test the possibility that a sterol might counteract the growth depressing agent, cholesterol, a sterol from animal sources, was added to the alfalfa containing diets.

The results of the tests indicated that cholesterol counteracted the growth depressing action of alfalfa meal and of the various inhibitory fractions obtained from alfalfa meal.

It appears probable that the growth depression is due to saponins, since all fractions producing growth depression had a hemolytic action which was prevented by cholesterol. It cannot be stated without qualification, however, that saponins are responsible for the growth inhibiting properties of alfalfa meal until they are isolated in pure form from alfalfa and shown by feeding tests to cause growth depression.

While it is not feasible to feed cholesterol in a practical ration, the type of information obtained in these studies leads to a probability that a practical means can be found for counteracting the growth inhibitor of alfalfa meal.

D. W. Peterson is Research Assistant, Division of Poultry Husbandry, in the Experiment Station, Berkeley.

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year-old Hardy pears bearing only leaf buds, brought into the warm greenhouse in November and used while still in the resting condition the following spring.

On April 1st three lots of ten trees were transplanted into peat moss and placed in cold storage at 37° F. Lot 1 was held continuously in the cold room for 71 days. Lot 2 was daily removed from the cold room and placed in a dimly lighted room at a mean temperature of 73° F for about six hours, then returned to the cold room. Lot 3 was likewise daily removed from the cold room and placed outdoors where the mean temperature was 64° F and it received direct daylight for about six hours before return to the cold room.

Of the total outdoor exposure 76% was

recorded by the United States Weather Bureau as sunshine, 24% as cloudy.

On April 20th a fourth lot of ten trees was placed in the cold room and held there continuously for 52 days.

On June 11th all trees were planted in the greenhouse. On June 20th a few buds were growing on lots 1 and 4 but none on lots 2 and 3. Visible growth of a few buds on the latter two lots did not occur until June 27th. In the succeeding days the number of growing buds increased on all lots of trees, but many more grew on lots 1 and 4 than on lots 2 and 3. The maximum numbers of growing buds were reached on July 3d on lots 1 and 4, but not until July 15th on lots 2 and 3. The final number of buds which grew were as follows: lot 1, 84%; lot 2, 29%; lot 3, 50%; lot 4, 72%; of the total buds present.

It is clear that the treatments applied to

lots 2 and 3 both retarded the rest breaking process in comparison with those applied to lots 1 and 4. The retardation expressed itself in two ways: in the smaller numbers of buds growing and in the slowness with which they started growth. In the smaller illustration are shown two trees from each lot, the one with least and the other with most buds growing. The trees in lots 2 and 3 resemble those in the larger photograph which had received too short cold treatment—less than 50 days.

Lots 2 and 3 received a cumulative exposure at 37° F equivalent to 52 days, the same as lot 4 received without interruption.

It is apparent that a few hours daily warm treatment partly offset the effect of 18 hours daily cold treatment.

Although lots 2 and 3 were both

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