Estrogens for Fattening Poultry

Treatment of chickens on increase but is not recommended for turkeys

F. W. Lorenz

The use of estrogens for fattening chickens is now an established procedure in some segments of the meat-bird industry.

In southern California alone an estimated 1½ million birds were treated with estrogens during the first half of 1949, and interest in the procedure appears to be still increasing.

Chickens

Estrogen treatment has been called chemical caaponization but this term is not entirely accurate. Although the testes are inhibited, they are not destroyed; also the caaponizing action is only a part of the effects of the treatment.

To be sure, estrogen-treated cockerals resemble capons in some respects during the course of the treatment. The birds' testes become very small and their combs shrink and become pale; fighting and crowing tend to cease. But if the treatment is withdrawn the testes will grow again and the birds become normal cockerals.

Estrogen-treated birds might better be said to be feminized. The hormone of the testes is replaced by a substance that has the same action as the ovarian hormone; treated birds act like hens in all respects except for actual egg production. Their pubic bones spread, their vents enlarge, and they may squat when approached. New feathers grown during the treatment have the feminine shape and color.

The treatment has two specific feminizing effects of considerable practical importance: it causes a thickening of the skin and it speeds the rate of fat formation. Both improve the appearance of the dressed bird. The thickened skin has a smooth, lucid, silky texture that produces a very attractive carcase, and extra fat deposited under the skin shows the bird to be well finished.

Extra fat is also deposited elsewhere throughout the body—in all the fat depots and in the muscle tissue. The meat is made more tender and juicy and the flesh color of cocks is lightened, probably due in part to the infiltration of fat and in part to suppression of the male hormone.

Two other effects may be disconcerting unless anticipated. The shanks are usually somewhat paler than normal. This, together with the pale shrunken comb, may look like evidence of unthriftness if one does not understand that both are normal consequences of the treatment. A tendency to bone fragility is slightly more serious, and some increase in care during killing and dressing may be necessary with treated birds.

Estrogen treatment has little or no effect on the rate of growth. Treated growing birds will deposit additional fat without disturbing their normal rate of gain, but also without appreciable extra gain. In older birds that have stopped growing, however, appreciable additional weight may be obtained during the treatment. Thus, in broiler and fryer production, estrogens are useful only for improving meat quality and grade; treated birds may be sold at the same ages and weights as untreated birds. On the other hand, with old cocks culled from the breeding flock, the weight gained during treatment plus the improved quality may justify feeding the birds the additional time necessary to complete the treatment.

Treatment

The essential requirement of estrogen treatment is simply to supply an adequate dosage of an active material continuously for a long enough time to produce the desired effect.

At present the only estrogen available to the poultry industry is diethylstilbestrol, prepared as pellets for subcutaneous implantation. Correct use of properly prepared pellets readily meets the requirements for successful treatment. The requirements for pellets are simple, and several brands of properly prepared pellets are now on the market. They are satisfactory if they contain 15 mg.—milligrams—of diethylstilbestrol and, at most, a small percentage of inert material. If more inert material is present the response is apt to be reduced or the pellet may be absorbed too rapidly to be effective. Prescription pellets, for example, are ineffective when implanted in poultry. These pellets are intended to be taken by mouth; they contain a large amount of rapidly soluble inert material and a dispersing agent, and they disappear completely within a few days.

The pellets are slipped under the skin with an implanting tool made of a large hypodermic needle with a plunger to eject the pellet. The pellet should be held in place with a finger on the bird's skin while the needle is withdrawn, to prevent loss of the pellet through the hole made by the needle. Should an occasional pellet be lost the bird’s comb will remain large, and the loss may thus be detected within about 10 days. Such birds may be retreated, but retreated birds should be separated from the rest of the flock and marketed later.

A single pellet is adequate for broilers and fryers. Adults should receive two or even three depending on the size of the bird. Where more than one pellet is used the bird should receive his entire dose at the same time, but each pellet should be inserted separately so that each remains a short distance from the others. Permitting the pellets to clump together may reduce their absorption rate and thus their effectiveness.

The first sign of the effects of treatment may appear within a week. The birds' combs are usually noticeably pale by this time. Fattening requires longer treatment, however; at least six weeks should be allowed for a full development of the response.

The age of the birds when treated is immaterial and should be governed by the grower’s marketing program.

Pellet implantation should be made six to eight weeks before the birds are to be killed. If a longer interval elapses between treatment and killing the effects of the treatment will regress. If the interval is shorter a full effect will not be obtained. A fair degree of fattening may be obtained as early as four weeks after implantation, but so short an interval is not recommended. Commercial pellets now on the market have been designed for a full response and will not be completely absorbed in less than the full treatment period.

The last consideration—above—also governs the choice of the place of implantation, high in the neck. Actually, pellets produce their effect wherever they are inserted, but should a small fragment of pellet remain unabsorbed, a pellet in the neck can be removed readily without damage to the appearance of the dressed carcase. Also, should such a remnant be missed, it will normally be removed when

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26th. Seven of the eight ewes came into estrus within 48 hours after the second injection, but none became pregnant though mated to a fertile ram. This dosage is within the super-ovulation range resulting in many ruptured or unruptured follicles which may be of doubtful value.

In contrast, a group of Hampshire ewes were treated with a 600 I.U. dose in 1939. Following a single injection, several ewes were sacrificed later and as many as nine freshly ruptured follicles per ewe were found. Some had only ripe unruptured follicles. Others of the same group were mated after a second injection and eight of the 16 became pregnant. Three of the eight had triplets, three had twins, and two had singles.

On the average, only 32% of ewes receiving two injections came into estrus after the second injection, but some experiments were more successful. For example, in 1944, 26 Southdowns were given 400 I.U. PMS on July 27th and 320 I.U. on August 13th; 19 of the 26 came into estrus within 10 days after the second injection, were bred, and 13 became pregnant.

Early experiments with estrogen and PMS were not successful in producing a normal estrus. When the dose of estrogen was too low—100 to 400 Rat Units—heat was not regularly induced, whereas if the estrogen level was too high, ovulation was inhibited.

In June, 1942, six Hampshire ewes received 100 R.U. of estrogen daily for 15 days and then PMS six days after the last injection of estrogen. Four other ewes received identical treatment except that the dose of estrogen was 400 R.U. daily. Six of the ten ewes came in estrus during the estrogen treatment, but only one after the injection of PMS when ovulation is expected. Five of these were force bred 72 hours after the injection of PMS, but none became pregnant. The five remaining ewes were sacrificed, but only one had ovulated.

Progesterone—the corpus luteum hormone—has been shown to inhibit estrus in the rat when large amounts were given.

The San Diego County Livestock Development of this material for commercial use has not yet been completed, whereas if the present diethylstilbestrol pellets are the only form of estrogen treatment available to the poultry industry.

The question of applying estrogen treatment to turkeys presents certain complexities.

The major interest has been in attempts to finish turkeys for market earlier than would otherwise be possible, but the results of trials have not been encouraging.

In spite of the suppression of the testes, the birds' aggressiveness is not quieted and sexual activity even tends to be increased. Some torn backs may result.

Turkeys respond with less dramatic fat deposition than is usually obtained with chickens, and the response is not improved by increasing the treatment period. In fact, lengthy trials usually resulted in birds of poorer quality than the controls. In a report of a recent trial maximum improvement was obtained only after two weeks of treatment. Some improvement in total weight was also observed. The advantage was lost in birds held beyond that time.

These results have not been entirely confirmed here, however, and at present estrogen treatment is not recommended for turkeys.

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the head is discarded prior to cooking the bird.

A word needs to be added about the possibility of adding estrogens to the feed. This method would have the advantage over pellet implantation of not requiring individual handling of the birds. Diethylstilbestrol is relatively ineffective when fed but some similar compounds produce fair fattening when mixed in the mash. Of these, some are unsuitable for commercial use due to undesirable side effects, but one, dienestrol diacetate, appears to have considerable promise as an estrogen that can be fed. Development of this material for commercial use has not yet been completed, however, and for the present diethylstilbestrol pellets are the only form of estrogen treatment available to the poultry industry.

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3. Once a cow has had Streptococcus agalactiae, her udder has been damaged. Even though apparently clean after treatment, the disease frequently reappears.

4. Regular testing, particularly of the disease-free cows, consistently milking infected cows last, and treating only dry or acutely infected cows is essential.

The San Diego County Livestock Department, cooperating with the Agricultural Extension Service analyzed monthly production and mastitis testing data on a 300-cow dairy.

In this herd, tests were made monthly for butterfat production and for mastitis. The production of all mastitis-free cows was averaged and compared to the average of the infected animals. Over 5,000 production records were included in the study.

All cows were handled and fed the same. The disease-free cows averaged 4.75 pounds butterfat per month more than the infected animals. This might be considered an average herd, starting with about 30% infection. Comparatively few of the cows had acute mastitis—most of the infected ones were hidden carriers.

On the last test the dairy owner had less than 5% of his cows shedding Streptococcus agalactiae.

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