AVIAN HYPOPHYSEAL STIMULATION AND SPERMATOGENESIS*

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The advent of a satisfactory method for semen collection (Burrows and Quinn 1937) has enabled artificial insemination to become of increasing economic importance to the poultry industry. It is now widely employed for improving fertility in turkey flocks and, to a lesser extent, in chicken flocks.

The number of males retained for semen production purposes (i.e. the male to female ratio) generally varies with the different breeds of birds (Parker and Bernier 1950; Parker 1958). In flocks reproduced by artificial insemination, semen volume and concentration are two parameters of paramount importance in determining the number of males required. Increased semen volumes without concomitant decreases in semen concentrations or quality would, therefore, reduce the male to female ratio, leading to a real economic advantage.

The drug ethamoxytriphetol (MER-25) acts as an anti-oestrogenic agent when administered to rats (Lerner *et al.* 1966) but not when administered to chickens (Jonsson and Terenius 1965). Also, in the chick, MER-25 stimulates hypophyseal secretion of gonadotropic hormones (Taber, Gardner, and Wood 1965) as well as the secretion of gonadal hormones (Van Krey and Siegel 1968). Conceivably then, MER-25 might stimulate an increased semen production when administered to adult birds. The following experiments were designed to test this premise.

Methods and Materials

Experiment 1.—Twelve F_{10} generation males from the low-weight line (Siegel 1962) were used for this study. Commencing at 36 weeks of age, six males were fed MER-25 as part of the diet for a period of 16 weeks. The remaining six birds served as controls. The daily consumption of MER-25 was calculated to be equivalent to the parenteral administration of 1·5 mg per day.

Semen samples were collected twice weekly at biweekly intervals. Semen volumes were measured with a pipette and semen concentrations determined spectrophotometrically at 650 nm (Kosin and Wheeler 1956). The average of the weekly value for each male was used in the analysis of the data.

Experiment 2.—Procedures were similar to those of the initial experiment with the exception that the MER-25 dosage was increased threefold (to 32 p.p.m.). Testes were weighed following completion of the experiment and analysed for differences between the control and the MER-25 treatment.

Experiment 3.—Procedures were basically similar to those described for the initial experiments with the exception that data were obtained weekly and the MER-25 concentration in the feed was increased to 500 p.p.m. Low-weight line males (12 control and 12 experimental) were also used in this experiment. Following randomization into experimental and control groups, a pretreatment semen evaluation of the males was made to be sure there were no differences among groups. The duration of this experiment was 10 weeks as opposed to 16 weeks in the initial two experiments. Body and testes weights were obtained at the end of the experiment, and histological sections were made of the testes.

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Results and Discussion

The influence of the oral administration of MER-25 on spermatogenesis in intact males (i.e. the hypothalamic–hypophyseal axis intact) was inconclusive following the first experiment. Although mean semen volume, concentration, and number of spermatozoa per ejaculate for the treated males were less than those for the control males, differences between groups were not significant, indicating no response to MER-25 (Table 1). Since previous publications were not available for a guide line in selecting an effective gametogenetic dosage of MER-25, the concentration of the drug in the feed was increased threefold in experiment 2.

As in the initial experiment, the mean semen parameters for the males receiving MER-25 were below that of the controls (Table 1). The differences in volume and number of spermatozoa per ejaculate, however, were significant in this experiment, suggesting an inhibitory gametogenetic response to MER-25. Mean testes weights for the control and MER-25 groups of males were $14{\cdot}9 \pm 1{\cdot}7$ g and $18{\cdot}2 \pm 2{\cdot}0$ g, respectively. The difference of 3·3 g was not significant.

TABLE 1

MEANS AND STANDARD ERRORS OF SEMEN VOLUMES, CONCENTRATIONS, AND NUMBERS OF SPERMATOZOA PER EJACULATE

	Control	MER-25	Diff.
		Experiment 1	
Volume (ml)	0·35±0·02	0·33±0·02	0·02
10^{-9} × number per millilitre	4·32±0·20	3·75±0·25	0·57
10^{-9} × number per ejaculate	1·50±0·10	1·28±0·11	0·22
		Experiment 2	
Volume (ml)	0·34±0·01	0·29±0·01	0·05*
10^{-9} × number per millilitre	5·05±0·31	4·72±0·35	0·33
10^{-9} × number per ejaculate	1·68±0·08	1·36±0·07	0·32*
		Experiment 3	
Volume (ml)	0·54±0·05	0·30±0·04	0·24**
10^{-9} × number per millilitre	5·17±0·28	5·86±0·28	−0·69
10^{-9} × number per ejaculate	2·77±0·31	1·76±0·23	1·01*

* $P \leqslant 0{\cdot}05$. ** $P \leqslant 0{\cdot}01$.

In experiment 3, as in experiment 2, semen volumes and number of spermatozoa per ejaculate were significantly lower for the males receiving MER-25 than for the control males (Table 1). Mean semen concentration was greater in the treated than in the control males, but the difference was not significant (Table 1). Linear regressions of treatment means on weeks were calculated to measure time trends for semen volume, concentration, and number of spermatozoa per ejaculate. These were as follows:

	Control	MER-25
Volume (ml)	0·005±0·006	−0·016±0·003*
10^{-9} × number per millilitre	−0·04 ±0·02	0·06 ±0·07
10^{-9} × number per ejaculate	−0·08 ±0·05	0·02 ±0·03

* $P \leqslant 0{\cdot}05$.

There was no change in semen volume for the controls, whereas there was a significant decline over time for those males fed the drug. Semen concentration and number per ejaculate did not change over time for either the treated or control males.

Mean body weights were 2905 g for the control and 2947 g for the treated birds. Mean testes weights of the control and treated birds were 17·98 and 21·49 g respectively. Differences between means were not significant in either case. Subjective estimations (coded slides) of the degree of spermatogenesis and spermiation (Lake 1956; Roosen-Runge 1962; Lacy and Lofts 1965) revealed no consistent differences between the control and MER-25 males. Hyperplasia of the interstitium was not evident in the sections.

Direct testicular stimulation with exogenously administered gonadotropins or gonadal hormones, or both, to increase spermatogenesis has, in general, been unsuccessful. Presumably the lack of success resulted from hormonal imbalances. To alleviate some of the difficulties associated with exogenously administered hormones, we attempted an indirect stimulation of spermatogenesis by forcing an increased endogenous gonadotropin and gonadal hormone production. We were, however, unsuccessful, which indicates that additional refinements are necessary. Apparently the temporal and quantitative intricacies of the hypophyseal gonadotropins and the gonadal hormones were disrupted in these as in the earlier experiments.

However, MER-25 may have potential as a means for stimulating an androgen-mediated anabolism. This is because MER-25 increases gonadal hormone secretion in immature males (Van Krey and Siegel 1968) and there was a suggestive, but non-significant, increased body weight in the adult males used in this experiment. This could be of some importance to an agricultural industry such as broiler production where an accelerated rate of growth is desirable.

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