TABLE 2. Concentrations ($\bar{x}$ ± SE) of hormones and peak characteristics

Hormone	Treatment	Concentration			Peak	
		Overall	Baseline	Amplitude	No./12 h	Length
		(ng/mL)			(no.)	(min)
Testosterone	Continuous[1]	1.62 ± .72	.83 ± .40	2.88 ± 1.85	6.0 ± 1.4	80 ± 28
	Intermittent[2]	1.93 ± .56	1.01 ± .30	2.77 ± 1.70	6.2 ± 1.0	78 ± 22
	$\bar{x}$	1.78 ± .58	.92 ± .29	2.76 ± 1.70	6.1 ± 1.2	79 ± 24
Luteinizing hormone	Continuous	3.52 ± .87	3.16 ± .88	2.22 ± .29	11.7 ± 4.5**	17 ± 5
	Intermittent	5.59 ± 2.38	5.26 ± 2.36	3.37 ± 1.25	6.8 ± 2.1	15 ± 4
	$\bar{x}$	4.56 ± 2.09	4.10 ± 2.11	2.80 ± .92	. . .	16 ± 3

[1]Continuous photoschedule of 14 h light (L):10 h dark (D) at an intensity of 10 lx; n = 3.

[2]Intermittent photoschedule of 1L:2D, 8× at an intensity of 10 lx; n = 3.

**$P < .01$ between Continuous and Intermittent photoschedules within hormone.

LH in sexually mature male turkeys. On the other hand, sampling every 10 min is adequate to determine the primary rhythm of secretion of T.

It was concluded that no difference in overall concentration of LH and only minor differences in the pattern of LH secretion occurred between Continuous and Intermittent lighting treatments in sexually mature male turkeys. The Continuous treatment was associated with an increased frequency of LH pulses. For T, no difference between lighting treatments for overall concentration or pattern of secretion were detected.

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Analysis of Subfertility Associated with Homozygosity of the Rose Comb Allele in the Male Domestic Fowl[1]

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ABSTRACT Sperm metabolism and duration of fertility were compared between roosters homozygous (*RR*) and heterozygous (*Rr*) for the rose comb allele. In the absence or presence of Ca^{+2}, a motility agonist, the metabolic rate of spermatozoa from *RR* males was 76 to 77% of that of spermatozoa from *Rr* males. In the presence of caffeine, the metabolic rate of spermatozoa from *RR* males was increased significantly ($P < .05$) to 86% of that of spermatozoa from *Rr* males. Although intramagnal insemination of ejaculated spermatozoa reduced the disparity in duration of fertility between *RR* and *Rr* males, the duration of fertility of *RR* males was still less than that of *Rr* males. No difference in duration of fertility was observed following intramagnal insemination of testicular spermatozoa. Subfertility associated with *RR* males seems to stem from an aberrant activation or regulation of spermatozoal metabolism and not a reduction in absolute metabolic capacity per se.

(*Key words*: chicken, rose comb allele, spermatozoa, subfertility)

1994 Poultry Science 73:871–878

INTRODUCTION

Since the 1950s (Cochez, 1951), roosters homozygous for the rose comb allele (*RR*) have been known to be subfertile. Although some authors, notably Petitjean and Cochez (1966) as well as Petitjean and Servouse (1981), have implicated poor spermatozoal motility as a contributing factor, the mechanism responsible for spermatozoal dysfunction is unknown. However, heritable subfertility of *RR* males cannot be attributed to spermatozoal degeneration as reported for subfertile Delaware roosters (Froman and Bernier, 1987; Kirby *et al.*, 1989).

Buckland and Hawes (1968) investigated variables typically used to assess male reproductive potential but could not define a difference between *RR* and *Rr* males that could account for subfertility in *RR* males. Buckland *et al.* (1969) reported a positive correlation between fumarase activity and fowl spermatozoal fertilizing ability, reporting that spermatozoa from *RR* males were characterized with reduced fumarase activity. However, Petitjean and Servouse (1981) observed no difference in fumarase activity when spermatozoa from *RR* and *rr* males were compared. Likewise, although Crawford and Smith (1964d) observed no difference in metabolic rate among spermatozoa from *RR*, *Rr*, and *rr* males, as evidenced by methylene blue reduction time, Kirby and Froman (1991a) reported that the metabolic capacity of *RR* males was only 63% of that of *rr* males as evidenced by the reduction of 2-(*p*-iodophenyl)-3-(*p*-nitrophenyl)-5-phenyl tetrazolium chloride (INT) to formazan. Therefore, studies of reproductive attributes, apart from fertility, have been inconclusive or contradictory.

Received for publication September 15, 1993.
Accepted for publication January 28, 1994.
[1]Supported by the Oregon Agricultural Experiment Station and Purina Mills Graduate Research and Chester M. Wilcox Memorial Fellowships. Oregon Agricultural Experiment Station Technical Paper Number 9503.
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The ability of the domestic fowl to lay multiple fertilized eggs following a single intravaginal insemination depends upon a tier of prerequisites. First, spermatozoa must be sequestered within the uterovaginal glands (for a review, see Bakst, 1987). Second, spermatozoa must reenter the lumen of the oviduct (for a review, see Zavaleta and Ogasawara, 1987). Third, spermatozoa must ascend the oviduct, presumably by antiperistalsis of oviduct smooth muscle (Solomon, 1983), to reach the infundibulum, where fertilization occurs (Olsen and Neher, 1948). Fourth, spermatozoa undergo an acrosome reaction as a consequence of contact with the inner perivitelline layer of the oocyte (Bakst and Howarth, 1977; Okamura and Nishiyama, 1978). This tier is apropos to the analysis to subfertility stemming from spermatozoal dysfunction within the oviduct.

The rate at which fowl spermatozoa reduce INT is not only an index of metabolic activity but is highly correlated with motility as well (Chaudhuri *et al.*, 1988). Consequently, the technique described by Chaudhuri and Wishart (1988) is parsimonious. Duration of fertility, as estimated by iterative least squares (Kirby and Froman, 1990, 1991b) has been an instrumental criterion in the analysis of heritable subfertility in Delaware roosters (Froman and Bernier, 1987; Kirby *et al.*, 1989, 1990) as well as subfertility induced by treatment of fowl spermatozoa with neuraminidase (Froman and Engel, 1989). Likewise, the fertilizing ability of testicular spermatozoa following intramagnal insemination can be used to diagnose sperm dysfunction (Kirby *et al.*, 1990). Therefore, the objectives of the present work were to 1) compare the metabolic capacity of ejaculated spermatozoa from *RR* and *Rr* males; 2) compare duration of fertility following intravaginal insemination of ejaculated spermatozoa from *RR* and *Rr* males; 3) to make a similar comparison following intramagnal insemination; and 4) evaluate the duration of fertility following intramagnal insemination of pooled testicular spermatozoa from the *RR* and *Rr* males used in the previous experiments.

MATERIALS AND METHODS

Animals

Experimental males were produced as follows: a Wyandotte rooster, homozygous (*RR*) for the rose comb allele, was bred to 10 Single Comb White Leghorn (SCWL) hens. In order to maximize fertility, hens were inseminated on a daily basis for 1 wk. Prior to each intravaginal insemination, semen was diluted 1:2 (vol/vol) with Beltsville Poultry Semen Extender (BPSE; a gift from Tom Sexton, USDA, Beltsville, MD 20705). Eggs collected throughout the week were incubated and chicks hatched.

At sexual maturity, a representative heterozygous (*Rr*) F_1 male was bred to 20 female half-sibs. Eggs were collected for 1 wk following a single intravaginal insemination. The single-combed (*rr*) F_2 chicks were culled at hatch. At sexual maturity, each rose comb F_2 male was bred to 10 SCWL (*rr*) hens in order to establish homo- or heterozygosity for the rose comb allele. Ten F_2 males were chosen randomly from each genotypic group.

Experiment 1

Semen was collected from each of 10 *RR* and 10 *Rr* males. Spermatozoal concentrations were determined fluorometrically according to Bilgili and Renden (1984). Spermatozoal metabolic capacity was estimated with a modification of the technique of Chaudhuri and Wishart (1988). Each ejaculate was diluted to 1×10^9 spermatozoa/mL in 20 m*M* HEPES (N-[2-hydroxy-ethyl]-piperazine-N'-[2-ethanesulphonic acid]), pH 7.4, containing 150 m*M* NaCl. Each of three replicate .2-mL volumes of spermatozoal suspension was mixed with 1.0 mL of one of three incubation media previously warmed to 30 C. The control medium was HEPES buffer containing 160 μM INT, 7.7 μM phenazine methosulphate, 7.8 m*M* glucose, and 2 m*M* KCN. The experimental media were identical to the control medium except that they contained either 8 m*M* $CaCl_2$ or 8 m*M* caffeine.

Following admixture of spermatozoal suspensions with incubation media, samples were incubated for 16 min at 30 C. Reduction of INT to formazan was terminated by admixture of .2 mL .1 *M* HCl containing 5% (vol/vol) Triton X-100 with

each sample. Spermatozoa were precipitated by centrifugation at 15,600 × *g* for 3 min, and the absorbance of the supernatant was measured at 520 nm. The concentration of formazan was calculated using a molar extinction coefficient of 15.5×10^3 M^{-1} cm^{-1}. Rates of formazan production, expressed as picomoles of formazan per one million spermatozoa, were analyzed with a two-way analysis of variance.

Experiment 2

Six roosters were selected randomly from each genotypic group. Each male was ejaculated, spermatozoal concentration determined as above, and each ejaculate diluted to 2.0×10^9 spermatozoa/mL with BPSE. Each spermatozoal suspension was used to inseminate an average of 22 SCWL hens. Each hen was inseminated intravaginally with 1×10^8 spermatozoa in a volume of 50 μL.

Egg collection began on the 2nd d after insemination and continued for 21 d. Eggs were set weekly. Fertility was assessed by breaking eggs open after 4 d of incubation and examining contents for embryonic development. Percentages of fertilized eggs were calculated per hen for the entire 21-d egg collection period. Each percentage was then transformed to a logit and analyzed using the General Linear Models procedure[5] according to Kirby and Froman (1990, 1991a). Duration of fertility was analyzed according to Kirby and Froman (1990). In brief, data from the 21-d egg collection intervals were plotted as a function of time. The parameters of $y(x) = [\gamma]/[1 + e^{\beta(\tau - X)}]$ were estimated by iterative least squares (Kirby and Froman, 1990). Extra sums of squares *F* tests were completed to determine whether the estimates of τ, the time of half maximal fertility, were estimates of common parameters.

Experiment 3

Ejaculates from the roosters used in Experiment 2 were pooled according to genotype in each of two replicate fertility trials. Spermatozoal concentration was determined as above and semen was diluted to 5×10^8 spermatozoa/mL. Each suspension was used to inseminate an average of 28 SCWL hens. Each hen was inseminated intramagnally according to Engel *et al.* (1991) with an insemination dose of 5×10^7 spermatozoa. Egg collection, incubation, and data analyses were performed as above.

Experiment 4

Intramagnal insemination of testicular spermatozoa was performed as follows. Roosters used in the preceding fertility trials were killed by cervical dislocation, and testes were removed. Testicular exudates were procured according to Howarth (1983). Due to the limited number of viable spermatozoa harvested by this method, exudates were pooled according to genotype. Each sample was diluted 1:2 (vol/vol) with minimum essential medium as prepared by Howarth (1981). The spermatozoal concentration was determined with a hemacytometer. Each spermatozoal suspension was used to inseminate 30 SCWL hens. The insemination dose was 2×10^7 spermatozoa per hen. Egg collection, incubation, and iterative least squares analysis of the duration of fertility for each pool of testicular spermatozoa were completed as described above. Differences in the proportion of fertilized eggs were evaluated with the Mantel-Haentzel statistic (Mantel and Haentzel, 1959).

RESULTS

Experiment 1

Formazan production was affected by both genotype ($P < .0001$) and incubation medium ($P < .0001$). A genotype by incubation interaction (P .0001) was also observed. As evidenced by formazan production (Table 1), the metabolic rate of spermatozoa from *Rr* roosters was only 76 to 77% of that of spermatozoa from *Rr* males in either the absence or presence of Ca^{+2}. In contrast, the metabolic rate of spermatozoa from homozygotes was 86% of that of heterozygotes in the presence of caffeine. Whereas Ca^{+2} exerted a similar effect on spermatozoa from both genotypes, caffeine was

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TABLE 1. Sperm formazan production as affected by rooster genotype and incubation medium

Incubation medium[2]	Formazan production by rooster genotype[1]	
	Rr	*RR*
	— (pmol/10^6 spermatozoa) —	
Control	233 ± 2.0^{A,X}	178 ± 2.4^{B,X}
8 m*M* Ca^{+2}	253 ± 2.2^{A,Y}	194 ± 1.8^{B,Y}
8 m*M* caffeine	244 ± 2.2^{A,Z}	210 ± 2.1^{B,Z}

^{A,B}Means within a row with no common superscript differ significantly ($P < .0001$).

$^{X-Z}$Means within a column with no common superscript differ significantly ($P < .0001$).

[1]Each value represents a mean (n = 12) ± SEM.

[2]Each medium contained 20 m*M* N–(2-hydroxyethyl)-piperazine-N'-(2-ethanesulphonic acid), pH 7.4, 150 m*M* NaCl, 160 μM 2-(*p*-iodophenyl)-3-(*p*-nitrophenyl)-5-phenyl-tetrazolium chloride, 7.7 μM phenazine methosulphate, 7.8 m*M* glucose, and 2 m*M* KCN.

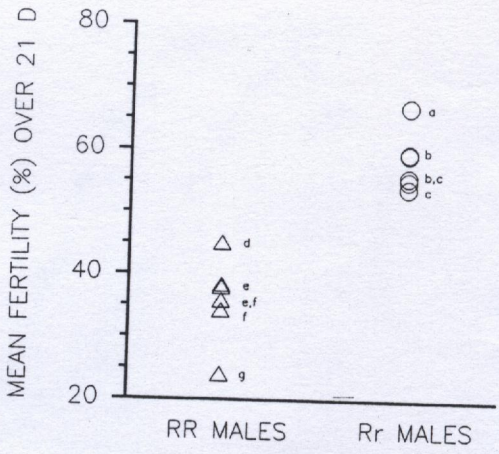

FIGURE 1. Mean fertility of eggs laid over a 21-d period by Single Comb White Leghorn hens (22 per rooster) following a single insemination with ejaculated spermatozoa from roosters heterozygous (*Rr*, ○) and homozygous (*RR*; △) for the rose comb allele. Individuals with significantly different ($P < .05$) mean fertility values are denoted with symbols (a to g). Notice, that although there are similar ranges in mean fertility values between the two groups, there is no statistically significant overlap between them.

more stimulatory to spermatozoa from homozygotes.

Experiment 2

Fertility was affected by both genotype (P .0001) and differences among males ($P <$ .0001). As shown in Figure 1, although a wide range of fertility was observed in which the most fertile homozygote was nearly comparable to the least fertile of the heterozygotes, any given male within a genotype was different ($P < .05$) from males of the other genotype.

As shown in Figure 2, the duration of fertility of homozygotes as defined by τ, an estimate of the time of half-maximal fertility, was 5 d less than that for heterozygotes ($P < .0001$). A second temporal distinction between homo- and heterozygotes was the lack of sustained fertility for even a few days following intravaginal insemination of ejaculated spermatozoa.

Experiment 3

Because no difference ($P > .05$) was observed among replicate fertility trials, data were pooled according to rooster genotype. Overall fertility of homozygotes was not different from that of heterozygotes following intramagnal insemination of ejaculated spermatozoa (Table 2). However, the duration of fertility of homozygotes was approximately 1 d less than that of heterozygotes ($P < .01$; Figure 3).

Experiment 4

Neither overall fertility (Table 2) nor duration of fertility (Figure 4) differed ($P > .05$) between the two pools of testicular spermatozoa when placed within the magnum.

DISCUSSION

The relationship between the rose comb gene and reproductive efficiency was reported independently by Cochez (1951) and Ponsignon (1951). The work of Cochez (1951) was more definitive in that he demonstrated that the reproductive anomaly observed with White Wyandottes was associated with homozygosity for the rose comb allele. Furthermore, Cochez (1951) demonstrated that hens with a genotype of *rr*, *Rr*, or *RR* were fertile when inseminated with spermatozoa from

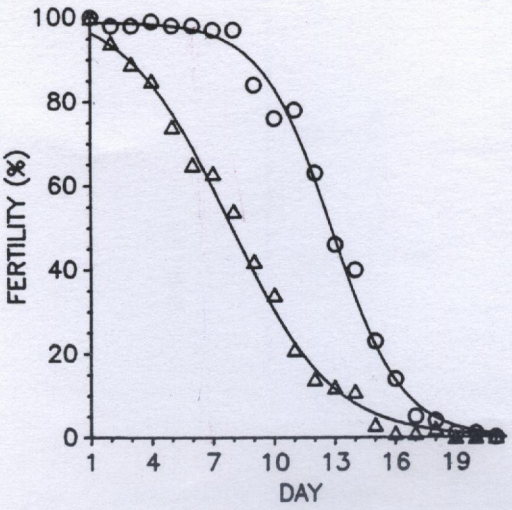

FIGURE 2. Duration of fertility following intravaginal insemination of Single Comb White Leghorn hens with ejaculated spermatozoa from roosters heterozygous (*Rr*, o) and homozygous (*RR*; △) for the rose comb allele. Each data point represents the mean fertility of 6 roosters mated to an average of 22 hens each, for a total of 132 hens per genotype. The insemination dose was 1×10^8 spermatozoa per hen. Solid lines represent the functions $y(x) = [99]/[1 + e^{-.5816(12.9-x)}]$ and $y(x) = [103]/[1 + e^{-.3956(7.8-x)}]$ for spermatozoa from *Rr* and *RR* males, respectively.

either an *rr* or *Rr* male. However, Cochez (1951) and Ponsignon (1951) both attributed poor reproductive efficiency to a lethal factor that reduced hatchability.

This hypothesis became untenable in the 1960s. Crawford and Merritt (1963) showed that reduced hatchability following insemination of hens with spermatozoa from *RR* males was due to reduced fertility. This observation was confirmed by Crawford and Smyth (1964a), who used light miscroscopy to evaluate blastodiscs from eggs categorized as nonfertilized by macroscopic evaluation. Crawford and Smyth (1964b) demonstrated that the fertilizing ability of *R*- and *r*-bearing spermatozoa from heterozygotes was equivalent, leading them to conclude that the subfertility of homozygotes was not due to a direct effect of the *R* gene on the spermatozoon.

Petitjean and Cochez (1966) measured the ejaculate volume and spermatozoal concentration and motility of *RR* and *Rr* males. Motility following short-term storage of spermatozoa at 3 C was the only variable that differed between genotypes. By mixing spermatozoa from one genotype with seminal plasma from the other, Petitjean and Cochez (1966) demonstrated that asthenospermia following incubation at 3 C was not attributable to a factor in seminal plasma. Like Petitjean and Cochez (1966), Buckland and Hawes (1968) studied variables traditionally used to assess reproductive efficiency. They reported no differences in sperm morphology, viability, or testicular attributes that could account for subfertility. Although Buckland *et al.* (1969) reported that spermatozoa from homozygotes were deficient in fumarase activity, Petitjean and Servouse (1981) were unable to identify any genotypic effect on spermatozoal fumarase activity.

Both Crawford (1965) and Etches *et al.* (1974) used spermatozoal competition to demonstrate that spermatozoa from *RR* and *Rr* males did not behave similarly following intravaginal insemination. However, Etches *et al.* (1974) demonstrated that the fertilizing ability of spermatozoa from *RR* males relative to *Rr* males could be improved by the use of intramagnal insemination. Because spermatozoal motility is not required for spermatozoal trans-

TABLE 2. Fertility over a 21-d interval following an intramagnal insemination with ejaculated[1] or testicular[2] spermatozoa from rose comb roosters

Rooster genotype[3]	Sperm type	Hens	Eggs	Fertilized eggs[4,5]
		—— (n) ——		(%)
RR	Ejaculated	54	971	66 ± 2.2
Rr	Ejaculated	57	1,020	73 ± 1.8
RR	Testicular	29	537	44 ± 3.1
Rr	Testicular	26	483	48 ± 2.8

[1]Each hen was inseminated with 5×10^7 spermatozoa.

[2]Each hen was inseminated with 2×10^7 spermatozoa.

[3]Pooled sperm suspensions were procured from six males per genotype.

[4]Each value represents a mean ± SEM.

[5]No differences ($P > .05$) between means within an experiment.

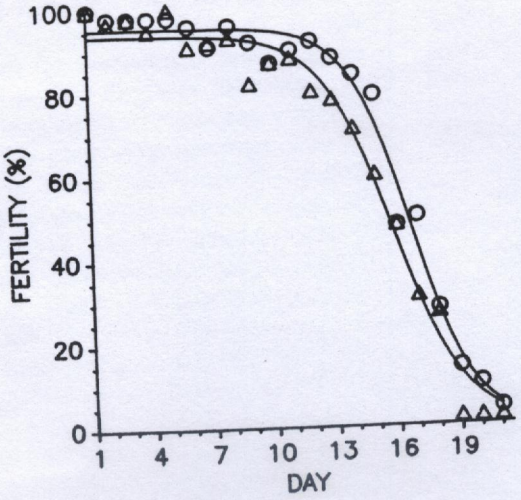

FIGURE 3. Duration of fertility following intramagnal insemination of Single Comb White Leghorn hens with ejaculated spermatozoa from roosters heterozygous (*Rr*, ○) and homozygous (*RR*; △) for the rose comb allele. The insemination dose was 5×10^7 spermatozoa per hen. In each of the replicate trials, semen was pooled from the ejaculates of six males per genotype. While the same males were used in both trials, each hen was used only once. Each data point represents the mean fertility of 54 or 57 hens per day for hens inseminated with spermatozoa from *RR* and *Rr* roosters, respectively. Solid lines represent the functions $y(x) = [96]/[1 + e^{-.6766(16.7-x)}]$ and $y(x) = [94]/[1 + e^{-.5910(15.8-x)}]$ for spermatozoa from *Rr* and *RR* males, respectively.

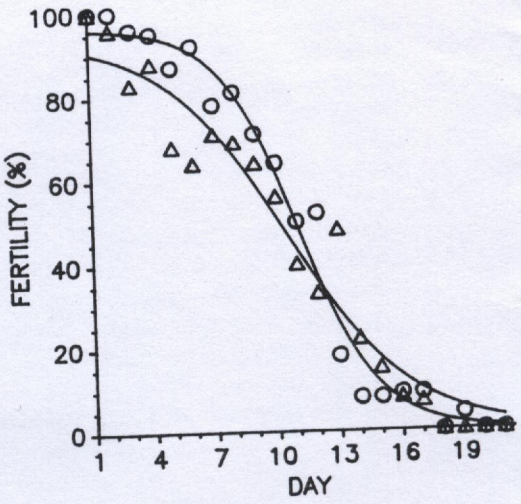

FIGURE 4. Duration of fertility following intramagnal insemination of Single Comb White Leghorn hens with pooled testicular spermatozoa from roosters heterozygous (*Rr*, ○) and homozygous (*RR*; △) for the rose comb allele. Each data point represents the mean fertility of 29 or 26 hens per day for hens inseminated with spermatozoa from *RR* or *Rr* roosters, respectively. The insemination dose was 2×10^7 spermatozoa per hen. Solid lines represent the functions $y(x) = [97]/[1 + e^{-.5256(11.1-x)}]$ and $y(x) = [94]/[1 + e^{-.3314(10.5-x)}]$ for spermatozoa from *Rr* and *RR* males, respectively.

port above the uterovaginal junction (Allen and Grigg, 1957), Etches *et al.* (1974) suspected that spermatozoal behavior within the vagina was related to aberrant spermatozoal metabolism.

Experiment 1 demonstrated (Table 1) that the metabolic capacity of spermatozoa from *RR* males was less than that of spermatozoa from *Rr* males even when spermatozoa were incubated in the presence of motility agonists (Wishart and Ashizawa, 1987). Due to the correlation between rate of formazan production and spermatozoal motility (r = .88; Chaudhuri *et al.*, 1988) as well as the fact that fowl spermatozoa are motile at 30 C in either the presence or absence of Ca^{+2} (Thomson and Wishart, 1988), differences in motility may be inferred.

Ionic calcium is a natural agonist of motility (Ashizawa and Wishart, 1987; Wishart and Ashizawa, 1987) and exists in deferent duct fluid at a concentration of 1 to 2.5 m*M* (Freeman, 1984). Ashizawa and Sano (1990) have proposed that a reduction in seminal temperature at ejaculation may be a critical factor in the initiation of fowl spermatozoal motility. Therefore, spermatozoa from *RR* males probably fail to become optimally motile either after release from temperature-dependent immobilization within the deferent duct or in response to extracellular Ca^{+2} within the vagina.

Experiment 2 demonstrated (Figure 1) that although the fertilizing ability of males within either genotype was variable, males tended to fall within distinct groups that were also differentiated by comb genotype. When data were pooled by genotype and fertility plotted as a function of time (Figure 2), *RR* males were characterized by an immediate decline in fertility. In view of the results of Experiment 1, this pattern of fertility is attributed to suboptimal spermatozoal filling of the uterovaginal glands.

This conclusion is supported by the results of Experiment 3 in that no difference ($P > .05$) in overall fertility was observed when spermatozoa were deposited above the uterovaginal junction (Table 2). However, spermatozoa from *Rr* males were characterized by a greater ($P < .05$) duration of fertility (Figure 3). Nonetheless, intramagnal insemination decreased the difference in duration of fertility between genotypes from 5.1 to .9 d. This contrasts with Crawford and Smyth (1964c), who compared duration of fertility between *RR* and *Rr* males following intravaginal and intrauterine insemination. In this work, duration of fertility was defined as the number of days between insemination and the day on which the last fertilized egg was laid. Differences in duration of fertility averaged 3.6 and 4.8 d following intravaginal and intrauterine insemination, respectively.

Neither overall fertility (Table 2) nor duration of fertility (Figure 4) differed ($P > .05$) between the pools of testicular spermatozoa following intramagnal insemination. Due to the limited numbers of viable spermatozoa procured by this procedure, and our desire to only use those males that were previously employed in fertility trials, we have a limited amount of information. Regardless, this experiment clearly demonstrates that there are no clearcut differences in fertilizing capacity between the two testicular spermatozoa pools from the males used in these experiments. Therefore, subfertility stemming from homozygosity for the rose comb allele may be associated with spermatozoal maturation; for ejaculated spermatozoa from *Rr* males had an advantage over their counterparts from *RR* males (Figure 3), whereas testicular spermatozoa did not (Figure 4). As shown by the effect of insemination site on the difference in duration of fertility between genotypes (Figures 2 and 3), the conferred advantage is more evident when sperm motility is of paramount importance. It is noteworthy that spermatozoa from *Rr* males were most responsive to Ca^{+2}, whereas spermatozoa from *RR* males were more responsive to caffeine (Table 1). Furthermore, caffeine attenuated the difference in sperm metabolic rate between genotypes (Table 1). Therefore, subfertility may stem from aberrant regulation of metabolism than metabolic rate per se.

A major assumption of research done to date with spermatozoa from *RR* males is that there is no direct effect of the rose comb allele on the spermatozoon (Crawford and Smyth, 1964b). However, this assumption may not be valid. Braun *et al.* (1989) used transgenic mice to demonstrate an exchange of mRNA via intercellular bridges between spermatids within a cohort. Thus, though a haploid spermatozoon may not possess a copy of any given allele, it may well contain the gene product. This phenomenon may answer the question of why *R*-bearing spermatozoa from *Rr* males function normally if the subfertility of *RR* males is due to a direct effect of the *R* allele on the spermatozoon. In conclusion, roosters homozygous for the rose comb allele continue to be a useful model for investigating the genetic control of sperm metabolism, particularly in the context of extra-gonadal sperm maturation.

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Skeletal Problems in Poultry: Estimated Annual Cost and Descriptions

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ABSTRACT This introductory, review presentation is a prologue to four full-length symposium presentations. Estimates of the annual cost of skeletal problems in poultry and definitions of noninfectious skeletal diseases in poultry are the focus of this article. Losses due to skeletal problems in poultry are caused by an increase in mortality and the number of cull birds, increased condemnations from septicemia-toxemia, and more downgrading from the trimming of breasts and legs. Reduced feed conversion and rate of gain also occur. Estimated annual losses in the United States due to skeletal problems are $80 to $120 million in broilers, and $32 to $40 million in turkeys. Eleven skeletal problems in poultry are described, and synonyms, symptoms, and possible causes of each are presented. These diseases include long bone distortion, tibial dyschondroplasia, rickets, spondylolisthesis, degeneration of the femoral head, spraddled legs, chondrodystrophy, osteomyelitis and synovitis, *Mycoplasma synoviae* infection, viral arthritis, and footpad dermatitis.
(*Key words*: broiler, turkey, skeletal diseases, annual cost, performance)

1994 Poultry Science 73:879–882

INTRODUCTION

Skeletal problems are recognized as one of the four major factors limiting the performance of meat-type birds (Day, 1990). Fast-growing broilers and turkeys that reach market weights at earlier ages are perceived to have more leg problems and suffer greater losses than slower growing birds. Survey data reported by Morris (1993) indicate that body weight of broilers is highly correlated to the severity of leg problems. Five primary causes of leg problems in broilers have been listed by Morris (1993). These include nutritional disorders, such as rickets; infectious diseases, such as viral arthritis; metabolic conditions, such as tibial dyschondroplasia; conformational problems, such as varus and valgus deformities; and toxins, such as mycotoxins.

ESTIMATED ANNUAL LOSSES ASSOCIATED WITH SKELETAL PROBLEMS

It is difficult to accurately determine the cost of skeletal problems in poultry due to the nature of these losses. Leg problems in broilers can increase mortality and the number of culls, increase condemnations from septicemia-toxemia, and increase downgrades from trimming breasts and legs (Morris, 1993). Reduced performance due to decreased feed consumption and disease can also occur.

A recent survey conducted and reported by Morris (1993) has placed the annual losses due to leg problems in broilers at $80 to $120 million. Survey forms were sent to all broiler companies in the United States. Sixty-five completed surveys were returned, representing 55% of the industry. Overall, 1.1% mortality was attributed to leg problems and an additional 2.1% of birds were reported to have been downgraded from leg disorders (Morris, 1993). Hot weather has a profound impact on leg problems, and these

Received for publication July 28, 1993.
Accepted for publication February 9, 1994.
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