Influence of Bovine Somatotropin on Subclinical and Clinical Mastitis

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Introduction

Recombinant bovine somatotropin (rBST) has been shown to enhance milk production in the lactating cow. (1-7). Producer and consumer acceptance and U.S. Food and Drug Administration approval for the field application of rBST is based upon the demonstration that such treatment does not produce deleterious effects upon the health of treated animals (1-8). Mastitis is one of the most costly diseaes affecting dairy cattle and the influence of rBST treatment on mastitis could be significant. The purpose of this investigation was to determine the influence of rBST treatment on subclinical and clinical mastitis.

Materials and Methods

Animals

Lactating Holstein cows of mixed parity were blocked according to parity and calving date and were randomly allocated to 4 levels of rBST treatment. Random allocation was performed separately for cows of parity 1 and foı parities 2-5. The period studied included October, 1986 until December, 1988. All treated cows were housed on 1 of 2 North Carolina Department of Agriculture research dairies located in the coastal plains of North Carolina. Each dairy consisted of >150 lactating cows. On dairy A, 60 cows were studied for a first lactation and 43 of the same cows were studied for a second lactation. On dairy B, 32 cows were studied for a single lactation, but all cows had been treated with the same dose of rBST during the previous lactation. Treated cows were housed with the entire lactating cow herd and were identified as to level of rBST treatment by unique animal identification and by color-coded neck chains. Cows were fed a corn silage-based total mixed ration with grain concentrate and whole cotton seeds and a minimum of 1.8 kg of hay/cow daily. Cows were milked twice daily in automated parlors and standard mastitis control procedures were practiced. The herds were on comprehensive dairy herd health programs, with bi-weekly visits by a veterinarian.

rBST treatment

Cows were allocated to one of 4 levels of rBST (0, 5.2, 10.3, and 16.5 mg of active available drug per day). The rBST (Agriculture Research Division, American Cyanamid, Princeton, NJ) was supplied in color-coded vials and refrigerated at 4°C until administered. Administration of rBST was via subcutaneous injection in the area of the tail fold, alternating sides daily. Administration was from early lactation (28-35 days in milk) until the earlier of either the end of lactation or 400 days postpartum.

Milk sampling

Quarter milk samples for microbiological analysis were collected aseptically in duplicate from each cow at trial entry and and at dry-off (9). Single quarter milk samples were aseptically collected at 60-day intervals during the trial. For milk samples from cows giving a change in infection status, quarter milk samples were collected in duplicate until duplicate samples were in agreement.

Clinical mastitis

Cows were observed by milkers at each milking for evidence of clinical mastitis, defined as the presence of grossly abnormal milk or mammary gland. Duplicate milk samples were aseptically collected from affected quarters of cows with clinical mastitis prior to treatment with a commercial lactating mastitis infusion product. All signs of clinical mastitis and treatments administered were recorded by milkers.

Microbiological analysis

Standard microbiological techniques were used in microbiological analysis of milk samples (10-11). For milk samples of cows with clinical mastitis, 0.01 and 0.05 or 0.10 ml (after January, 1988) of milk were plated on to the sur-

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faces of Columbia agar plates. Major pathogens were considered as *Staphylococcus aureus* (positive by tube coagulase), *Streptococcus agalactiae, Str. dysgalactiae, Str. uberis, Escherichia coli, Klebsiella* spp., *Enterobacter* spp., *Citrobacter* spp., *Actinomyces pyogenes,* and *Nocardia* spp. Minor pathogens included coagulase negative Staphylococci, *Streptococcus* spp, excluding those above, *Corynebacterium* spp., *Serratia marcescens, Prototheca* spp., and yeasts.

Indices of mastitis

Indices of mastitis monitored (9) included:

1. Point prevalence of intramammary infection (IMI): Prevalence of IMI by cow for major and minor pathogens by rBST treatment level was determined from milk samples collected at trial entry, 60-day intervals during the study and at dry-off.
2. Occurrence of new IMI: New IMI detected at samplings during the trial or as a result of clinical mastitis were evaluated by level of rBST treatment.
3. Duration of IMI: Duration of IMI (new IMI and IMI existing at trial entry) ws evaluated on the basis of the maximum duration of any infection within a cow during each lactation.
4. Incidence of clinical mastitis: Incidence of clinical mastitis by level of rBST treatment was evaluated for the entire period of study.

Statistical analysis

Infection prevalence data were analyzed by a repeated measures categorical analysis procedure which considered frequency of responses among rBST treatment levels over time (CATMOD Procedure, SAS, Inc., Cary, NC). Remaining data were analyzed using a modified Mantel-Haenzel test (FREQ Procedure, SAS, Inc., Cary, NC).

Results

Milk production response to rBST treatment was dose-dependent, similar to previous reports and is reported elsewhere (12). There were no significant differences in somatic cell concentrations in milk from cows among treatment groups (12).

Point prevalence of IMI

The prevalence of IMI by treatment level and collection period are given in Table 1. Prevalence of IMI by major and minor pathogens did not differ significantly among rBST dose levels ($P=0.06$ for major pathogens and $P=0.67$ for minor pathogens). Neither major ($P=0.11$) nor minor pathogen ($P=0.09$) cow prevalence infection rates varied significantly over time during the period of treatment.

Table 1 Point prevalence of IMI by major pathogens by treatment level and period of collection for Dairy A for first lactation*

Treatments—% cows infected (no. sampled in parentheses).

Collection	16.5 mg	10.3 mg	5.2 mg	0 mg
1st--trial entry	20%(15)	47%(15)	27%(15)	27%(15)
2nd	13%(15)	53%(15)	33%(15)	20%(15)
3rd	13%(15)	53%(15)	33%(15)	33%(15)
4th	8%(12)	62%(13)	31%(13)	36%(11)
7th--dry off	21%(14)	47%(15)	33%(12)	36%(14)

IMI by major pathogens for Dairy A for second lactation*

Collection	16.5 mg	10.3 mg	5.2 mg	0 mg
1st--trial entry	25%(8)	27%(11)	27%(11)	15%(13)
2nd	25%(8)	27%(11)	27%(11)	31%(13)
3rd	25%(8)	27%(11)	27%(11)	31%(13)
4th	25%(8)	40%(10)	27%(11)	31%(13)
7th--dry-off	25%(8)	60%(10)	22%(9)	23%(13)

IMI by major pathogens for Dairy B for first lactation

Collection	16.5 mg	10.3 mg	5.2 mg	0 mg
1st--trial entry	0%(7)	43%(7)	23%(13)	0%(5)
2nd	0%(7)	43%(7)	15%(13)	0%(5)
3rd	0%(7)	43%(7)	8%(13)	0%(5)
4th	0%(7)	43%(7)	8%(12)	0%(5)
5th	14%(7)	33%(6)	0%(10)	0%(3)
6th--dry-off	14%(7)	29%(7)	15%(13)	0%(5)

IMI by minor pathogens for Dairy A for first lactation*

Collection	16.5 mg	10.3 mg	5.2 mg	0 mg
1st--trial entry	20%(15)	40%(15)	20%(15)	13%(15)
2nd	13%(15)	47%(15)	20%(15)	27%(15)
3rd	13%(15)	40%(15)	20%(15)	27%(15)
4th	17%(15)	38%(13)	23%(13)	18%(11)
7th--dry-off	21%(14)	27%(15)	17%(12)	7%(14)

IMI by major pathogens for Dairy A for second lactation*

Collection	16.5 mg	10.3 mg	5.2 mg	0 mg
1st--trial entry	38%(8)	36%(11)	9%(11)	15%(13)
2nd	25%(8)	45%(11)	18%(11)	15%(13)
3rd	25%(8)	64%(11)	9%(11)	23%(13)
4th	25%(8)	60%(10)	9%(11)	15%(13)
7th--dry-off	25%(8)	70%(10)	33%(9)	23%(13)

IMI by minor pathogens for Dairy B for first lactation

Collection	16.5 mg	10.3 mg	5.2 mg	0 mg
1st--trial entry	14%(7)	29%(7)	15%(13)	20%(5)
2nd	29%(7)	29%(7)	23%(13)	20%(5)
3rd	14%(7)	43%(7)	23%(13)	40%(5)
4th	14%(7)	43%(7)	17%(12)	40%(5)
5th	14%(7)	33%(6)	20%(10)	67%(3)
6th--dry-off	29%(7)	29%(7)	23%(13)	60%(5)

*Due to small sample size, data from 5th and 6th collections on Dairy A are not presented.

Total new infections (TNI)

Total new IMI acquired during the trial are given in Table 2 and did not differ significantly among treatment groups ($P=0.48$). Total new IMI by major pathogens did not differ significantly ($P=0.20$) among treatment groups. Similarly, total new IMI by minor pathogens did not differ significantly ($P=0.21$) among treatment groups.

Table 2 Frequencies of all new IMI (major and minor pathogens) and clinical mastitis by treatment groups.

Frequency of new TNI

Treatment	0	1	2	3	Total*	Clinical#
16.5	21	9	0	0	30	4
10.3	21	8	2	2	33	3
5.2	30	6	3	0	39	4
0	21	8	3	1	33	4
TOTAL	93	31	8	3	135	15

* = Cow lactations studied. # = No. cows affected with clinical mastitis.

Clinical mastitis

Reported clinical cases of mastitis by treatment groups are given in Table 2. Because of the small number of cases, these data were not analyzed statistically. However, there was no indication of differences among treatment groups.

Duration of infections

Maximum duration of IMI within cows by treatment levels is given in Table 3. Duration of new or existing IMI gave no clearly significant differences among dose levels ($P = 0.08$ for duration of new IMI and $P = 0.06$ for existing IMI). A tendency was observed for a dose-related increase in maximal duration of existing IMI during the second lactation in Dairy A.

Table 3 Mean + S.E.M. maximal duration of IMI within cows for new and existing IMI

Maximal duration (days) of new IMI

Dairy/lactation	16.5 mg	10.3 mg	5.2 mg	0 mg
A, first	28.6±15.3	82.6±25.9	25.1±20.5	35.0±14.0
A, second	19.8±19.8	57.1±25.9	10.6± 5.6	37.±+20.4
B, first	42.1±37.7	43.7±31.0	33.8±23.4	70.8±40.7

Maximal duration (days) of existing IMI

Dairy/lactation	16.5 mg	10.3 mg	5.2 mg	0 mg
A, first	58.3±27.2	126.2±37.9	102.4±35.7	52.8±28.5
A, second	137.8±51.8	131.1±40.0	92.4±39.7	55.2±29.2
B, first	7.1± 7.1	125.6±53.2	44.8±25.1	54.2±54.2

Discussion

Prevalence of IMI did not vary among treatments over time during the period of study. This portion of the experiment was designed to determine if there was any association between rBST dose level and prevalence of IMI. If rBST had detrimental effects on treated cows with respect to susceptibility to IMI or elimination of existing IMI, one would expect to see differences in IMI prevalence rates. The present study did not indicate any consistent differences in this regard. No significant differences were noted in the maximal duration of new or existing IMI.

No significant differences were observed in the occurrence of new IMI or cases of clinical mastitis during our study. Because of the small number of clinical cases observed in the present trial studies, employing larger numbers of animals or analysis of data combined from several experiments may be indicated in order to address the issue of whether rBST treatment influences the incidence of clinical mastitis. However, the present study does not provide evidence for deleterious effects of rBST treatment on the incidence of new infections or of clinical mastitis.

There was no direct evidence provided in this study for any detrimental influence from rBST treatment on subclinical and clinical mastitis. The dairies studied were similar to commercial Southeastern dairies, indicating that these results should apply to actual field use of rBST.

Summary

The purpose was to investigate the influence of treatment of lactating cows with recombinant bovine somatotropin (rBST) on subclinical and clinical mastitis. Lactating Holstein cows of mixed parity were randomly allocated to 4 levels of rBST (0, 5.2, 10.3, and 16.5 mg/day) administered into the tail fold area from early lactation (28-35 days in milk) to the end of lactation. On dairy A, 60 cows were studied for the first lactation and 43 of the same cows were studied for 2 complete lactations. On dairy B, 32 cows were studied for a single lactation, but all cows had been treated with the same dose of rBST during the previous lactation. Duplicate quarter milk samples were aseptically collected from all cows at trial entry, at 60-day intervals during the trial and at trial end or dry-off. All cases of clinical mastitis were recorded and milk samples were collected from affected quarters for microbiological analysis. Milk samples were analyzed by standard microbiological procedures. Preliminary analysis of data indicated that treatment with rBST did not adversely affect incidence, prevalence and duration of subclinical intramammary infections or incidence of clinical mastitis.

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For Your Library

Food Safety and Quality Assurance: Foods of Animal Origin

William T. Hubbert, Harry V. Hagstad, and Elizabeth Spangler
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Written as a textbook for students in veterinary medicine, food science, and related studies, *Food Safety and Quality Assurance* teaches the basics in food production technology for foods of animal origin. Emphasis is on how these foods may become contaminated and serve as sources of foodborne disease. *Food Safety and Quality Assurance* provides an overall view of the food chain, so that the user may clearly recognize potential sources of food contamination, and focuses on efficient prevention and consumer protection.

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This book is composed of three parts: "Food Production Technology: The Food Chain," "Foodborne Disease," and "Consumer Protection." The specific aims are to (1) identify human health hazards in foods of animal origin, (2) identify the role of veterinarians in preventing introduction of hazards into the food chain, (3) identify agencies and their activities in maintaining safety in foods, (4) identify principles of safe food handling and processing, and (5) collect and analyze data relevant to investigation of foodborne disease outbreaks.

New to this edition are sections on the production of ducks and rabbits and hazard analysis critical control points, as well as inspection, in Canada. The section on aquatic animal production has undergone substantial revision necessitated by rapid change in the industry. Likewise, the section on controlling chemical adulteration has been revised in accordance with increasing public and governmental concern. Current figures and tables are presented throughout the text, and each of the three parts concludes with a complete bibliography.

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