Clinical Mastitis in Cows Treated with Sometribove (Recombinant Bovine Somatotropin) and Its Relationship to Milk Yield

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ABSTRACT

Effect of sometribove (methionyl bovine somatotropin) on mastitis in 15 full lactation trials (914 cows) in Europe and the US and 70 short-term studies (2697 cows) in eight countries was investigated. In full lactation studies, sometribove (500 mg/2 wk) was given for 252 d, commencing 60 d postpartum. Al-

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though herds varied considerably, incidence of clinical mastitis within a herd was similar for cows receiving control and sometribove treatments. Relative risk analyses indicated no treatment effect, and percentage of mastitis during treatment was similar for control and sometribove groups. A positive linear relationship existed between peak milk yield and mastitis incidence (percentage of cows contracting mastitis or cases per 100 cow days); sometribove treatment did not alter this relationship. Increases in mastitis related to milk yield increase from sometribove or related to genetic selection were similar. When expressed per unit of milk, mastitis incidence declined slightly as milk yield increased; this relationship was not altered by sometribove. No effect on clinical mastitis was observed in 70 commercial herds utilizing sometribove for 84 d. However, effects were significant for stage of lactation and milk yield. Overall, studies represented a wide range of research and commercial situations demonstrating that sometribove had no effect on incidence of clinical mastitis during the lactation of treatment. Furthermore, sometribove did not alter typical relationships between milk yield or herd factors and incidence of clinical mastitis.

(**Key words**: milk yield, mastitis, somatotropin)

INTRODUCTION

Improvement of efficiency and economic return is an important goal in dairy farming. Bovine somatotropin improves efficiency and, thus, represents one of the first products of biotechnology for animal production. Evaluation of new technology should include the impact on animal health. Studies (2, 3, 12, 17, 28, 29, 34) have consistently shown that administration of bST increases milk yield and productive efficiency without catastrophic effects on animal health. However, detection of possible subtle effects on cow health is more difficult and thus requires large numbers of cows studied under a range of environmental and management conditions (14).

The impact of any new technology on mastitis is of interest because mastitis generally is recognized as the most costly and widespread nonepizootic disease among dairy cows. Susceptibility to mastitis is related to many factors, especially environmental conditions and milking management practice (7, 33, 35, 38). Mastitis depresses subsequent milk yield, probably through damage to secretory tissue, and reduced milk yield is the major component of the total cost of mastitis (6, 21). Thus, surveys based on retrospective data (7) tend to indicate negative correlations between clinical mastitis incidence and milk yield; however, these surveys give no indication of the extent to which the yield of a healthy cow may predispose it to mastitis. Other approaches suggest that high milk yield may be associated with an increased susceptibility to mastitis. For example, genetic correlations between milk yield and clinical mastitis or SCC consistently are positive (13, 41). Other genetic studies have indicated that the annual gain in yield through genetic improvement (approximately 45 to 60 kg/yr of milk) is accompanied by a small increase of .4 to 1.3% in the incidence of clinical mastitis (33, 48) and an annual increase of .02 cases of clinical mastitis per cow (42). In addition, epidemiological studies (1, 38, 45) have shown that higher herd milk yield was associated with a higher rate of mastitis. Consideration of these data indicates that selection for higher milk yield inevitably increases susceptibility to mastitis. However, the actual increase in the rate of clinical mastitis that is due to increased milk yield is extremely small and largely obscured by factors that cause mastitis and by simultaneous improvements in mastitis control (5, 13, 22, 23, 25, 35, 38, 40, 41, 43, 48).

Our objective was to examine the effect of administration of bST on the incidence of clinical mastitis. To aid in the detection of possible subtle effects, research trials with a similar protocol were combined, including 15 full lactation studies conducted in Europe and the US. We also examined the relationship between milk yield and incidence of clinical mastitis and evaluated whether bST altered this relationship. In addition, we evaluated data from field trials involving 70 short-term studies conducted on commercial herds in eight countries.

MATERIALS AND METHODS

Full Lactation Studies

General Procedures. Data on milk yield and clinical mastitis were from 15 full lactation studies that are listed in Table 1. All cows were of the Holstein or Holstein-Friesian breeds, except for the Jersey cows used in one study (32). At each site, cows were assigned randomly within two parity groups (primiparous and multiparous) to either of two treatment groups. Neither mastitis history nor pretreatment data for mastitis variables were considered in treatment assignments. One group received injections of a prolonged-release formulation of 500 mg of sometribove (methionyl bST; Monsanto Co., St. Louis, MO), and the other group received an equal volume of excipient. Treatments were initiated at 60 ± 3 d of lactation and repeated at 14-d intervals for 252 d (18 injection cycles). Injections were given intramuscularly (4, 20, 24, 31, 36) or subcutaneously [P. J. Eppard, 1988, unpublished data, (18)]. One study (46) confirmed the bioequivalence of milk yield response by both routes of administration.

Cows throughout lactation at all sites, independent of treatment group allocation, were housed in free stalls or tie stalls and were individually fed a TMR (4, 18, 20, 24, 31, 32, 36, 46) or forage with concentrates for ad libitum intake according to yield [(31); France, Germany].

Cows were milked twice daily, and individual milk yields were recorded. Composite milk samples were obtained weekly from individual cows for determination of fat content. These values were used to adjust daily milk yields to 3.5% FCM.

Clinical Mastitis Assessment. Clinical mastitis was determined by the presence of abnormal foremilk or signs of udder inflammation (i.e., heat, swelling, or redness); each quarter was considered independently. A new case of clinical mastitis was defined as when ≥21 d had elapsed between reports of clinical mastitis, when a different quarter was affected, or

TABLE 1. Characteristics of the full lactation studies.

Reference	Location	Cows		Treatment period milk yield	
		Control	Sometribove	Control[1]	Sometribove[2]
		(no.)		(kg/d)	
(36)	Missouri[3]	62	59	23.9	5.4
(4)	New York[3]	39	40	27.9	3.1
(20)	Arizona[3]	40	39	26.3	2.3
(24)	Utah[3]	35	36	18.4	3.8
(31)	England	45	44	19.8	4.0
(31)	France	27	29	23.1	4.2
(31)	Germany	30	29	23.4	2.5
(31)	Netherlands	31	31	26.6	5.0
	Missouri[4,5]	21	21	26.2	5.2
(46)	Missouri[6]	19	42	26.2	6.2
(32)	Vermont	21	21	17.9	5.3
(18)	Arizona[4]	15	14	26.1	1.9
(18)	New York[4]	18	17	26.7	3.9
(18)	Florida[4]	16	18	19.9	4.2
(18)	Utah[4]	12	10	28.7	4.1

[1]Average daily milk yield (3.5% FCM) for control cows over the 252-d treatment period (d 60 to 312 postpartum).

[2]Increase in average daily milk yield (3.5% FCM) in sometribove-treated cows over controls for the 252-d treatment period.

[3]Intramuscular clinical.

[4]Subcutaneous fose.

[5]Unpublished data.

[6]Subcutaneous intramuscular.

when a different pathogen was isolated. Primary detection of clinical mastitis was by personnel involved in milking the cows, and, in all cases, these individuals did not know the treatment assignments of the cows. Additional observations were recorded during clinical examinations by veterinarians who also were unaware of treatment assignments. Each location followed the standard operating procedures for that herd so that routine practices and treatments related to mastitis varied over sites. However, at each site, control and sometribove-treated cows were similarly assessed and treated for mastitis. Abnormalities in milk or signs of udder inflammation were recorded at each milking. Whenever practical, a milk sample was collected from the affected quarters for microbiological culture. The microbiological identification of causative agents of clinical mastitis was not uniformly determined at all trial locations, and data were not summarized. The decision to treat clinical mastitis was at the herder's discretion or, when applicable, at the herd veterinarian's discretion and followed the routine policy for that herd. If clinical signs persisted after treatment, or if mastitis was severe or accompanied by systemic clinical signs, those cows were examined by the herd veterinarian. These consulting veterinarians also were not aware of the treatment group assignments of individual cows.

Statistical Analyses. The incidence of clinical mastitis for each cow and period was characterized by a binary category indicating whether or not the cow had mastitis starting in the period and a case rate per cow per 100 d. The period was pretreatment (parturition to start of treatment) or a standard 252-d treatment period. Thirty-three cows (16 control and 17 treated) were removed from the study prior to completion of two-thirds of the treatment period for reasons unrelated to treatment. These cows were excluded from statistical analysis of previously reported milk yield because they had incomplete lactation data, but they were included in the mastitis analyses.

Contingency table analyses were used to analyze measures associated with mastitis. Frequencies of cows that had ≥1 case of mastitis were analyzed with tables showing 15 studies × two parities × two treatments × two cases of mastitis or not. For statistical evaluation, case rate was put into three categories: none, low (>0 to 1), or high (>1). The difference between control and treated cows was tested with the Cochran-Mantel-Haenszel chi-square statistic for combining data from several sites, which also was used to calculate relative risks of a cow ever having mastitis (16).

Normality and heterogeneity of variances were examined using study, parity, treatment, and interactions of study × parity, study × treatment, and parity × treatment in the model for mastitis cases per unit of 3.5% FCM. Normality was tested using the PROC UNIVARIATE program (37). Because the data did not pass the normality test, data were transformed: $\log_{10}$, square, square root, and reciprocal. No transformation succeeded in removing nonnormality or heterogeneity; therefore, weighted analysis of variance was performed on untransformed data.

The analysis was used to examine the effect of milk yield on mastitis incidence adjusted for herd differences that arose from factors such as management practices and environmental conditions. As a measure of yield, the highest weekly average milk yield during treatment, exclusive of milk associated with mastitic days, was considered to be a more reliable indicator than total lactation yield or average yield to the onset of mastitis because mastitis can occur at any time in the treatment period, and subsequent milk yield may be depressed. Logistic regression analysis was used to examine the effect of bST treatment in the full lactation studies; peak milk adjusted for study and parity effects was included.

The SCC data were determined at weekly intervals for 13 of the 15 sites. Data for the remaining 2 sites were not included because determinations were at different frequencies [biweekly for England and monthly for The Netherlands (31); Table 1]. Data were analyzed for 13 studies that had weekly SCC observations. Monthly averages for individual cows were covariately adjusted for pretreatment SCC and then analyzed as a split plot with repeated measures.

Short-Term Studies

Additional data on clinical mastitis incidence were obtained in 70 short-term trials using sometribove in commercial dairy herds in eight countries (France, 20 herds; England,

18 herds; South Africa, 15 herds; US, 9 herds; Zimbabwe, 4 herds; Czechoslovakia, 2 herds; Italy, 1 herd; and Malaysia, 1 herd). Cows were randomly allocated to either of two treatment groups on the basis of parity, previous yield, and stage of lactation. Treated cows received subcutaneous injections of sometribove (500 mg every 14 d) for a continuous 12-wk period commencing after d 60 of lactation. Controls were untreated and were monitored over a similar 12-wk period. Cows were managed and fed according to the usual practices on each farm. All cows were milked twice daily, and individual milk yields were recorded on two consecutive milkings at a minimum of weekly intervals.

As with the full lactation studies, clinical mastitis and milk yield data pooled from 70 short-term (12-wk) studies involving 2697 cows were subjected to statistical analysis. Because of the short duration of these studies, cows were at various stages of lactation. Similarly, logistic regression analysis was used to examine the effect of bST treatment on occurrence of mastitis in the short-term studies after adjustments were made for peak milk yield and other sources of variation.

RESULTS AND DISCUSSION

Characteristics of the full lactation studies are presented in Table 1. The 15 US and European studies [P. J. Eppard, 1988, unpublished data, (4, 18, 20, 24, 31, 32, 36, 46)] involved a total of 914 cows. Over the treatment period (d 60 to 312 postpartum), the 3.5% FCM yield averaged 17.9 kg/d for the study involving Jerseys (32) and ranged from 18.4 to 28.7 kg/d for the 14 studies involving cows of the Holstein or Holstein-Friesian breeds [P. J. Eppard, 1988, unpublished data; (4, 18, 20, 24, 31, 36, 46)]. Treatment with sometribove increased daily milk yield 4.1 kg/cow, on average, across herds.

A pooled analysis of the mastitis data from the full lactation studies in which sometribove was administered is summarized in Table 2. During pretreatment and treatment, clinical rates of mastitis were comparable with those for well-managed commercial dairy herds in the US (19) and England (47). Investigators have previously shown that the incidence of mastitis is greater during the first portion of lactation (8, 30), as observed in the present study (Table 2). Comparison of the two groups indicated that the incidence of mastitis during pretreatment was greater in the sometribove group. Thus, prior to initiation of sometribove or excipient injections, a greater percentage of cows in the sometribove group became mastitic, and incidence of cases per 100 cow days was higher (P = .033; Table 2). During treatment, the sometribove group continued to have a greater percentage of cows that contracted mastitis (P = .001) and a higher incidence of cases per 100 cow days (P = .002).

The incidence of mastitis can differ dramatically among herds. Variation among herds also was evident in our full lactation studies [P. J. Eppard, 1988, unpublished data; (4, 18, 20, 24, 31, 32, 36, 46)] and offered the opportunity to compare mastitis indices between control and sometribove groups during the 60 d prior to initiation of treatment and the 252 d of treatment. For the percentage of cows that became mastitic, the correlation between sometribove and control groups was .57 during pretreatment (d 1 to 60 postpartum) and .87 during treatment (d 60 to 312 postpartum) (Figure 1). When data were expressed as mastitis cases per 100 cow days, the correlations between sometribove and control groups were .67 and .48 for pretreatment and treatment, respectively (Figure 2). Thus, although herds varied considerably in incidence of clinical mastitis, incidence within a herd tended to be similar for the control and sometribove groups during pretreatment and treatment.

Monthly average SCC and $\log_{10}$-transformed SCC from the 15 full lactation studies increased linearly over time (P = .0001) for control and sometribove-treated cows (Figure 3). Although monthly means tended to be numerically larger for cows in the sometribove group, neither treatment effect (P = .189) nor the treatment × time interaction (P = .204) was significant for the untransformed data. For the log-transformed data (Figure 3), the overall treatment effect was not significant (P = .191), but the treatment × time interaction was significant (P = .032). A treatment difference in slopes over time was indicated (P = .0006). However, treatment comparisons within each month were nonsignificant (P = .189), demonstrating that slope differences were minimal during the study.

Variable	Control	Sometribove	Significance
Cows, no.	447	467	
Pretreatment[2]			
Total cases	76	134	
Cows with mastitis,[3] %	11.4	16.3	.033
Cases per 100 cow days	.28	.47	.033
Treatment[2]			
Total cases	227	339	
Cows with mastitis,[3] %	21.3	29.6	.001
Cases per 100 cow days	.22	.35	.002
Distribution over lactation[4]			
Pretreatment, %	31.5	33.4	.680
Treatment, %	68.5	66.6	

[1]Data pooled for the 15 full lactation studies.

[2]Pretreatment was from parturition to d 60 postpartum, and treatment was from d 60 to d 312 postpartum.

[3]Percentage of cows that contracted mastitis during the period indicated.

[4]Proportion of cases of clinical mastitis occurring during pretreatment and treatment. Calculated on an individual cow basis (SD = 3.9% for control group and 3.2% for sometribove group) using only records for cows with mastitis.

Mastitis-related variables were not used in assignment of cows to groups. The higher values of the sometribove groups for mastitis variables prior to the initiation of treatments suggest that those cows may have a greater predisposition to mastitis (Table 2); the distribution of herds and the similarity of the correlation coefficients for mastitis variables between sometribove and control groups during pretreatment and treatment provide strong sup-

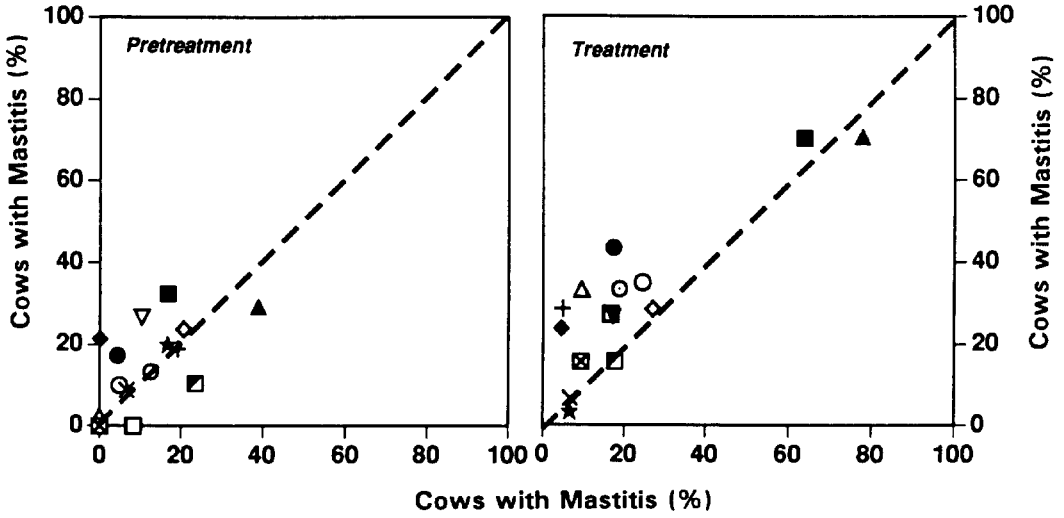

Figure 1. Comparison of the percentage of cows that contracted mastitis in control and sometribove groups in the same herd during pretreatment (d 0 to 60 postpartum) and treatment (d 60 to 312) periods. Symbols depict 15 studies at Missouri (36) (◊), New York (4) (△), Arizona (20) (○), Utah (24) (■), England (31) (X), France (31) (▽), Germany (31) (★), Netherlands (31) (⊠), Missouri (P. J. Eppard, 1988, unpublished data) (+), Missouri (46) (♦), Vermont (32) (●), Arizona (18) (⊙), New York (18) (□), Florida (18) (◰), and Utah (18) (▲).

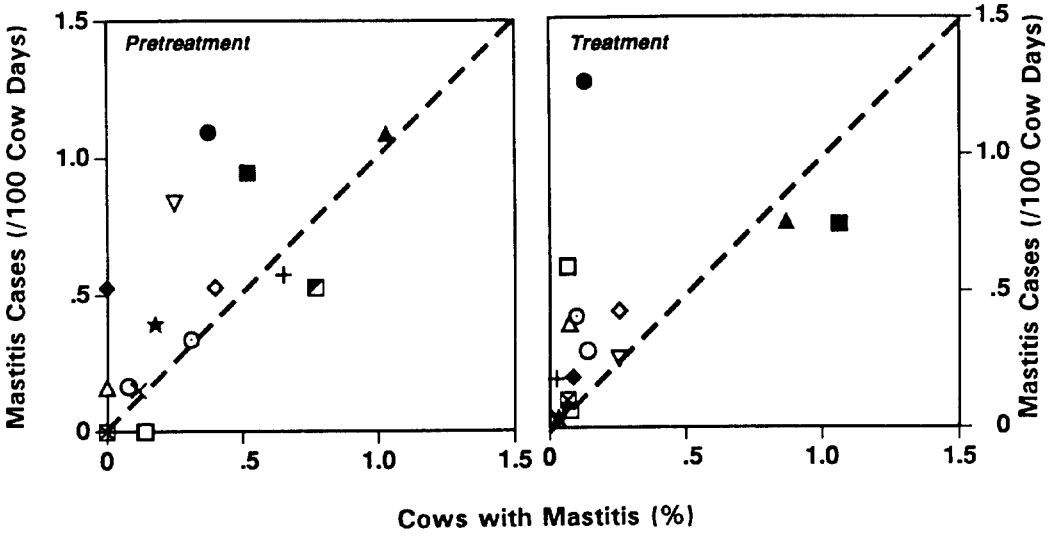

Figure 2. Comparison of the mastitis cases per 100 cow days in control and sometribove groups in the same herd during pretreatment (d 0 to 60 postpartum) and treatment (d 60 to 312) periods. Symbols depict 15 studies at Missouri (36) (◊), New York (4) (△), Arizona (20) (○), Utah (24) (■), England (31) (X), France (31) (▽), Germany (31) (★), Netherlands (31) (⊠), Missouri (P. J. Eppard, 1988, unpublished data) (+), Missouri (46) (♦), Vermont (32) (●), Arizona (18) (⊙), New York (18) (□), Florida (18) (◩), and Utah (18) (▲).

port for this idea (Figures 1 and 2). This conclusion also can be supported by comparison of mastitis cases per cow over the full lactation (Table 2). For control cows, 31.5 and 68.5% of the mastitis cases occurred during pretreatment and treatment, respectively, and 33.4 and 66.6% for the sometribove group. The lack of difference between the groups (P = .680) suggests that the higher incidence of mastitis in the sometribove group during treatment was due to a greater predisposition for mastitis. However, when records for cows that became mastitic during pretreatment were removed from the analysis, the percentage of cows developing mastitis and the cases per 100 d during treatment were still higher in the sometribove group (P = .002 and P = .005, respectively).

Major factors affecting the incidence of mastitis relate to environmental conditions and management practices (7, 33, 35, 38). However, in studies involving large numbers of cows and an appropriate statistical design, a positive relationship has also been observed for incidence of mastitis and milk yield. Although this relationship is small and represents only a minor causative component of mastitis, it has been observed for genetic studies (13, 33, 41, 42, 43, 48) and phenotypic correlations (1, 38, 43). We further examined the relationship between milk yield and mastitis indices

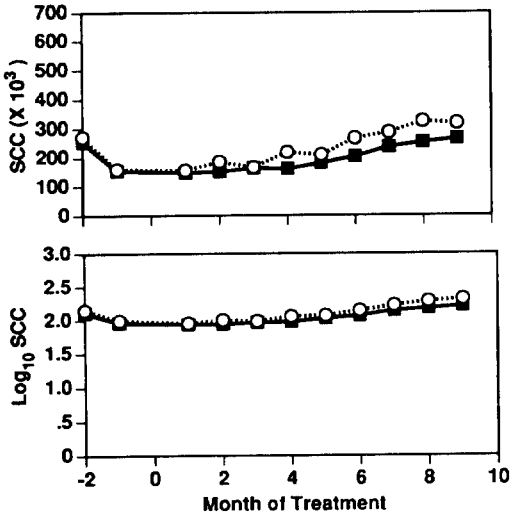

Figure 3. Milk SCC from sometribove and control group cows. Mean SCC ($\times 10^3$) (top) and mean of $\log_{10}$ SCC (bottom) for sometribove (○) and control (■) cows by month of study.

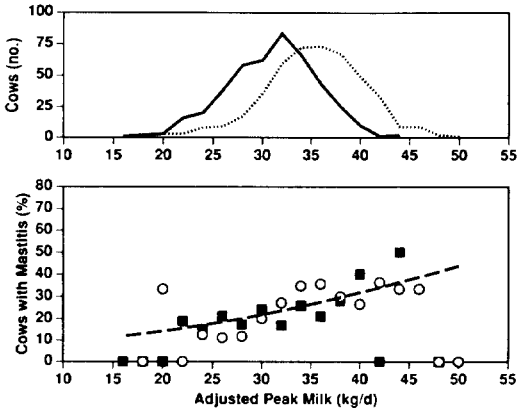

Figure 4. Relationship between percentage of cows with mastitis and peak milk yield. Number of cows in sometribove (■ ■ ■) and control (—) groups at peak yield (increments of 2 kg/d) (top). Percentage of cows that contracted mastitis for sometribove (○) and control (■) cows for each increment of 2 kg/d in peak milk yield (bottom).

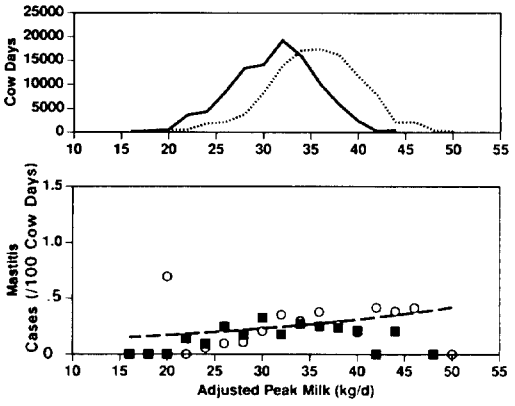

Figure 5. Relationship between mastitis cases per 100 d and peak milk yield. Cow days in sometribove (■ ■ ■) and control (—) groups at peak yield (increments of 2 kg/d) (top). Mastitis cases per 100 cow days in sometribove (○) and control (■) cows plotted against peak milk yield (adjusted for site and parity) in increments of 2 kg/d.

and determined the effects of bST on this relationship. Figure 4 presents the number of cows with mastitis at increments of 2 kg/d in peak milk yield. Because mastitis may depress subsequent milk yield, peak weekly average milk yield was utilized to minimize potential confounding effects. Both treatment groups had a binomial frequency distribution for milk yield. However, the curve for the sometribove-treated group was shifted to the right, which is consistent with the average increase of 4.1 kg/d over the control group. The relationship during treatment between the percentage of cows that became mastitic and peak milk yield was linear ($P = .010$) and did not differ between the control and sometribove groups ($P = .168$; Figure 4). The relationship between mastitis cases per 100 cow days and peak milk yield was also linear ($P = .009$) and unaffected by bST treatment ($P = .086$; Figure 5). Thus, milk yield increased as the indices of clinical mastitis increased. Although a positive relationship between mastitis incidence and milk yield has been noted in genetic and phenotypic correlations (1, 13, 33, 38, 41, 43, 48), our study is the first to examine the effect of supplemental bST and to demonstrate that sometribove treatment does not alter this relationship (Figures 4 and 5).

The relationship between milk yield and mastitis incidence in sometribove-treated cows also can be compared with previously reported genetic relationships. Shook (41) used a conservative correlation (.3) between mastitis incidence and milk yield and reported that the annual genetic response to selection for milk yield (53.4 kg/yr) was accompanied by an increase of .02 in cases of mastitis. Differences between groups during treatment for mastitis (Table 2) and milk yield can be used to calculate the increment that is due to sometribove treatment, an increase of .01 cases per 53.4 kg of milk. In other genetic studies (33, 48), this relationship has been expressed as a percentage of change in the incidence of mastitis; based on a conservative genetic correlation (.3), the annual increase in mastitis cases would be .4 to 1.4% in response to genetic gains in milk yield. Differences in mastitis cases (Table 2) and the proportion of the mastitis occurring during treatment were used to calculate an increase of 1.2%/53.4 kg of milk yield for the sometribove group. Thus, treatment differences in mastitis variables for the sometribove group were within the range predicted using estimates from genetic studies. Clearly, this comparison indicates that sometribove treatment,

TABLE 3. Relative risk for clinical mastitis expressed as the percentage of cows that contracted mastitis.

	Pretreatment	Treatment	CMH Relative risk[1]	95% Confidence interval	
				Lower	Upper
Control	11.4	21.3	1.86	1.39	2.49
Sometribove	16.3	29.6	1.82	1.44	2.29
CMH Relative risk	1.42	1.42			
95% Confidence interval					
Lower	1.03	1.15			
Upper	1.97	1.74			

[1]Cochran-Mantel-Haenszel (CHM) relative risk is a weighted average of the separate odds ratios from each study.

per se, had no adverse effects on mastitis variables during the treatment period (Table 2).

Because mastitis incidence increases as milk yield increases, the incidence of mastitis per unit of milk yield is important to establish. This relationship is shown in Figure 6. In control and sometribove-treated cows, mastitis incidence per unit of milk yielded declined slightly as milk yield increased. Thus, the number of cases of mastitis per unit of milk produced was not increased in sometribove-treated cows, and the overall average was .096 cases per 1000 kg of 3.5% FCM over the treatment period.

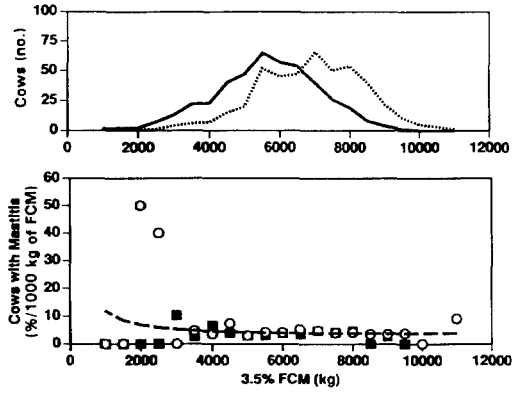

Figure 6. Relationship between milk yield and mastitis incidence per unit of milk yield. Number of cows plotted against the total lactation milk yield (d 60 to 312 of lactation) for sometribove (■■■) and control (—) groups (top). Percentage of cows that contracted mastitis per 1000 kg of 3.5% FCM for sometribove (o) and control (■) cows plotted against total milk yield (adjusted for site and parity) (bottom).

Relative risk analyses were conducted to determine whether the relative risk of contracting mastitis was altered in sometribove-treated cows (Tables 3 and 4). Relative risk was calculated using the percentage of cows with mastitis (Table 3) and the number of days on which mastitis was reported per 100 cow days (Table 4). Both analyses indicated no increase in relative risk for the sometribove-treated cows from pretreatment to treatment and no difference in relative risk for treated cows relative to control cows.

We also examined clinical mastitis in 70 studies (2697 cows) with commercial herds. These studies involved a 12-wk treatment period in which treatment with sometribove or excipient were initiated after 60 d postpartum. Maximum likelihood analysis of variance of the 70 short-term studies detected a significant effect of lactation stage on clinical mastitis (P = .058). The influence of milk yield (after data were fitted to account for stage of lactation) was also significant (P = .035). However, differences among studies were not significant, and sometribove treatment did not affect incidence of clinical mastitis (P = .322). Because milk yield and stage of lactation were confounded, the analysis of variance was also performed without stage of lactation. Influence of milk yield on clinical mastitis incidence tended to be significant (P = .100), but no effect of sometribove treatment existed (P = .369).

CONCLUSIONS

Incidence of clinical mastitis in sometribove-treated cows was consistent with

TABLE 4. Relative risk for clinical mastitis expressed as days mastitis reported per 100 cow days.

	Pretreatment	Treatment	CMH Relative risk[1]	95% Confidence interval	
				Lower	Upper
Control	.66	1.08	1.55	1.33	1.81
Sometribove	1.02	1.05	1.01	.89	1.15
CMH Relative risk	1.53	.98			
95% Confidence interval					
Lower	1.27	.91			
Upper	1.84	1.07			

[1]Cochran-Mantel-Haenszel (CMH) relative risk is a weighted average of the separate odds ratios from each study.

genetic relationships between mastitis incidence and increased milk yield. Within herd and parity (i.e., under similar conditions of management, exposure to pathogens, and age), higher yielding cows contracted more mastitis than lower yielding cows. Furthermore, the observed incidence of mastitis in control and sometribove-treated cows of comparable milk yield was similar. These results provide evidence that increased incidence of clinical mastitis in sometribove-treated cows primarily reflects their higher average yield relative to untreated controls.

Several reports (9, 10, 11, 15, 26, 27, 28, 34, 39, 44) have summarized the impact of somatotropin on mastitis-related variables in lactating dairy cows. Although not as extensive, some of these have used portions of the data summarized in the present study. These summaries individually and collectively indicate that the incidence of mastitis in sometribove-treated cows is indistinguishable from known effects of herd, parity, season, milk yield, and stage of lactation or that the estimate is biologically insignificant. Results of the present report lead to similar conclusions and further demonstrate that sometribove does not alter the normal relationship between milk yield and mastitis. In addition, we observed that sometribove treatment did not alter the relative risk of clinical mastitis or the incidence of mastitis per volume of milk. Furthermore, in studies in which mammary glands of somatotropin-treated cows were bacterially challenged, sometribove protected the mammary gland from excessive loss of milk and accelerated normalization of milk composition (9, 45). Thus, with respect to mastitis variables, the performance and health of sometribove-treated cows appears to be similar to that of untreated cows of equivalent high yield.

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