PEER REVIEWED

The Impact of Free-Stall Housing on Somatic Cell Counts In Bulk Tank Milk

Terry Lehenbauer, *DVM, MPVM, PhD*
Department of Infectious Deseases and Physiology
College of Veterinary Medicine, Oklahoma State University
Stillwater, OK 74078
Ted Jones, *PhD*
Veterinary Medicine Teaching and Research Center
University of California, Davis
Tulare, CA
Larry Collar, *MS*
California Milk Producers
Pixley, CA

Abstract

Dairy housing is generally regarded as a major risk factor for environmental mastitis. Weekly somatic cell counts (SCC) spanning more than a three-year period were analyzed from herds located in the central San Joaquin Valley of California to determine the impact of free-stall housing on somatic cell counts in bulk-tank milk compared to conventional dry-lot housing. Herds with evidence of contagious mastitis pathogens were excluded from analysis. Weather data for daily rainfall and temperatures were also collected.

All herd groups had mean SCC less than 300 000 cells/mL. During winter months when most rainfall occurred, herds with open dry-lot housing had a significant increase in SCC that averaged 34 000 cells/mL more than herds with free-stall housing. Although some differences were observed in the summer, SCC were not significantly different between herds with respect to housing type.

Economic impacts upon milk production related to SCC changes were estimated to provide a seasonal benefit of $10.25 per cow per winter. The present value of this benefit alone over a 20-year planning horizon would potentially account for 36% of the additional investment cost for free-stall housing.

Introduction

Dairy herds which have achieved low somatic cell counts (SCC) through the control of contagious pathogens often experience increased frequency of infections caused by environmental bacteria.[8,19] Environmental mastitis in dairy cattle may be defined as intramammary infections with the primary reservoir being the environment in which the cow lives and not the infected mammary quarters.[19] Dairy housing obviously plays a major role in the degree of exposure to environmental organisms, especially if there are high levels of moisture and associated contamination of the cow's body, particularly the udder. Efforts in environmental design and management should be aimed towards reducing exposure and stress so that the axiom, "keep them clean, dry, and comfortable," is achieved.[12]

There are over 300,000 dairy cows in Tulare and Kings counties of the central San Joaquin Valley milkshed of California. This area lies south of Fresno where average annual rainfall is less than 254 mm (10 in.) with a historical range of 102 mm (4 in.) to 518 mm (20 in.).[14] Most of this precipitation occurs during the winter months, December through March, accompanied with heavy fog. Winter temperatures are generally mild with the coldest temperatures typically not less than -4°C (25°F).[23] Rainfall is insignificant during the summer, but summertime maximum temperatures are often very hot, exceeding 38°C (100°F) for a total of more than 15 to 20 days during this season. Daytime relative humidity is generally less than 40% during the summer.[23]

Because of the generally mild to moderate weather conditions in this part of the San Joaquin Valley, most dairy producers have relied on corral or open dry-lot housing, usually with shades. The traditional logic has been that the climatic conditions in the area south of

Fresno are sufficiently mild so that the extra costs to build free-stall housing are not warranted. However, during recent years there has been an increasing trend to build free stalls to house dairy cows in an attempt to improve cow comfort and productivity. Many of these free-stall barns have a central feed delivery lane with lock-up stanchions and slab mangers on both sides. Free-stall areas usually consist of double rows of head to head stalls on both sides of the barn, adjacent to the lock-up stanchions.[20] It is recognized that poorly designed or badly maintained free stalls could reduce potential benefits or even cause a negative impact on efficiency, production, and milk quality.[2]

Housing is generally regarded as a major risk factor for environmental mastitis,[4,8,10,17,22] although an earlier review[5] cited a lack of statistical evidence for the effect of housing or bedding on the incidence of mastitis. Moisture levels[12,21,24] and temperatures[9,21] are the two specific environmental factors that have the greatest impact on the relationship between housing and mastitis.

Earlier studies have considered the effect of milk cow housing type on mastitis.[1,8,10,15,19,22] Three of these reports showed a trend of increasing risk of environmental mastitis with confinement as opposed to systems which incorporated pastures or grazing during part of the year.[8,10,22] Type of milk cow housing lacked sufficient significance as an explanatory variable and was not included in the models developed by Miller and Bartlett[15] which studied economic effects of mastitis prevention and Schukken *et al.* [19] which evaluated risk factors for environmental mastitis. The distribution of housing type for these reports also consisted of confinement and pasturing systems typical of mid-western and eastern areas of the United States. Only the paper by Acharya[1] analyzing data from three herds in California compared open dry-lot and free-stall housing. In this study, the single herd which was continuously housed in a free-stall system showed that the incidence of environmental mastitis was independent of the effects due to either rainfall or temperature.

Seasonal trends related to mastitis or SCC in milk have been associated with confinement housing[1,4,8,10,21] although Carroll[5] discounted the existence of climatic relationships to udder infection and mastitis based on an earlier review of the literature. Goldberg *et al.*,[10] Erskine *et al.*,[8] and Smith *et al.*[21] all found higher rates of intramammary infections from environmental pathogens associated with confinement housing. Maximum rates of infection in these studies occurred in the summer months and were related to conditions of high temperature and humidity which promoted increased exposure to environmental pathogens, especially through contact with bedding material. Goldberg *et al.*,[10] however, was unable to detect any significant impact of housing on SCC in bulk-tank milk. Bramley's research[4] also showed increased risk of infection related to confinement. In that study, the greatest incidence of coliform mastitis occurred during the fall months, which was related to the predominant autumn calving pattern found in the United Kingdom and was associated with early lactation when susceptibility to udder infection was believed to be the greatest. Acharya's research[1] using data from three California herds showed a seasonal trend for increased clinical and environmental mastitis during the winter period of low temperatures and high rainfall only for the two herds that did not have total, covered free-stall housing.

Somatic cell counts provide an excellent tool for evaluating the effect of management factors, such as housing type, and the relationship of those factors to mastitis.[15] Because of the relationship of SCC to the prevalence of intramammary infection in dairy cows[7] and to milk production losses,[3,13,18] SCC provide a measure for use in large-scale observational studies on the epidemiology of mastitis.[11]

The overall objective of this retrospective, cohort study was to measure the seasonal differences in bulk-tank SCC between dairies in central California that used either open dry-lot housing or free stalls and had no significant evidence of contagious mastitis pathogens.

Materials and Methods

Data

Data consisted of weekly SCC values from bulk-tank milk samples collected from June 1, 1990 through August 11, 1993 for dairies that shipped milk to Dairyman's Cooperative Creamery Association (DCCA) in Tulare, California (Fig. 1a). These dairies were principally located in Tulare and Kings counties of central California. SCC were measured at the DCCA Patron Laboratory[a] using a Fossomatic Model 215 Cell Counter.[b] Milk samples from each dairy were also cultured approximately each month for identification of bacteria associated with contagious and environmental mastitis as well as Mycoplasma spp. using accepted microbiological methods.[16] Current dairy herd-size data were obtained from comprehensive dairy producer lists that were compiled by University of California Cooperative Extension offices for Kings and Tulare counties. Field personnel for DCCA provided information whether member dairies used free stalls or open dry-lot housing for milking cows.

Daily weather data consisting of maximum and minimum temperatures and precipitation from a weather station located in Hanford (Kings Co.), California were obtained from the Statewide IPM (Integrated Pest Management) Project[23] (Fig. 1b, 1c).

[a]DCCA Patron Laboratory is an Interstate Milk Shipper (IMS) Certified Laboratory employing technicians licensed by the State of California.

[b]Foss Food Technology Corp.; Eden Prairie, MN.

Statistical Analysis

To reduce bias caused by elevations in SCC due to contagious mastitis pathogens, data from dairies with evidence of contagious mastitis were excluded from analysis. Milk cultures were evaluated for positive results for *Staphylococcus aureus, Streptococcus agalactiae, Mycoplasma bovis,* and *Mycoplasma californicum* from bulk-tank samples collected during January 1991, January 1992, and March 1993. SCC data for a particular dairy were excluded if the number of colonies for *S. aureus* or *S. agalactiae* were greater than 100 colonies/mL on any sample date or if greater than 10 colonies/plate for *M. bovis* or *M. californicum.* Data were retained for analysis for a particular dairy if a result was positive for only one of the screening dates and colony counts were 100 colonies/mL or less for *S. aureus* or *S. agalactiae* or, correspondingly, 10 colonies/plate or less for *M. bovis* or *M. californicum.* If these respective levels of bacteria were found on more than one of the screening dates, the data were excluded.

Data were also excluded from analysis if a dairy had a combination of free-stall and open corral housing, if member dairies were located in southern California, outside of the San Joaquin Valley, or if herd size data were not available.

Because there was not independence among weekly observations over the time period evaluated for each dairy, a decision was made to analyze data only for winter and summer seasons using a single mean SCC value for each dairy for each season being considered. Winter and summer seasons were specified by the appropriate solstice and equinox occurrences. Only SCC measure-

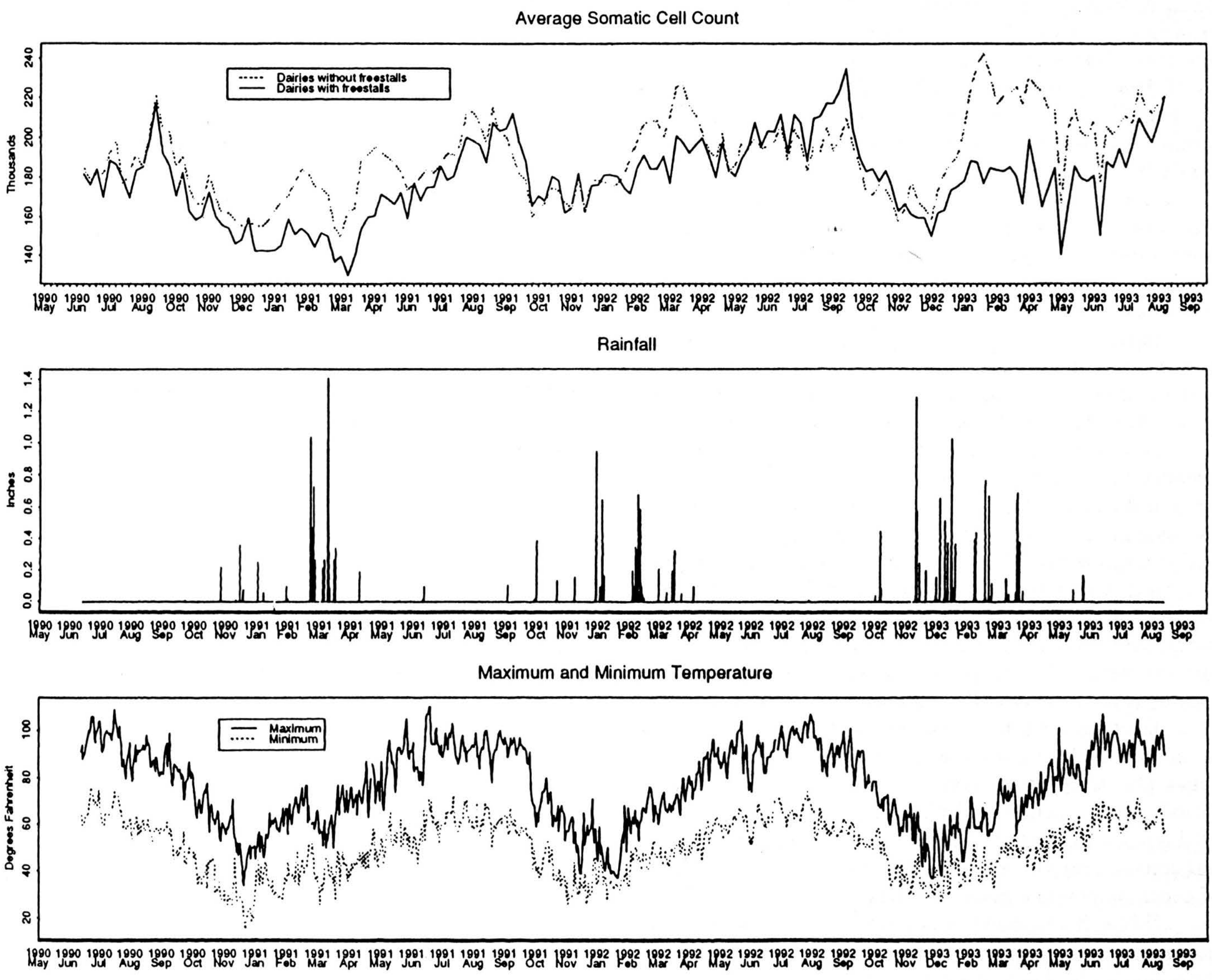

Figure 1. (a) Weekly somatic cell count averages for dairies with and without freestalls; (b) Daily rainfall in inches; (c) Daily maximum and minimum temperatures (°F).

ments observed during these seasons were used for statistical analysis for comparisons between herds with open dry-lot and free-stall housing (Fig. 1a).

Due to variation in herd size and incomplete data for each week for some herds, weekly SCC data were weighted by the product of these two parameters according to the general theory of weighted least squares regression.[6] This approach was used to deal with unequal variances which were anticipated in this situation. Data were analyzed by weighted analysis of variance (ANOVA) which provided statistics for comparisons between herds with different housing types.

Results

Descriptive Statistics

Over the study period, 252 dairies from the San Joaquin Valley shipped milk to DCCA from herds comprising more than 180,000 milking cows. Mean herd size was >700 cows (Table 1). Based on 1992 California Dairy Herd Improvement (DHI) data for participating herds in Tulare and Kings counties, average herd milk production exceeded 8,800 kg/cow (19,400 lb/cow) which would be representative of herds in this study.

Eighteen additional dairy herds located in southern California shipped milk to DCCA and were excluded from analysis. SCC data were removed from analysis for 5 member dairies for which herd size information was unavailable.

Table 1 provides descriptive statistics about the herds according to their classification with respect to housing type and evidence of contagious mastitis pathogens. Variation in herd size was minimal among the various classification groups with the exception of a small proportion of larger herds with a combination of both open and free-stall housing. It is important to note than the mean SCC for all herds as well as each of the classified groups were below 300 000 cells/mL. The overall ranges in mean SCC tended to be small among the different classification groups. The largest range occurred within the free-stall group between herds with and without evidence of contagious mastitis pathogens with counts of 261,000 and 179,000 cells/mL, respectively.

Those herds and cows from Table 1 were tabulated according to their distribution among different housing types and their respective mean SCC were calculated (Table 2). Herds housed in free stalls, representing 12% of the sample, had the lowest overall mean SCC without considering the influence of the presence or absence of contagious pathogens in herds.

Tables 3 and 4 provide information on herd distribution according to contagious mastitis pathogen status for open dry-lot herds and free-stall herds, respectively.

Data were analyzed from 124 herds with open dry-lot housing and 22 herds with free-stall housing that had no significant evidence indicating prevalence of contagious mastitis pathogens (Table 1). A higher proportion of herds with open dry-lot housing (Table 3) were removed from analysis due to contagious pathogens (43%) than were free-stall herds (27%) (Table 4). However, there were still more than 5 times as many cows included in the analysis from herds with open dry-lot housing (88,668 cows) than herds with cows housed in free stalls (15,528 cows) (Table 1) due to the large majority of herds with dry-lot housing (Table 2).

Data were incomplete over the time period examined for 22 of the 124 herds analyzed with dry-lot housing. Data were missing from either the beginning or the end of the time period and not from the middle of a sequence for these 22 herds. Data were continuous for all of the 22 free-stall herds analyzed.

Comparative Statistics

SCC weighted by the product of herd size and weekly observations were significantly lower for herds housed in free stalls compared to herds with conven-

Table 1. Herd and cow allotment, mean herd size, and mean SCC according to housing type and contagious mastitis pathogen status.

Classification	Herd Count	Number of Cows	Mean Herd Size	Mean SCC (10^3 cells/mL)
All Herds	252	182,131	723	220
Combination: Open Dry-Lot & Free-Stall Housing	5	5,345	1,069	227
Open Dry-Lot Housing	217	155,084	715	214
with Contagious Pathogens	93	66,416	714	246
without Contagious Pathogens	124	88,668	715	190
Free-Stall Housing	30	21,702	723	201
with Contagious Pathogens	8	6,174	772	261
without Contagious Pathogens	22	15,528	706	179

Table 2. Herd and cow distribution and mean SCC by housing type.

Classification	Herd Distribution	Cow Distribution	Mean SCC (10^3 cells/mL)
Combination: Open Dry-Lot & Free-Stall Housing	2%	3%	227
Open Dry-Lot Housing	86%	85%	214
Free-Stall Housing	12%	12%	201
All Herds	100%	100%	220

Table 3. Herd distribution and mean SCC according to contagious mastitis pathogen status for herds with open dry-lot housing.

Classification	Herd Distribution	Mean SCC (10^3 cells/mL)
Open Dry-Lot Housing		214
with Contagious Pathogens	43%	246
without Contagious Pathogens	57%	190

Table 4. Herd distribution and mean SCC according to contagious mastitis pathogen status for herds with free-stall housing.

Classification	Herd Distribution	Mean SCC (10^3 cells/mL)
Free-Stall Housing		201
with Contagious Pathogens	27%	261
without Contagious Pathogens	73%	179

tional, open dry-lot housing for the winter seasons but not for the summer (Table 5). Free-stall housing provided a mean reduction in SCC of 34,000 cells/mL during winter months ($P<0.05$) with the reduction ranging between 24,000 to 48,000 cells/mL. While a mean reduction in SCC of 20,000 for the summer of 1990 approached significance ($P=0.086$), the other summer seasons did not provide a significant trend (Table 6). The changes in SCC for free-stall housing for the summers of 1991 through 1993 ranged from a reduction of 20,000 cells/mL to a slight gain of 2,000 cells/mL but were not statistically significant.

Discussion

Housing type had a significant impact on SCC during the winter which is the season when most of the rainfall occurs. In contrast to earlier studies from other areas of the country which showed negative impacts of confinement housing on mastitis and SCC,[8,10,22] confinement housing in the form of free stalls for dairies located in the central San Joaquin Valley improved the SCC during winter months compared to loose housing provided by dry lots. Winter is the most challenging season from a housing standpoint in this area for controlling excessive moisture in the environment since rainfall is often negligible during the months of May through September. Correspondingly, relative humidity is reasonably low during this period of minimal rainfall.

The amount of reduction in herd SCC during the winter seasons from the weighted observations provided by free-stall housing over conventional housing varied in proportion to the amount of rainfall received during

Table 5. Weighted ANOVA: SCC by housing type for dairy herds without contagious mastitis pathogens during winter seasons.

Season	Variable	SCC (10^3 cells/mL)	Std. Error	t value	Pr (>|t|)
Winter 1990-1991	(Intercept)	163.06	4.25	38.39	
	Free-Stall Effect	-24.64	10.73	-2.30	0.023
Winter 1991-1992	(Intercept)	195.92	5.48	35.76	
	Free-Stall Effect	-30.02	13.70	-2.19	0.030
Winter 1992-1993	(Intercept)	212.17	6.28	33.79	
	Free-Stall Effect	-47.68	15.62	-3.05	0.003

Table 6. Weighted ANOVA: SCC by housing type for dairy herds without contagious mastitis pathogens during summer seasons.

Season	Variable	SCC (10^3 cells/mL)	Std. Error	t value	Pr (>|t|)
Summer 1990	(Intercept)	189.06	4.67	40.46	
	Free-Stall Effect	-20.18	11.68	-1.73	0.086
Summer 1991	(Intercept)	191.57	5.06	37.88	
	Free-Stall Effect	-11.35	12.89	-0.88	0.380
Summer 1992	(Intercept)	189.60	5.20	36.44	
	Free-Stall Effect	1.78	12.94	0.14	0.891
Summer 1993 (thru Aug. 11)	(Intercept)	206.83	5.60	34.50	
	Free Stall Effect	-20.17	14.92	-1.35	0.178

the winter (Fig. 2). Because rainfall amounts were not critically analyzed for these three winter seasons, this trend requires further study. However, this tendency intuitively supports the expected relationship that free stalls become more valuable for controlling environmental exposure as rainfall increases.

Bunching of cows housed in dry lots under shades and around water troughs during periods of excessive heat in the summer tend to produce wet, contaminated areas which soil udders of some cows. These conditions are generally believed to promote cases of environmental mastitis during hot weather. While no obvious differences were detected in temperature patterns among the summer seasons that were analyzed, considerable variation occurred in the results from year to year for the effect of free stalls on SCC. As a result, significance was not shown for summer data.

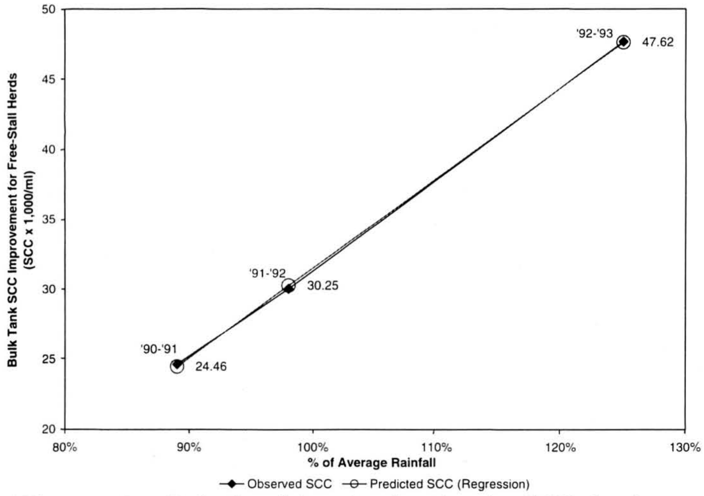

Figure 2. Relationship of reduction in SCC for free-stall herds to seasonal rainfall during 1990-91, 1991-92.

It is believed that reasonably strict criteria were used to classify herds as having evidence of contagious mastitis pathogens. However, because only one set of monthly bacteriology data per year was used to screen herds for these bacteria, it is possible that some herds may have been classified incorrectly. This screening of herds undoubtedly served to some degree as a proxy for good dairy and mastitis management. To the extent that this occurred, the resulting data used for analysis may have provided a more specific measure of the influence of free-stall housing on reducing SCC during periods of environmental stress beyond improvements in udder health that can be achieved with good management practices.

Economic Analysis

The mean reduction in SCC of 34,000 cell/mL derived from the weighted analysis for herds with free stalls has potentially significant economic implications. Based on previous work describing the relationship between SCC and milk production in dairy cows from herds in Tulare County,[25] this change, which occurred in the 160,000 to 220,000 cells/mL range of SCC for bulk tank milk, could be translated into a daily loss of 0.46 kg (1.02 lb) per cow per day during the winter season for cows housed in conventional corrals. In this range of SCC, milk losses were reported to be the same for both first lactation and multiple lactation cows. Using a milk price of $11.00 per cwt., this production loss would represent $0.1122 per cow per day or $10.25 per cow per winter season. Current free-stall investment costs over conventional loose housing using corrals and shades in central California is approximately $350 per cow. If a 20 year planning horizon and a discount rate of 8% are used, the present value of considering the milk production benefit that is only related to SCC reduction during the winter for free stalls is $126. This figure represents 36% of the free-stall investment. Other positive economic benefits are likely to exist for properly designed and maintained free stalls besides the one described in this report, which was limited to improvement in bulk tank SCC during wet weather.

References

1. Acharya, K. 1986. Influence of precipitation and air temperature on the incidence of clinical and environmental mastitis in three California dairies. M.P.V.M. Thesis. University of California, Davis. 36 pp. 2. Barnes, M. M. 1989. Update on dairy cow housing with particular reference to flooring. Br. Vet. J. 145:436-45. 3. Bartlett, P. C., Miller, G. Y., Anderson, C. R., Kirk, J. H. 1990. Milk production and somatic cell count in Michigan dairy herds. J. Dairy Sci. 73:2794-2800. 4. Bramley, J. 1985. The control of coliform mastitis. Proceedings, 24th Annu. Meet., Natl. Mastitis Council 24:4-17. 5. Carroll, E. J. 1977. Environmental factors in bovine mastitis. J. Am. Vet. Med. Assoc. 170:1143-49. 6. Draper, N. R., Smith, H. 1981. Applied Regression Analysis, Second Edition, pp. 108-16. New York: John Wiley & Sons, Inc. 709 pp. 2nd ed. 7. Erskine, R. J., Eberhart, R. J., Hutchinson, L. J., Spencer, S. B. 1987. Herd management and prevalence of mastitis in dairy herds with high and low somatic cell counts. J. Am. Vet. Med. Assoc. 190:1411-16. 8. Erskine, R. J., Eberhart, R. J., Hutchinson, L. J., Spencer, S. B., Campbell, M. A. 1988. Incidence and types of clinical mastitis in dairy herds with high and low somatic cell counts. J. Am. Vet. Med. Assoc. 192:761-65. 9. Francis, P. G., Sumner, J., Joyce, D. A. 1981. The influence of the winter environment of the dairy cow on mastitis. Bovine Pract. 16:24-27. 10. Goldberg, J. J., Wildman, E. E., Pankey, J. W., Kunkel, J. R., Howard, D. B., et al. 1992. The influence of intensively managed rotational grazing, traditional continuous grazing, and confinement housing on bulk tank milk quality and udder health. J. Dairy Sci. 1992:96-104. 11. Hueston, W. D., Heider, L. E., Harvey, W. R., Smith, K. L. 1987. The use of high somatic cell count prevalence in epidemiological investigations of mastitis control practices. Prev. Vet. Med. 4:447-61. 12. Jarrett, J. A. 1984. Environmental effects on mastitis and milk quality. In Veterinary Clinics of North America: Large Animal Practice, ed. J. A. Jarrett, 6:371-75. Symposium on Bovine Mastitis. Philadelphia: W.B. Saunders Co. 13. Jones, G. M., Pearson, R. E., Clabaugh, G. A., Heald, C. W. 1984. Relationships between somatic cell counts and milk production. J. Dairy Sci. 67:1823-31. 14. Kaweah Delta Water Conservation District. 1993. Monthly precipitation for Visalia, California, 1951-1992, P.O. Box 1246, Visalia, CA 93279. 15. Miller, G. Y., Bartlett, P. C. 1991. Economic effects of mastitis prevention strategies for dairy producers. J. Am. Vet. Med. Assoc. 198:227-31. 16. NMC, I. 1987. Laboratory and Field Handbook on Bovine Mastitis. Fort Atkinson, WI: W. D. Hoard & Sons Co. 17. Oz, H. H., Farnsworth, R. J., Larson, V. L. 1985. Environmental mastitis. Vet. Bull. 55:829-40. 18. Raubertas, R. F., Shook, G. E. 1982. Relationship between lactation measures of somatic cell concentrations and milk yield. J. Dairy Sci. 65:419-25. 19. Schukken, Y. H., Erb, H. N., Sears, P. M., Smith, R. D. 1988. Ecologic study of the risk factors for environmental mastitis in cows. Am. J. Vet. Res. 49:766-69 20. Shultz, T. A., Fairbank, W. C., Hutton, G. A. 1986. California freestall management factors. Dairy Free Stall Housing Symposium Proceedings Jan. 1986:n. pag. 21. Smith, K. L., Todhunter, D. A., Schoenberger, P. S. 1985. Environmental mastitis: cause, prevalence, prevention. J. Dairy Sci. 68:1531-53. 22. Smith, L. K. 1986. A practical look at environmental mastitis. Bovine Pract. 21:73-76. 23. Statewide IPM Project. 1993. Hanford and Visalia, California weather data: Jan. 1990 - Aug. 1993, Univ. of Cal., Davis. 24. Thomas, C. B., Jasper, D. E., Rollins, M. H., Bushnell, R. B., Carroll, E. J. 1983. Enterobacteriaceae bedding populations, rainfall and mastitis on a California dairy. Prev. Vet. Med. 1:227-42. 25. Tyler, J. W., Thurmond, M. C., Lasslo, L. 1989. Relationship between test-day measures of somatic cell count and milk production in California dairy cows. Can. J. Vet. Res. 53:182-87.