Affected by environment and climate, mastitis incidence often has a seasonal pattern. However, this pattern can also result from a pronounced seasonal calving pattern, putting many cows in a more susceptible lactation stage at the same time.

Bulk milk somatic cell count (SCC) has decreased considerably in Canada and most European countries as dairy farmers adopt a standard mastitis prevention program. This resulted partially from the introduction of bulk milk SCC penalty limits at 500,000 cells per millilitre in Canada and 400,000 in Europe. As well, low bulk milk SCC premiums, such as the British Columbia and Prince Edward Island bonus programs, have encouraged better udder health.

However, every region of the world has high bulk milk SCC problem herds. In Canada, where the 2006 bulk milk SCC averaged 230,000, five per cent of herds averaged SCCs above 400,000. In 32 per cent of herds, average SCC topped 400,000 at least once a year.

The percentage of cows with high SCCs caused by subclinical mastitis heavily influences bulk milk SCC. Southern hemisphere herds with a seasonal calving pattern have the highest bulk milk SCC around the winter calving period, July to September. In New Zealand, for example, the lowest bulk milk SCC in these herds occurs in September to October, shortly after calving, and bulk milk SCC then slowly increases again towards the end of the season in April to May.

In northern hemisphere countries, herds with year-round-calving have the same bulk milk SCC seasonal pattern, peaking from July to October (see Figure 1, page 45). It is no surprise individual SCCs are also highest in this period. The question is whether the higher percentage of cows with high SCCs in summer is caused by a larger number of new high-SCC cases or by a lower cure rate with a longer duration.

A study of 300 Dutch free-stall herds, with average bulk milk SCCs comparable to Canadian dairy herds, found a peak of 15 per cent of new high-SCC cases in older cows in May (see Figure 2). The number for heifers was less pronounced, peaking in August and September at six to seven per cent.

The percentage of new high-SCC cases that became chronic—high SCC at two or more consecutive DHI test-days—in older cows was relatively stable over the year, ranging from 75 to 80 per cent (see Figure 3). A one-time peak in SCC occurred more often in heifers—60 per cent became chronic, while there was a pronounced August peak of 70 per cent. Older cows had a new high SCC...
more often, which stayed high more frequently. Cows later in lactation more often had a new high SCC.

So, both a peak in new high-SCC cases in spring and a peak in chronic cases in summer caused the summer bulk milk SCC peak. The most likely cause of the April peak of new high-SCC cases is an increased incidence of *Staphylococcus aureus* udder infections. The August rise can be explained by an increased incidence of *Streptococcus uberis* udder infections.

The percentage of new high-SCC cases that became chronic was surprisingly large. If SCC is high for the first time, the odds are high that SCC will still be high at the next test, particularly in older cows, and even more so at the end of lactation.

Clinical mastitis incidence is also seasonal, and location is a major factor. In confined U.S. dairy herds, incidence is highest in summer months, June to August, when streptococci and coliforms (*E. coli* and *Klebsiella*) particularly play a role. Summer humidity and temperatures increase coliform counts in bedding material, increasing environmental mastitis incidence.

In countries with moderate climates, such as Canada and Northern European nations, the situation is quite different from that in the southern U.S. In the Dutch study, clinical mastitis cases peaked in December and January—1.5 times higher than the number in summer. There was a pronounced difference in the bacterial cause of mastitis between seasons. *Streptococcus uberis* mastitis peaked in August, while the peak in the other pathogens such as *E. coli*, *Staph. aureus* and *Streptococcus dysgalactiae* occurred in winter (see figures 4 and 5). We have no explanation for the short July peak in clinical *Staph. aureus* and *E. coli* mastitis.

Housing also made a difference. The *E. coli* mastitis incidence in semi- and totally confined herds peaked in summer. In herds with cows on summer pasture, the clinical *Streptococcus uberis* mastitis peak was three to four times higher than in confined herds.

In New Zealand, where cows are pastured all year, *Streptococcus uberis* most commonly causes mastitis, and *E. coli* mastitis is relatively rare. *Streptococcus uberis* likely maintains a cycle through contamination of grass with infected manure.
In the Dutch dairy herds, most *Streptococcus uberis* infections occurred during lactation, contrary to what has been reported in confined U.S. dairy herds.

Bulk milk SCC, new and chronic udder infections and clinical mastitis incidence are seasonal. Clinical mastitis incidence cannot explain bulk milk SCC increases in August and September. It is caused by an increased number of new high-SCC cows and longer periods of high SCC. Incidence of *Streptococcus uberis*
mastitis is related to pasture, while mastitis with other streptococci and E. coli are more barn-related. The study demonstrates the importance of milk culture and differentiation of mastitis pathogens to make specific recommendations in udder health control programs.


A research priority for Canadian dairy farmers
The CBMRN research program is divided into two themes: mastitis monitoring and mastitis control. Three monitoring projects are directed at answering questions raised by this study:
1. Investigating the occurrence and monitoring of new udder infections. With the help of questionnaires on management practices and milk sample collection from the National Cohort of Dairy Farms, and bacteriological analysis, this project will describe udder infection dynamics.
2. Validation of on-farm mastitis pathogen identification systems. The first phase of this project examined on-farm culture media tools for rapidly identifying bacteria in milk samples. The second phase, started in December 2007, will formulate guidelines for mastitis treatment and monitoring effects of farm antibiotic use, disease outcome, mastitis cure rates and overall clinical mastitis incidence on farms.
3. Identifying genes that enable bacteria to cause chronic intramammary infections and clinical mastitis. This project uses molecular techniques to identify genes that permit bacteria, more specifically S. aureus, to infect udders and cause mastitis.

All Canadian dairy farmers participate in funding the research program and are involved in all CBMRN decision-making levels. To find out more or to share your ideas and comments, visit the CBMRN’s website, www.mastitisnetwork.org, or contact Julie Baillargeon: Julie.baillargeon@umontreal.ca.