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Effect of udder health on milk

S.J. LACY-HULBERT AND M.J. AULDIST[1]

Dexcel Ltd., Private Bag 3221, Hamilton, New Zealand.

ABSTRACT

Mastitis is an inflammation of the udder tissue, normally arising from a bacterial infection. The effects of mastitis on the processing properties of milk have been widely researched over the past 40 years. This paper summarises the main effects of udder health on raw milk composition and its processing properties, reviews the progress made to date by the New Zealand dairy industry with regard to reducing the impact of mastitis on milk processing, and highlights continuing avenues of enquiry.

Keywords: mastitis; milk composition; processing properties.

Milk and udder health

Milk is a complex substance produced in a gland with a complicated biology. Most of the components that make milk unique are synthesised in the secretory tissue and released directly into the milk. Other components are derived from the serum or produced from post-secretory breakdown of other components (Table 1). Factors that affect the udder, such as infection, cow health, stage of lactation and season (Auldist *et al.*, 1998), degree of milk let-down or level of nutrition (Lacy-Hulbert *et al.*, 1999) have a substantial impact on milk synthesis, secretion and eventual composition. Of these factors, the most influential is mastitis but other changes that cause the somatic cell count (SCC) to rise tend to alter milk composition in a similar way. The changes in milk related to mastitis have been extensively reviewed by Kitchen (1981), Hoare (1982), Munro *et al.*, (1984), and by Auldist & Hubble (1998) and will be discussed only briefly here.

Mastitis is an inflammatory reaction of the udder tissue, usually in response to infection by pathogenic bacteria. Inflammation is characterized by redness, swelling, heat, and pain in the tissues but the reaction ensures that pathogenic organisms are identified and neutralised, that associated toxins and diseased or necrotic tissue are removed or isolated and enables tissue repair to take place. Inflammatory reactions are complex and vigorous processes (Guidry & O'Brien, 1997). In the mammary gland, inflammation is typified by an influx of leucocytes into the milk (Harmon, 1994), as indicated by an elevation in SCC. The changes in milk composition associated with mastitis and/or an elevated SCC are summarised in Table 2 whilst the subsequent impacts on yield and quality of processed products are summarised in Table 3.

Effect of mastitis on milk composition

It is well established that mastitis reduces milk yield but the extent of this reduction is variable. One review reported milk yield losses in the range of 10 to 23% per cow (Hoare 1982) whilst studies of identical twins (Woolford *et al.*, 1983) suggest that adult cows can compensate for milk losses sustained in one quarter, afflicted by sub-clinical *Staphylococcus aureus* infection, by increasing production in the unaffected quarters. However, heifers were unable to compensate and experienced 7-10% losses over the first lactation and beyond (Woolford *et al.*, 1984). The reduction in milk yield associated with mastitis is probably due to the reduced synthetic and secretory capability of the udder tissue (Oliver & Calvinho, 1995). Reduced synthesis and

TABLE 1. Origins of major milk components

Milk Component	Derived from: Blood	Synthesis in udder	Breakdown of other components
Proteins:			
Casein proteins			
α-, β-, and κ-caseins		✓	
γ-casein and protease peptones			✓
Whey proteins			
α-lactalbumin, β-lactoglobulin		✓	
Serum albumin	✓		
Immunoglobulins	✓		
Fat:			
Short chain fatty acids		✓	
Medium chain fatty acids	✓	✓	
Long chain fatty acids	✓		
Free fatty acids			✓
Lactose		✓	
Minerals, vitamins, hormones	✓		
Somatic cells	✓		

(reproduced from Mackle & Bryant, 1995)

[1] Now at Natural Resources and Environment, Agriculture Victoria Ellinbank, RMB 2460, Hazeldean Rd., Ellinbank, VIC 3821, Australia

TABLE 2. Effect of intramammary infection or high SCC on concentration of milk components

Milk Component	Change	Milk Component	Change
Total casein	↓	Serum albumin	↑
Casein:total protein	↓	Immunoglobulin G	↑
β-casein	↓	Lactoferrin	↑
α_s-casein	↓	Transferrin	↑
κ-casein	**?**	Plasmin	↑
γ-casein	↑	Non-casein N	↑
Whey protein	↑	Na	↑
α-lactalbumin	↓	K	↓
β-lactoglobulin	↓	Total Ca	↔
Total protein	**?**	Cl	↑
Fat	**?**		
Free fatty acids	↑		
Lactose	↓		

(reproduced from Auldist and Hubble, 1998)

TABLE 3. Summary of reported product defects associated with elevated somatic cell count.

Product	Effects
Cheese	Reduced yield and yield efficiencies
	Elevated moisture content
	Increased rennet clotting time
	Soft cheese and texture defects
	Higher loss of solids in the whey
	Inferior organoleptic properties
UHT milk	Accelerated age gelation
Cultured products	Increased coagulation time
	Inferior organoleptic properties
Butter	Extended churning times
	Reduced shelf life
	Inferior organoleptic properties
Milk powder	Altered heat stability
	Reduced shelf life
Cream	Altered whipping properties

(reproduced from Auldist and Hubble, 1998)

secretion of lactose by the inflamed tissues may also contribute indirectly to the milk yield loss since lactose is the main osmotic regulator of milk volume.

Mastitis affects milk composition via one of three main mechanisms:

1. The mammary epithelial cells responsible for milk secretion can be damaged by toxins released by the invading bacteria (Paape *et al.*, 1995) or by the chemical mediators of inflammation, such as histamine and cytokines. This damage leads to a reduction in synthesis and secretion of the milk components that are synthesised within the epithelium itself. Components that are particularly at risk from this mechanism include α-casein, β- casein, α-lactalbumin, β-lactoglobulin and lactose.

2. Bacterial toxins and chemical mediators can cause the tight junctions between the mammary epithelial cells to rupture, and increase permeability of the adjacent blood vessels. This leads to an increase in the paracellular pathways between milk and blood, allowing leakage of blood constituents into milk and milk constituents into blood (Stelwagen *et al.*, 1997). This mechanism may be partially responsible for the reduced concentration of lactose in mastitis milk by allowing leakage of lactose into the plasma (Sissoko *et al.*, 1984, Auldist & Hubble 1998). Increased leakiness of the mammary epithelium is also responsible for a change in the mineral balance of milk, in particular a reduction in potassium concentration and an increase in sodium and chloride ionic concentrations (Kitchen, 1981). These changes in mineral ions affect the pH and electrical conductivity of milk, which forms the basis of a widely used mastitis diagnostic tool (Woolford *et al.*, 1998). Increased movement into milk of somatic cells and the blood serum proteins, serum albumin and immunoglobulins, are also aided by leakiness of the epithelium.

3. The activity of hydrolytic enzymes in milk increases during mastitis, leading to post-secretory breakdown of milk components. These enzymes may be released by the bacteria themselves (Saggers & Stewart, 1968; Leigh, 1994), or by the leukocytes or by the damaged tissues. The most important enzyme is probably plasmin, a proteinase normally present in milk in its inactive form, plasminogen. Mastitis increases activation of plasminogen to plasmin, resulting in increased breakdown of β-casein into γ-casein and smaller polypeptide fragments (proteose peptones). Hydrolysis of α-casein may also occur. Plasmin is heat stable, and survives the high temperatures of processing so can continue to cleave casein in the final product (Bastian & Brown, 1996). Plasminogen activators have been isolated from damaged tissue and certain mastitis pathogens (Leigh, 1994). Other enzymes that show an increased activity in mastitic milk include catalase, lactate dehydrogenase, lipases, and various esterases and glycosidases (Kitchen, 1981). Increased lipolytic activity in the milk has important implications for the milkfat, damaging the milk fat globule membrane and cleaving the triacylglyceride molecules, to release free fatty acids (Auldist & Hubble, 1998). These fatty acids are responsible for the development of rancid flavours in milk (Duncan *et al.*, 1991) and in butter products.

In summary, the effect of mastitis on milk composition typically includes a reduction in casein concentration and concomitant increase in whey protein concentration, which results in a reduction in the ratio of casein:whey; an increase in milk pH; and a degradation of the milkfat.

Effect of mastitis on quality and yield of milk products

Most studies of the effects of mastitis on product yield and quality have focussed on cheese manufacture but mastitis has detrimental effects on the manufacture of most dairy products (Table 3).

Cheese

The reduction in casein:whey concentration usually results in a reduction in cheese yield whilst the increased pH of mastitic milk, coupled with a sub-optimum ratio of casein:whey, increases coagulation times and moisture content of the final product (Auldist *et al.*, 1996a). Elevated moisture content is one of the more serious cheese quality defects and is responsible for reducing curd firmness and a deterioration in texture and organoleptic properties of cheese. Hydrolytic enzymes associated with high SCC milk may be responsible for flavour defects occurring during the ripening process (Auldist & Hubble, 1998).

Changes detrimental to cheese manufacture have been observed in milks with a SCC above 100,000 cells/ml (Politis & Ng-Kwai-Hang, 1988a,b,c; Barbano *et al.*, 1991) but changes in raw milk up to a SCC of 500,000 cells/ml are generally tolerated by cheese manufacturers.

Other dairy products

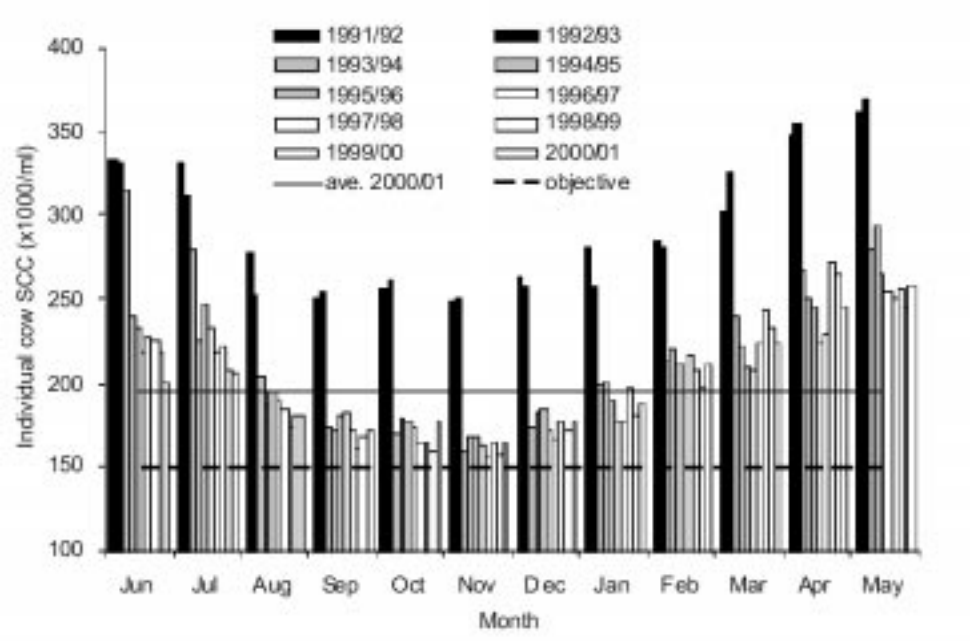
For products other than cheese, mastitis tends to reduce the functional (Needs *et al.*, 1988) and organoleptic properties, and increase coagulation times (Szakaly *et al.*, 1990a, b). Products made from milks with high SCC tend to show more deterioration on storage (Ma *et al.*, 2000) and are less heat stable (Auldist *et al.*, 1996b,c).

Milk quality incentive schemes for SCC

Dairy companies around the world routinely include SCC as a measure of milk quality. They normally operate milk quality payment schemes with premium payments for high quality milk and/or penalties for below-standard results (Hubble, 1997) and countries that seek to market dairy products within the European Union must operate systems that aim to exclude from the processing supply bulk milks with a SCC above 400,000 cells/ml. The EU is currently discussing whether this regulatory level should be dropped to 300,000/ml or even to 250,000/ml (Smith *et al.*, 2000).

Substantial progress has been achieved by New Zealand in terms of reducing its national average SCC in the past 10 years (Figure 1). This has been achieved by

FIGURE 1: Changes in individual cow SCC over the past 10 years for 95% of NZ dairy cows[1]. Since 1993/94, financial penalties have been imposed for bulk milk above 400,000 cells/ml, tested once every 10 days (grey bars) and per consignment testing for bulk milk SCC has occurred for an increasing proportion of herds since 1996/97 (white bars). Solid line represents the average SCC, weighted for milk yield, for the 2000/2001 season, the dashed line represents the objective for the national average SCC, set by the National Mastitis Advisory Committee.



[1] data from Livestock Improvement Corporation

adoption of a penalty scheme for milk quality, initially for a SCC above 500,000/ml (1993-1994) and then for a SCC above 400,000/ml (1994-1995), and by increasing the frequency of bulk milk SCC monitoring to per consignment testing (since 1996; Lacy-Hulbert, 1998). Introduction of the SAMM plan (Seasonal Approach for Managing Mastitis) in 1993 provided practical on-farm tools for managing and reducing the incidence of mastitis (Woolford *et al.*, 1995) and aided the reduction in SCC, but little further progress in SCC trends has been achieved since 1997, and new incentives are now required. Other countries have made good progress in reducing their SCC average over the past 10 years (Table 4), particularly our competitors in the EU marketplace, such as the UK, Germany, and the Scandinavian countries. The development and marketing of new products that capitalise on improved functionality, yield and consumer perception of very low SCC milk is likely to provide incentives to encourage further declines in SCC. With whom the responsibility lies for capturing this market is not clear. Dairy industry processors and marketers may be the most obvious group but the farming community could also take the lead by demonstrating capability to consistently supply very high quality milk.

TABLE 4. Changes in national average bulk milk SCC over the past 10 years for selected countries.

	Mean cell count (thousands/ml)			
Country	1990[1]	1993[1]	1998-2000[2]	Type of Mean[3]
Austria	379	313	-	A
Belgium	307	265	-	G
Denmark	368	309	247	A
Finland	282	186	129	A
Germany	274	237	West – 195 East - 276	G
Hungary	419	351	-	A
Iceland	471	408	-	A
Israel	-	-	382	
Italy	434	426	-	A
Japan	260	280	300	A
Netherlands	320	280	-	A
Norway	206	194	125	A
New Zealand	345	255	180	W
Sweden	230	231	200	G
Switzerland	117	104	112	A
UK	329	277	160	G
USA	-	-	350 (estimate)	

[1] Data from Booth, 1995

[2] Data from Smith *et al.*, 2000

[3] Means for 1990 & 1993: A = arithmetic, G = geometric, W = weighted

Conclusions and future research

Mastitis can be caused by a variety of different bacteria, each with a different capability and pathogenic mechanism for infecting the gland. It is highly probable that different pathogens will affect milk composition in different ways, irrespective of the SCC (Auldist, 2000). The leucocytes that are released into the gland have the potential to differentially affect milk composition. In low SCC milk the predominant leucocytes, macrophages, lymphocytes and polymorphonuclear neutrophils (PMN) are present in similar proportions but in high SCC milk, PMN are present in much greater numbers, representing a much higher proportion of the SCC. There is evidence that the composition of milk may be affected more severely by PMN than by other types of cells (Cooney *et al.*, 2000). Recent research in Ireland has observed that the percentage of PMN varies widely between quarters, cows and herds but that the numbers of PMN present are correlated reasonably with SCC (O'Brien *et al.*, 1999). Examining the effects of different pathogens and different types of leucocytes on milk composition will enable improved targeting of mastitis control measures and illustrate the possible benefits of increased segregation of milk, on-farm and at the factory.

The effect of very low SCC milk on all dairy products has not yet been fully explored. The reduction in SCC so far achieved in NZ has increased the casein:whey ratio of milk received at the factory (Franks, R., unpublished data) and this is likely to have increased the yield of casein based products, such as cheese. Determining the actual value of these gains, and incorporating this information into an appropriate incentive scheme would likely encourage the production of high quality, low SCC milk.

A recent study, comparing high with very low SCC milk for production of pasteurised milk, observed that the high SCC milk (>800,000/ml) showed deterioration in quality after 14 days storage whilst the very low SCC milk (<100,000/ml) showed no reduction in quality ratings after 21 days storage at 5ºC (Ma *et al.*, 2000). An increase in cheese yield of 5% and reduced rennet coagulation time of 2% was observed by Politis & Ng-Kwai-Hang (1988b) when comparing production of cheese from milks with a SCC of 500,000/ml or below 100,000/ml. Whether the majority of gains are made through reducing the SCC from 800 to 400,000 or from 400,000 to below 100,000 is debatable but there appear to be measurable gains to be achieved in terms of quality and functionality when products are manufactured from raw milks with a very low SCC (below 100,000/ml).

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