

Editorial

Bovine Mastitis: Current Concepts and Future Control Approaches

El-Hamid MIA*

Department of Microbiology, Zagazig University, Egypt

***Corresponding author:** Marwa Ibrahim Abd El-Hamid, Department of Microbiology, Zagazig University, Lecturer of Bacteriology, Mycology and Immunology, Faculty of Veterinary Medicine, Egypt

Received: October 25, 2016; **Accepted:** October 28, 2016; **Published:** October 31, 2016

Editorial

The general health and well being of individuals depends largely on meeting basic nutritional needs. Milk and milk products have formed an important part of daily nutrition. An increase in global population coupled with the increasing demands for milk as an economic food has necessitated an increase in production by dairy farmers.

In a commercial milking environment, dairy cattle need to be in perfect physical condition to maintain a high level of milk production. The risk of lesions and infections that develop in modern dairy farming has consequently increased.

Bovine mastitis is defined as inflammation of the mammary gland. It is the most serious and economically significant disease of dairy herds in dairy milk production worldwide [1].

Bovine mastitis has huge effects on farm economics with many related losses associated with reduction in milk yield, increased treatment costs, discarded milk, increase in culling and associated dairy cow replacement rates, and financial penalties for exceeding legal milk quality limits [2]. If the disease is diagnosed in early stages, the greater portion of this loss can be avoided.

Etiological agents of mastitis can be infectious or noninfectious. A wide variety of microorganisms has been implicated as causative agents of bovine mastitis including bacteria, viruses, mycoplasma, yeasts and algae (3). The majority of mastitis is of bacterial origin and just a few of species of bacteria account for most cases, such as *Escherichia coli*, *Staphylococcus aureus*, *Streptococcus uberis*, *Streptococcus dysgalactiae* and *Streptococcus agalactiae*, *Streptococcus bovis* and *Klebsiella pneumonia* [4].

Classically, mastitis pathogens have been classified as contagious and environmental pathogens based upon their primary reservoir and mode of transmission. The contagious pathogens are capable of causing subclinical infections which are typically manifested with an elevation in the somatic cell count (SCC) of milk. In contrast, the environmental pathogens are best described as opportunistic invaders of the mammary gland [5].

There is evidence that pathogens use various mechanisms to induce cell death pathways. A number of pathogens are armed with an

array of virulence determinants, which interact with key components of a host cell's death pathways or interfere with regulation of transcription factors monitoring cell survival. These virulence factors act by a variety of mechanisms such as pore-forming toxins, which interact with the host cell membrane and permit the leakage of cellular components, toxins that express their enzymatic activity in the host cytosol, effector proteins delivered directly into host cells by a highly specialized type-III secretory system, superantigens that target immune cells, and other modulators of host cell death [6].

Monitoring udder health performance is impossible without reliable and affordable diagnostic methods. Early diagnosis is of the utmost importance due to the high costs of mastitis. Diagnostic methods have been developed to check the quality of the milk through detection of mammary gland inflammation and diagnosis of the infection and its causative pathogens. Therefore, there is a constant need to improve these methods, for accuracy, cost, or convenience.

Currently, assays often used include measurement of SCCs, enzymatic analysis, California Mastitis Test (CMT), Bromo Thymol Blue (BTB), modified white side test, trypsin inhibition test, milk pH, and electric conductivity [7]. Colourimetric and fluorometric assays have been developed for measuring the concentrations of enzymes elevated in milk during mastitis as N-acetyl- β -D-glucosaminidase (NAGase) and Lactate Dehydrogenase Activity (LDH).

The most frequently used diagnostic methods are SCC and bacteriological culturing of milk. The identification of pathogens causing mastitis is important for disease control and epidemiological studies. Use of culturing techniques for the detection of mastitis-causing microorganisms is still the gold standard, although it is very labor-intensive and therefore expensive.

Recently, PCR assays have been developed for detection and quantifying mastitis pathogens in milk [8]. Advances in relevant proteomics techniques, such as two-dimensional gel electrophoresis and mass spectroscopy, have led to the identification of different protein expression pattern obtained from mastitis-infected milk and on the proteins expressed by invading pathogens. This information can be applied not only to the discovery of new therapeutic targets but also to the search for new diagnostic biomarkers [1].

The "five point plan for mastitis control" has been the gold standard for control strategies for many years and has been successful in reducing the incidence of mastitis. The five points include teat disinfection after milking, proper hygiene and milking procedures and adequate milking equipment, culling of chronically mastitis cows, antibiotic dry-cow therapy and prompt treatment of clinical mastitis during dry period and during lactation [9].

Currently, the administration of antibiotics is the most common method of treatment of bovine mastitis. However, this kind of

strategy has some disadvantages including low cure rate, increasing occurrence of resistance, and the presence of antibiotics residues in the milk. Therefore, there is an urgent need for alternatives to antibiotics for controlling bovine mastitis. A wide array of alternatives to antibiotics was investigated by several groups of researchers in order to find an effective approach for management of bovine mastitis. Bacteriophages, vaccines, nanoparticles, cytokines, and natural compounds from plants, animals, and bacteria are some examples of valid substitutes to antibiotics [10].

Finally, *in vitro* studies testing the potential uses of these compounds for future use as therapeutic strategy to treat mastitis-infected cows showed encouraging results, but the authors suggest more studies namely *in vivo*, are still critical.

References

1. Viguier C, Arora S, Gilmartin N, Welbeck K, O'Kennedy R. Mastitis detection: current trends and future perspectives. *Trends Biotechnol.* 2009; 27: 486-493.
2. Bennedsgaard TW, Enevoldsen C, Thamsborg SM, Vaarst M. Effect of mastitis treatment and somatic cell counts on milk yield in Danish organic dairy cows. *J. Dairy Sci.* 2003; 86: 3174-3183.
3. Chaneton L, Tirante L, Maito J, Chaves J, Bussmann LE. Relationship between milk lactoferrin and etiological agent in the mastitic bovine mammary gland. *J. Dairy Sci.* 2008; 91: 1865-1873.
4. Kuang Y, Tani K, Synnott AJ, Ohshima K, Higuchi H, Nagahata H, et al. Characterization of bacterial population of raw milk from bovine mastitis by culture-independent PCR-DGGE method. *Biochem. Eng. J.* 2009; 45: 76-81.
5. Philpot WN, Nickerson SC. *Mastitis: Counter Attack.* Westfalia Surge LLC. Illinois. USA. 1999.
6. Weinrauch Y, Zychlinsky A. The induction of apoptosis by bacterial pathogens. *Annu. Rev. Microbiol.* 1999; 53: 155-187.
7. Joshi S and Gokhale S. Status of mastitis as an emerging disease in improved and periurban dairy farms in India. *Annals of the New York Academy of Sciences.* 2006; 1081: 74-83.
8. Phuektes P, Browning G, Anderson G, Mansell P. Multiplex polymerase chain reaction as a mastitis screening test for *Staphylococcus aureus*, *Streptococcus agalactiae*, *Streptococcus dysgalactiae* and *Streptococcus uberis* in bulk milk samples. *J Dairy Res.* 2003; 70: 149-155.
9. Giesecke WH, Du Preez JH, Petzer IM. *Practical mastitis control in dairy herds.* Butterworth Publishers. Durban. South Africa. 1994.
10. Gomes F, Henriques M. Control of bovine mastitis: old and recent therapeutic approaches. *Curr. Microbiol.* 2016; 72: 377-382.