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MASTCURE™

Mastitis TREATMENT via feed -
with a Unique Micro-Encapuslated Technology

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MOST PROBABLE CAUSE(S) OF MASTITIS

Thus far, the most common causes of mastitis in dairy animals have been primarily imputed to the infectious agents (Zhao and Lacasse 2007). On the basis of infectious causes of mastitis a procession of drugs purported to be effective against these culprits emerged on the scene for controlling this ailment in dairy animals. In the beginning these drugs appeared specious. However, continual use of these chemicals proved palliative and confronted with biggest problem of drug resistance and milk and meat residue dangers for humans (Costa and others 1997). Moreover, the effectiveness of these antimicrobials was rarely more than 50% to control mastitis in dairy animals (Deluyker and others 2005). Different management practices e.g., dry-cow therapy, teat dipping, hygienic measures etc., were evolved to alleviate effects of this formidable problem, but the devil of mastitis is still starring and unrelenting. Nevertheless, delving disparately into the milk synthesis the mechanisms of injury to the parenchymatous tissue of the udder appears to becoming a bit clearer. It has been widely demonstrated that citrate is the “harbinger of lactogenesis” (Peaker and Linzel 1975). They further reported that the level of citrate in udder of cow, goat and women shoots up 46 times around parturition. These findings enthuse one to speculate that citrate is apparently playing a pivotal role in milk synthesis and might be associated with mastitis in dairy animals. It has been reported extensively that mastitic milk is significantly low in citrate (Oshima and Fuse 1981). Our investigations have also revealed that citrate levels are very low in milk of quarters affected with mastitis (33.71mg/ 100ml). A certain minimum concentration of citrate is essential for the normal synthesis of milk in the alveoli in the udder. Therefore, any in the citrate content would result in faulty synthesis of milk in a particular quarter(s) of the udder. We have observed that the affected quarters had very low concentration of citrate as compared with healthy quarters of the same animal (Dhillon and others 1989). The deficiency of citrate in a particular quarter may be due to nutritional, metabolic or some other intrinsic unknown factors which need further investigation.

Citrate, indeed, is the main constituent of the buffer system responsible for the maintenance of pH (~6.50) in the udder and regulates the homeostasis between Ca2+ and H+ ions and is the mainstay for the fluidity of milk through its effect on casein micelles (Faulkner and Peaker, 1982; Shennan and Peaker 2000). Citrate in udder also ensures the sequestration of soluble Ca2+ in milk (Kon and Cowie, 1961) and there is significant synchronization between the two (Holt and Muir, 1979). Hence, deficiency of citrate in udder would lead to the “clumping” of Ca2+ which manifest as flakes in the mastitic milk. These flakes of Ca2+ behave like lime and probably injure the parenchymatous tissue in the udder alveoli due to reduced moderator effect of citrate. Following this injury the impermeable barriers to citrate in both directions between blood and milk is disrupted and the inflammatory reaction sets in leading to an array of subsequent events. Such injuries due to free Ca2+ has been reported in myocardium (Fleckenstein and others 1974 ; Singal and others 1979). It has also been recorded that a calcium-dependant endonuclease is associated with necrotic type changes in tissues (Arends and others 1990). Furthermore, important ions e.g., bicarbonate, chloride, sodium etc., transudates from blood into milk during mastitis due to permeability of tight blood-milk barriers.

Thus, swapping of ions between blood and milk brings the pH of milk equal to that of blood or even higher and changes the pH of udder towards alkalinity i.e., 7.4 or higher. The lesions inflicted by free Ca+2 and most conducive alkaline milius in the udder prompts the environmental pathogens (commensals in the udder?) to invade and establish clinical/ subclinical “infectious mastitis”. Consequently pathogenesis is further exacerbated with the involvement of body defense mechanisms and severe inflammatory reaction ensues (Zhao and Lacasse 2007).

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Control and Treatment of Mastitis

Mastitis is a perpetual problem of all milk producing animals. The conservative estimates of economic losses from this malady have been made almost in each and every state world-wide. Several groups of scientists are working disparately to find out the exact cause and effective treatment of this most formidable disease. Though much of the work on intricate biochemical interactions at the molecular level directed towards unveiling the nub of this malady are being elucidated but still the problem appears elusive.

Coming down to the versatile dairy animal, buffalo (Bubalis bubalis), the “Asian Black Gold” a population of about 130 million globally suffer extensively from mastitis (Fagiolo and Lai 2007). Despite the use of best available facilities at hand to understand the pathobiology of mastitis, the problem still remain economically most important to the dairy industry throughout the world. The ideal modus operandi to prevent or reduce the economic losses, the definite cause of mastitis must be identified and then possible control measures implemented. While scanning the literature on mastitis and biosynthesis of milk in the udder it became apparent that citrate plays a very crucial role in the lactogenesis and maintenance of udder health through ionic equilibration (Peaker and Linzel 1975, Hyvonen and others 2010). Citrate level is always low and concomitant pH high in mastitic milk (Dhillon and others 1989). It was hypothesized that replenishment of citrate deficiency with extraneous tri-sodium citrate might play some protective role against mastitis, hence, these studies were undertaken and the results are communicated in this paper. Author : K. S. Dhillon, Ph.D and Jasmer Singh, Ph.D (College of Veterinary Science GADVASU)

PRESENT STANDARD TREATMENT

Treatment of mastitis with this salt has been standardized by enhanced doses to cut-short the recovery period (Dhillon and others 2007). The oral dose has been raised to 30gm in 250ml of water daily as a drench and recovery period cut-short to 3-5 days depending upon the severity of mastitis (Singh and others 2007). The disruption of the impermeable barrier between blood and milk in udder, as stated above, formed the basis of intravenous administration of tri-Sodium citrate. The dosage of this salt was standardized in cow-calves. Intravenous administration of medication directly reaches at the site of injury and normalizes the pH (~6.5) in the udder and the infectious agents are scavenged off and restoring ionic equilibrium. Moreover, the slightly acidic pH in normal udder is not suitable for generally isolated microbes from mastitic milk. With intravenous administration of tri-Sodium citrate in sterilized normal saline as 5% given morning and evening in 50ml doses the recovery period shortened to 1-3 days (Dhillon and Singh 2009, 2011). This treatment was safe, economical, very effective, avoided culling and discarding of milk with the minimal pain to the animal. Moreover, there is no withdrawal periods and hazards from residual problems in milk and meat. Presently, tri-Sodium citrate is employed extensively for the control of mastitis in dairy animals at farms with remarkable success. This treatment has also been found to be very effective in cases of mastitis refractory to antibiotics. On the basis of our investigations some pharmaceuticals have come-up with formulations intended for prevention and treatment of mastitis in dairy animals., Italy. 635

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Mastitis is the most costly dairy cattle problem to the industry. As mastitis problems increase, profits decrease. Reduced milk production accounts for approximately 3/4 of the total loss associated with mastitis. Without an effective mastitis control as much as 40% of the cows can be affected. The legal limit of SCC in milk is 750,000 cells per ml of milk. My specialists indicated that management practices to reduce the SCC from 400,000 to 200,000 cells/ml of milk will increase milk production by 1.5 lbs/day. Given a 100 cow herd this can account for 150 lbs of milk per day loss. At today's prices this amount of milk has a value of $24.75/day or $7,548/305 day milking for the reduction to 200,000 SCC. An estimated total loss of milk production with 400,000 SCC is 4.5 lb milk/day which is valued at $226 per cow per year or $22,600 per 100 cow dairy.

Wayne Greene, Ph.D., PAS, Diplomate ACAS Professor and Head Department of Animal Sciences Auburn University

EXAMPLE OF THE COST OF STANDARD MASTITIS TREATMENTS

Treatment statistics per lactation.

	Antibiotic and supportive	***Supportive Alone***
Average number of days with clinical mastitis	10	15
Average treatment cost	$49.16	$27.81
Average cost/day clinical mastitis	$4.78	$1.91
Average number of cases of clinical mastitis	1.21	1.16
Average 305-day milk yield loss (lb)	151.60	526.53
Milk loss due to unmarketable and unproduced milk (lb)	1221.37	526.53
Cost of lost marketable and unproduced milk	$144.34	$62.23
Total cost	$193.50	$90.04
Proportion of total clinical mastitis		
with severity score 1	0.86	0.70
with severity score 2	0.09	0.19
with severity score 3	0.05	0.11

Lactations that utilized antibiotics in addition to supportive therapy incurred $21.35 more in average treatment costs than lactations utilizing supportive therapy alone. Although total cost was greater in antibiotic lactations, the duration of the clinical mastitis episode was shorter. Lactations that utilized the antibiotics along with supportive therapy also had lower severity scores than supportive therapy alone. Cows treated with supportive therapy alone lost 374 pounds more milk than cows treated with both antibiotics and supportive therapy, but cows treated with antibiotics along with supportive therapy had twice the amount of marketable milk over supportive therapy alone due to use of antibiotics and the associated withdrawal period. Although the addition of antibiotics to supportive therapy decreased the magnitude of milk loss, the total cost was $103 more per lactation than use of supportive therapy alone.

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MASTCURE:

Is a micro-encapsulated form of enhanced trisodium citrate in a special matrix, administered as a feed additive for the prevention and treatment of MASTITIS, instead of the previously referred conventional treatment forms.

The matrix protects the active ingredients as it passes through the animal rumen and start to release the active ingredient as it enters the small intestine. The release is gradual and distal and is caused by the natural enzymes produced by the animal.

DOSAGE:

MASTCURE is dosed daily at 50 grs. Per head per day. The period of adlinistration should be 60 days prior to calving and shpuld go 305 days post calving

MASTCURE Physical Characteristic:

MASTCURE is a free flow white powder with fruity smell and a particle size of 700 to 900 microns.

BENEFITS:

In addition to milk loss prevention, administering MASTCURE in the feed will make it unecessary to handle the animal daily to adminsiter injections and/or drinking liquid preparation for treating MASTITIS.

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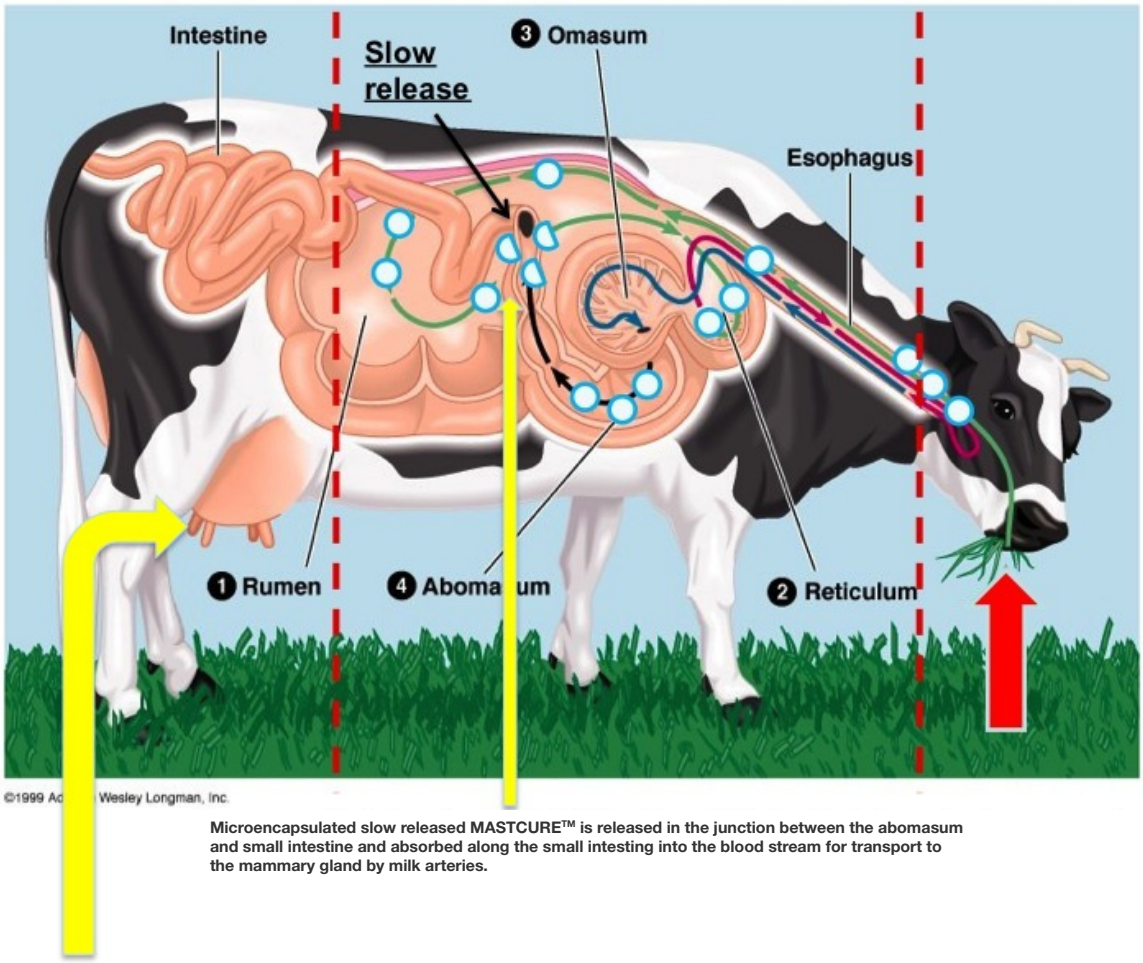

Microencapsulated slow released MASTCURE™ is released in the junction between the abomasum and small intestine and absorbed along the small intesting into the blood stream for transport to the mammary gland by milk arteries.

Once in the udder, the MASTCURE™ compound is disassociated releasing its two major components into the milk cistern to help regulate the pH of the udder towards a pH of 6.5. At pH of 7 or higher the alkalinity of the udder is such that it promotes the growth of harmful mastis pathogens such as Staphylococcus, Streptococci, E coli, and Kiebsiella.

A key component of MASTCURE™ maintains the pH of the milk at 6.5 which prevents the growth of mastic organisms and thereby decreases the incidence of clinical and subclinical mastis.

MASTCURE Mode of Action

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PACKAGING:

50 KG Drums with quality controled security on pallets as shown in picture below:

PICTURE OF 50 KG DRUMS

Appearance: Powder, white free fllowing

Is a micro-encapsulated form of enhanced trisodium citrate in a special matrix, administered as a feed additive for the prevention and treatment of MASTITIS, instead of the previously referred conventional treatment forms.

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