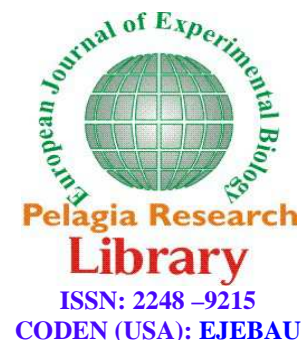




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Effect of vitamin D supplementation in treatment of naturally occurring mastitis in cattle

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ABSTRACT

The aim of this study was the investigation of effect of vitamin D₃ supplementation on antibiotic therapy of mastitis in cattle. The overall 14 head of dairy cow clinically infected with mastitis was divided into two control and treatment groups. In treatment group we used intramuscular vitamin D₃ supplementation in addition to antibiotic therapy. Measured indices were: rectal temperature, daily milk yield, somatic cell count, Californian mastitis test. Repeated measurements method was used for statistical analysis. The results showed that there was no significant difference between two groups ($P > 0.05$). Although in prior study, intra-mammary infusion of vitamin D₃ has therapeutic effect in experimental mastitis, intramuscular injection of this supplement couldn't improve the antibiotic therapy process of naturally occurring mastitis.

Key words: dairy cattle, mastitis, vitamin D₃

INTRODUCTION

The relationship between vitamin D status and efficiency of immune system to prevent disease is a topic of several studies in both human and veterinary medicine [1],[2]. Vitamin D, following its conversion to its active form 1, 25-dihydroxyvitamin D₃ (1,25(OH)₂D₃), is a primary regulator of calcium and skeletal homeostasis [1]. However, additional functions in the immune system became evident in the early 1980s when it was found that 1,25(OH)₂D₃ was produced by monocytes in diseased tissues. Indeed, the vitamin D receptor was identified in immune tissues and some immune functions were shown to be influenced by 1,25(OH)₂D₃[4],[5]. More than 80 years prior to the demonstration of the role of vitamin D in immune function, cod liver oil or exposure to sun, both sources of vitamin D, were used to treat tuberculosis (Reviewed in: [7],[15]. Then in 1986, Rook and co-workers showed that 1,25(OH)₂D₃ induced anti-tuberculosis activity in cultured monocytes [21]. Additionally, 1,25(OH)₂D₃ has been found to affect monocyte chemotaxis[10] and act as an adjuvant in the production of bacterial-specific antibodies [20]. In 2006, a seminal paper was published by Liu *et. al.*[14] in which they demonstrated that toll-like receptor (TLR) activation of monocytes induced 25-hydroxyvitamin D-1 α -hydroxylase (1 α -hydroxylase). 1-hydroxylase converts 25-hydroxyvitamin D₃ (25(OH)D₃) to the active 1,25(OH)₂D₃. 1,25(OH)₂D₃ induced the antimicrobial peptide cathelicidin and inhibited the growth of *Mycobacterium tuberculosis*. Furthermore, they showed that cathelicidin induction was compromised when using serum from donors with low 25(OH)D₃. This suggested that maintaining vitamin D status above that needed for normal calcium homeostasis was required for optimal immune responses.

Associations between serum 25(OH)D₃ concentrations and optimal immune function is now a subject of significant scrutiny. Levels of serum 25(OH)D₃ sufficient for full functionality of the immune system are thought to be higher than levels needed for proper skeletal formation [3],[12]. In humans only 20–25% of the population has 25(OH)D₃ levels considered immunologically sufficient (>30 ng/ml) [3],[8]. There is an inverse correlation between serum 25(OH)D₃ levels and the risk for upper respiratory tract infections [9], tuberculosis [19], and multiple sclerosis [16]. Dietary supplementation of vitamin D has been shown to decrease the risk of relapse in multiple sclerosis patients [6] and decreases the risk of influenza A infections [22]. Together this information indicates an important role of vitamin D in the clearance of infections and containment of inflammation by the body's immune cells.

Some evidences encourage the relation between serum vitamin D level with immune response to pathogens in dairy cattle. First, it is known that intra-mammary infections activate bovine macrophages found in the milk through the TLR pathways resulting in the up-regulation of the expression of the 1 α -hydroxylase gene. The expression of 1 α -hydroxylase is responsible for the conversion of 25(OH)D₃ to active hormone 1,25(OH)₂D₃[18]. The production of 1,25(OH)₂D₃ leads to changes in gene expression in macrophages isolated from milk of an infected gland [17]. Therefore, the intracrine pathway described in humans [14] is active in the bovine mammary gland macrophages during a bacterial infection, but fails to induce the induction of cathelicidin[18]. A second important aspect of studying the role of vitamin D in mammary gland infections, is that milk is deficient in 25(OH)D₃. The levels of 25(OH)D₃ in milk are only 0.3–0.6 ng/ml [11], thus immune cells are devoid of a source of 25(OH)D₃ after they enter the infected mammary gland. Intra mammary infusion of vitamin D could be beneficent in treatment of experimental mastitis. As in a study conducted by Lippolis *et al.* (2011) intra-mammary infusion of vitamin D₃boosted the mammary local immune system to combat the experimentally udder infection by *Streptococcus uberis*[13]. Data in this field of study is limited. Especially, the effect of intramuscular injection of vitamin D₃ was not clearly understood. So, the aim of this study was to evaluate the effect of intramuscular vitamin D₃ naturally occurring cases of mastitis.

MATERIALS AND METHODS

Animals

Fourteen mid-lactation non pregnant multi-parous, clinically mastitic Holstein cows selected and weretreatedwith10 ml of intra-mammary ceftriaxone injection at the end of completion of each milking period up to recovery. Cows randomly divided in two groups: the control group received no other supplementation but cows of treatment group were injected with intramuscular single dose of vitaminD₃ (1500 IU/KBW).Cows were feed a standard ration, which included between 30,000 and 40,000 IU of vitamin D per day and were milked three times a day.

Collection of milk, blood and temperature data

Milk samples of all cows were aseptically collected from alludders at each milking and overall daily milk production recorded. Somatic cell counted by EKOMILK Scan (Bulgarian company EON Tradig SCC machine manufacturer). To perform Californian mastitis test(CMT) 2ml of each quarter milk mixed on a CMT paddle with 2 ml of CMT solution(made in Germany).

After 20seconds, the coagulation of milk samples evaluated and scored qualitatively (trace, 1⁺, 2⁺& 3⁺).

Blood samples were taken by venipuncture of the tail vein before and after Injection vitamin D₃ supplementation. The levels of 25(OH)₂D₃ in the serum were measured by Chemiluminescencemethod (LIAISON made in UK).Rectal temperatures were obtained three times a day, at the time of milking.

Statistical analysis

Data were analyzed as a completely randomized design (SAS 9.1). Cow presumed as the experimental unit in the analysis of all data. Effects of treatments on variables (i.e. rectal temperatures, SCC, serum albumin, CMT, milk production) were analyzed with repeated-measures ANOVA. SCC values were log₁₀ transformed prior to analysis. Vitamin D as fixed effects and animal as a random effect was considered. The values presented for all variables as mean \pm SEM.

RESULTS AND DISCUSSION

The mean±SEM values of measured parameters are shown in table 1. The results showed no significant difference between two groups ($P>0.05$).

Table 1-The mean±SEM values of several parameters in control and treatment groups

Parameter	Treatment ± SEM	Control ± SEM
Rectal temperature (°C)	37.88 ±0.053	37.78 ±0.060
SCC (×1000/ml)	6.44 ±0.078	6.32 ±0.076
CMT	1.91 ±0.123	1.74 ±0.129
Milk Production (kg)	31.94 ±1.751	29.71 ±1.634
Calcium (mg/dl)	7.94 ±0.872	8.29 ±0.300
Phosphorus (mg/dl)	4.71 ±0.565	4.59 ±0.320
Serum Vit D ₃ (ng/ml)	40.09 ±2.565	50.49 ±4.559

Finding new strategies to reduce antibiotic use in clinically infections is an interesting field of study. Indeed, presence of antibiotic residues in animal food products, especially dairy food industry is a major challenge. Use of vitamin and mineral supplements and/ or herbal remedies in treatment of mastitis is one of the alternative methods. Lippolis *et al.* (2011) showed that the intra-mammary injection of vitamin D₃ -instead of intramammary antibiotic therapy – could boost the mammary local immune system to combat the experimentally udder infection by *Streptococcus uberis*[13]. The question is whether use of vitamin D₃ could improvenatural mastitis? Could we use general injection instead of intra-mammary infusion? Therefore, in this study the effect of intramuscular (IM) injection of vitamin D₃ in natural cases of mastitis were studied.

The IM injection of vitamin D₃ had not statistically significant effect on rectal temperature, SCC, CMT and milk production in both groups ($p>0.05$). This was contradictory to Lippolis *et al.* (2011) [13]. This phenomenon can occur because of the true agent of mastitis. In fact, different type of contagious and environmental bacterial agents isolated from the studied cases of naturally occurring mastitis samples. For example *Streptococcus* and *Staphylococcus* species can cause much more severe form of mastitis than *Streptococcus uberis* can do.

In other word, theoretically the effect of IM injection of vitamin D₃ overcomes the effect of intra-mammary infusion of this vitamin so we expected to see much more effect of IM injection but the results were controversy. Indeed, the virulent species of mastitis agents covered the minimally healing effect of injected vitamin D₃. Complementary studies could clear the point.

CONCLUSION

Despite the significant effect of vitamin D in the treatment of experimental mastitis, but the study did not show a positive effect of vitamin D therapy in general muscle. The reasons for this discrepancy could be the result of: a. Due to the experimental nature of the different types of mastitis. Differences in the virulence and pathogenicity of bacteria mastitis mastitis bacteria c. Treatment with vitamin D supplementation can be of local or limited. According to a new field of study in this regard, it is suggested further research be carried out for the above mentioned requirements and setting up a clearer understanding of the effect of vitamin D obtained.

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REFERENCES

- [1] Adams JS, Hewison M, *Nat ClinPractEndocrinolMetab*, **2008**, 4, 80–90.
- [2] Adams JS, Ren S, Liu PT, Chun RF, Lagishetty V, *et al.*, *J.Immunol*, **2009**, 182, 4289–4295.
- [3] Adams JS, Hewison M, *J.ClinEndocrinolMetab*, **2010**, 95, 471–478.
- [4] Barbour GL, Coburn JW, Slatopolsky E, Norman AW, Horst RL, *N. Engl. J. Med*, **1981**, 305, 440–443.
- [5] Bhalla AK, Amento EP, Clemens TL, Holick MF, Krane SM, *J.ClinEndocrinolMetab*, **1983**, 57, 1308–1310.
- [6] Burton JM, Kimball S, ViethR, Bar-Or A, Dosch H-M, *et al*, *Neurology*, **2010**, 74, 1852–1859.
- [7] Chocano-Bedoya P, Ronnenberg AG, *Nutrition Reviews*, **2009**, 67, 289–293.

- [8] Ginde AA, Liu MC, Camargo CA, *Arch. Intern. Med*, **2009**, 169, 626–632.
- [9] Ginde AA, Mansbach JM, Camargo CA, *Curr Allergy Asthma Rep*, **2009**, 9, 81–87.
- [10] Girasole G, Wang JM, Pedrazzoni M, Pioli G, Balotta C, et al., *J.Immunol*, **1990**, 145, 2459–2464.
- [11] Hollis BW, Roos BA, Draper HH, Lambert PW, *J.Nutr*, **1981**, 111, 1240–1248.
- [12] Hollis BW, *J.Nutr*, **2005**, 135, 317–322.
- [13] Lippolis JD, Reinhardt TA, Sacco RA, Nonnecke BJ, Nelson CD, *PLoS ONE*, **2011** 6(10): e25479.
- [14] Liu PT, Stenger S, Li H, Wenzel L, Tan BH, et al., *Science*, **2006**, 311, 1770–1773.
- [15] Liu PT, Modlin RL, *Current Opinion in Immunology*, **2008**, 20, 371–376.
- [16] Munger KL, Levin LI, Hollis BW, Howard NS, Ascherio A, *JAMA*, **2006**, 296, 2832–2838.
- [17] Nelson CD, Reinhardt TA, Beitz DC, Lippolis JD, *PLoS ONE*, **2010**, 5: e15469.
- [18] Nelson CD, Reinhardt TA, Thacker TC, Beitz DC, Lippolis JD, *J. Dairy.Sci*, **2010**, 93, 1041–1049.
- [19] Nnoaham KE, Clarke A, *Int. J.Epidemiol*, **2008**, 37, 113–119.
- [20] Reinhardt TA, Stabel JR, Goff JP, *J. Dairy.Sci*, **1999**, 82, 1904–1909.
- [21] Rook GA, Steele J, Fraher L, Barker S, Karmali R, et al., *Immunology*, **1986**, 57, 159–163.
- [22] Urashima M, Segawa T, Okazaki M, Kurihara M, Wada Y, et al., *Am. J.ClinNutr*, **2010**, 91, 1255–1260.