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Management of Sub-acute Ruminal Acidosis in Dairy Cattle for Improved Production: A Review

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Abstract

Sub-acute ruminal acidosis (SARA) is a well-recognized digestive disorder that is an increasing health problem in most dairy herds. Feeding diets high in grain and other highly fermentable carbohydrates to dairy cows increases milk production, but also increases the risk of SARA. Sub-acute ruminal acidosis is defined as periods of moderately depressed ruminal pH, from about 5.5 to 5.0. Sub-acute ruminal acidosis may be associated with laminitis and other health problems resulting in decreased production. Dairy herds experiencing SARA will have a decreased efficiency of milk production, impaired cow health and high rates of involuntary culling. Reduced ruminal efficiency, liver and lung abscesses, and laminitis are all thought to be related to SARA. The risk of developing SARA can be reduced by adopting a feeding regime, which balances ruminal buffering with the production of volatile fatty acids from fermentation of carbohydrates. Recommendations made for effective characterization, important management factors and good management practices of SARA in dairy cattle are further highlighted.

Keywords: Cattle; SARA; Rumen; Acidosis

Introduction

The average milk production per cow has increased dramatically over the last 20 years. Not only genetic improvement, but also changed feeding management as basis for optimal production has contributed to this development. Use of total mixed rations and increased energy supply are today's standard in successful dairy farming. Here, the proportion of energetic dense feed components is often maintained at the highest level possible to deliver proper energy supply for the high producing dairy cow.The complexcity of managing a large dairy herd and the tendency of cattle to prefer concentrate rather than structured components, however, does not always deliver the necessary intake of feedstuffs high in structure. This may lead to an imbalance of energy with the proportion of structure, a component of crucial importance for the ruminant digestive system (De Brabander *et al*., 2002).

The rise of acid in the rumen originating from carbohydrates and the decreasing buffering capacity of structured feeding components lead to an acidic ruminal environment. Already in the early 1960's, the tendency of increasing the carbohydrates providing necessary energy supply at the cost of roughage-components has been characterized as putting the animals at the edge between maximal productivity and illness due to acidosis (Dunlop, 1961). Although today modern dairy farming is based on research and feeding technology, it has to be expected, that non-acute forms of ruminal acidosis are indeed present in high-producing dairy herds. These forms are believed to contribute to milk fat depression (MFD), laminitis and other impairment of health status, finally leading to increased culling rates on herd-level (Oetzal, 2000). In the last years, this form of non-acute ruminal acidosis in dairy farms has been described as sub acute ruminal acidosis (SARA). Although it has been characterized in a number of publications, the real prevalence could only be estimated. A field study in the U.S. in the early 1990's revealed a SARA-prevalence of up to 26% in the whole study population of 15 dairy farms reaching as much as 40% on single farms (Garrett *et al*., 1997). This would furthermore enable researchers to establish the possible influence of SARA on milk production and evaluate the proposed diagnostic schemes made for the evaluation of SARA-prevalence.

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The physiological ruminal pH

A physiological pH depends on the physiologic morphology of the rumen as well as the type of ration fed to the animal. A ration consisting mostly of roughages thus leads to a higher pH mainly in the range from about 6.0 up to 6.8, whereas a diet high in concentrate leads to a pH range mainly from 6.0 down to about 5.5. Hence, a milieu dominated by cellulolytic bacteria suitable for roughage fermentation establishes a higher pH, on the other hand a lower pH range is maintained in an amylolytic milieu utilizing concentrates (Kaufmann, 1979; Dirksen, 1985, Owens and Goetsch, 1988; Dirksen, 1990). The mechanisms of these regulations are based on the management, the type, composition and quality of the ration, its physical structure and moisture, these factors for their part influencing taste and palatability, hence feed intake, chewing time and saliva production (Mackie and White, 1990; Hutjens *et al*., 1996). The pH of the ruminal environment is stabilized by different mechanisms, which have to be understood as an interaction between bases, acids and buffers (Owens *et al.*, 1996). In higher pH-ranges the main active component is the buffering by the HCO^-/CO_2 – system and the phosphate-buffer, both of which to a large extent are based on the saliva-flow (Owens *et al.,* 1996; Ivany et al. 2002). In lower pH-ranges the short-chain-fatty acids (SCFA) buffer (Gurtler, 1989; Hoover and Miller 1991; Owens *et al.*, 1996). It has also to be taken in account the buffering capacity of the feed stuffs themselves, determined by quality and processing, active through proteins which buffer at a higher pH-range, moreover the indirect buffering via mechanical induction of saliva flow (Church, 1979; Gabel, 1990; Mackie and White, 1990; Van Soest, 1986), the resorption of SCFA (Dirksen *et al*., 1984; Owens *et al*., 1996) and the transportation of rumen content to the omasum (Martens, 2000). The question, whether a reticulo-ruminal pH can be judged as physiological has therefore to be answered in context with the diet fed to the animals. For bovines fed solely on roughage, a pH ranging from around 6.4 up to 6.8 can be interpreted as physiologic (Pfeffer, 1987). In bovines fed on a high-concentrate diet a lower pH has to be expected. Most authors thus declare pH 5.5 being acceptable for an acidic milieu and at least non-pathologic (Owens and Goetsch, 1988; Leek, 1993), or explicitly being physiological (Dirksen, 1985; Martens, 2000). Garrett (1996) recommended a ruminal pH of ≤ 5.5 to be judged being abnormal while ≥ 5.8 should be viewed as normal.

Pathophysiology of the ruminal pH

The different forms of rumen acidosis have the initial pathways in common. The difference consists in the regulating mechanisms taking place, once a low ruminal pH is established (Oetzel, 2000). The development of a low ruminal pH, ranging from about pH 6.4 down to 5.5 and even lower, mostly starts with the intake of a diet high in energy. Easily digestible carbohydrates, like starch, sugar or pectin, provide this energy (Moller, 1993, Nordlund *et al*., 1995). Certain factors contribute to the development of an acidic ruminal environment: Decrease of saliva flow. Concentrates are usually given processed and thus easily taken up, forage components are often fed in small particle size. Chewing and ruminating time decrease, so does the flow of buffering saliva into the reticulo-ruminal compartment (Dirksen, 1985; Garrett, 1996). Change of microbial population. The change in substrates to ferment leads to a lower ruminal pH and a shift in the reticulo-ruminal flora.Growth of protozoa is restricted, so is growth of gram-negative bacteria (Slyter, 1976; Dawson and Allison,1988; Ivany *et al*., 2002). Decrease of buffering capacity from feedstuffs. Because more concentrates are taken in, less buffering capacity is supplied by ingested roughage. Concentrates do not buffer as forage does (Gabel, 1990; Van Soest, 1988; Owens *et al*., 1996; Garry, 2002). Increase of short chain fatty acids (SFCA)-production. The presence of rapidly fermentable substrates leads to a rising production of the SCFA thus reducing the ruminal pH. The pattern of SCFA changes in this process: The ratio of acetic towards butyric and propionic acid becomes narrow. Instead of the normal range, which can be seen at about 2:1. it changes towards round about 1.2:1 (Slyter, 1976; Moller, 1993; Enemark *et al*., 1998). Increase of lactate-production. The sudden presence of abundant energy leads to a slowly rising production of lactate by certain bacteria like Streptococcus bovis or protozoa like Dasytricha. These organisms are otherwise growth-restricted for they have to compete with those who use energy more efficiently and provide the substrates to form SCFA and lac-

tate. Both L-form as well as the D-form of Lactic acid are produced (Slyter, 1976; Dirksen, 1985; Dawson and Allison, 1988; Underwood, 1992) The low reticulo-ruminal pH is therefore caused by an absolute rise of the SCFA-production, combined with a relative fall of acetic acid, and an enlarging production of lactate. This rising of acids is insufficiently neutralized by the decreasing saliva flow and presence of feedstuffs with a low buffering capacity (Dirksen, 1985). These physiological mechanisms, which may on one hand lead to a reticulo-ruminal milieu ideal for the fermentation of the feedstuffs ingested, on the other extreme possibly to development of acute acidosis, open a fine-tuned, however unstable mechanism of action and counter-action: The rise of SCFA and lactate may be diminished by the metabolism of microorganisms and the resorption of the products by the rumen wall. Lactate is being used by certain bacteria, like Selenomonas ruminantium or Megasphera elsdenii to form acetic or propionic acid. Moreover, it is to a certain extent being absorbed by the ruminal wall and neutralized in the bloodstream. The SCFA are absorbed or metabolized by the ruminal wall as well as transported to omasum and abomasum by the normal rumen motility (Slyter, 1976; Dirksen *et al.*, 1984; Murphy, 1993; Martens, 2000). It has to be taken into account, however, that the rise in lactate goes on. Lactate-producing organisms are more resistant to the lower pH-environment and carry on in formation of lactate. This process can be accelerated due to the presence of free glucose in the rumen, which is used by lactate-forming bacteria. Moreover, the low pH developing may reduce the number and activity of lactate-using organisms (Slyter, 1976; Dirksen, 1985; Gabler, 1990). The absorption of SCFA by the ruminal wall is initially promoted by the more acidic medium. The negative effect of the low pH and high SCFA-level on rumen motility, however, impairs this process. Different mechanisms are suspected to cause this phenomenon (Underwood, 1992; Garry, 2000).

Patho-mechanism and definition of SARA

The onset of SARA is marked by the intake of a diet low in structure and high in energy, while the ruminal environment is not yet prepared to ferment it adequately while keeping the ruminal pH within those borders.The ruminal wall and its papillae play an important role herein. The adaptational growth has been described (Dirksen *et al*., 1984). The ruminal papillae are of crucial importance in the absorption of SCFA; the proliferation of the papillae is promoted by the SCFA arising from the fermentation. If the ruminal mucosa is not adapted, which is the case at the shift from a dry-period to a high-lactation diet, the papillae are too short, and this means that the resorbing surface is too small to deal with the sudden increase of SCFA (Nordlund *et al*., 1995). Also the bacterial population, which has to metabolise the lactic acid arising, is insufficiently developed (Slyter, 1976, Nordlund *et al*., 1995). Thus, in cases of SARA, these mechanisms cannot prevent a transient fall of ruminal pH in areas less than pH 5.5. Therefore some hours after intake of a concentrate-rich diet the ruminal-pH first reaches unphysiological acidic areas before returning to a higher, physiological level: The line of the ruminal-pH is characterized by subacute, intermittent acidosis. Generally spoken, SARA therefore has to be defined as an intermittent fall of ruminal pH to non-physiological areas after concentrate-uptake due to maladaptation of the ruminal environment in terms of ruminal microflora and ruminal mucosa. Sub-acute ruminal acidosis will have clinically detectable consequences which will become manifest after a certain delay to the initial insult. The critical pH-threshold of the ruminal pH can be identified at pH 5.5 some hours after concentrate feeding (Nordlund *et al*., 1995, Garrett *et al*., 1999, Oetzal, 2000).

Occurrence of SARA

As earlier defined, SARA has to be seen as a sequel of maladaptation in the ruminal environment to the uptake of diets high in concentrate. In the rumen basically the ruminal flora and the ruminal mucosa are elements of regulation concerning the ruminal pH, influenced by buffering capacity of the saliva-flow and the feedstuffs themselves. Sub-acute ruminal acidosis, therefore may occur if sufficient fibre is not provided to ensure a non-acidotic environment within the reticulo compartment or, on the other hand, the proportion of concentrates is too high and predisposes for the development of this condition. There are several ways in which a deficient fibre-intake or elevated concentrate-intake may occur.

The prevalence of SARA is so far not re-

searched intensively. Garrett *et al*. (1997) found in a survey of 15 Holstein herds that 19 % of the early-lactation cows and 26 % of the mid-lactation cows suffered from SARA. In one third of the herds even more than 40 % of the total number of cows within the herd were diagnosed to have SARA at the time of examination.

Sub-acute ruminal acidosis in the early post-partum period

In the period around calving dairy cattle suffers a considerable impact of stress. Calving process, onset of lactation, depressed feed intake and managerial changes lead to a situation of negative energy balance going along with loss of body condition and higher susceptibility to disease. Besides changes in housing and group of cows the change from dry-period diet to the (high) lacational diet puts cows at a high risk for developing SARA (Brand and Warner, 1996; Nocek, 1997). The ruminal status of the dry cow is adapted to fermentation of a diet, which is relatively low in energy and usually high in structure, compared to the lacational diet(s). Early lactation cows are at higher risk due to reduced absorptive capacity of rumen epithelium, poorly adapted rumen micro flora, and rapid introduction to high energy dense diets. The ruminal mucosa is therefore less developed. There is little microbiologic competence to ferment lactate (Nordlund *et al*., 1995). Therefore, instead of gradually being adapted to the high-energy- diet, the cows in the early post-partum-period may be confronted with a rapidly rising energy-content of the ration (Nocek, 1997).

Situations of occurrence of SARA

These two factors combined, a rumen non-adapted to concentrates, and a possibly high concentrate-uptake around parturition, may lead to development of SARA in the post-partum period. In any case, the arising of SARA within this period can usually be controlled by management, providing a slow transition period from dry-period to lactational period ration. This will ensure better adaptation of the reticulo-ruminal environment (Nordlund *et al*., 1995).

Sub-acute ruminal acidosis in mid-lactation

In mid-lactation the development of SARA is linked to managerial factors like feeding frequency, processing of feed and housing and similar influences. Su-acute ruminal acidosis may occur when the intake of easily fermentable concentrate feedstuffs meets a non-adapted ruminal environment. Because the rumen in mid-lactational cows is usually well-adapted to the uptake of concentrates as included in the diet, other factors are contributing to the occurrence here. Mistakes in automatic feeding or incorrect preparation of total mixed rations are some of the documented issues accidentally leading to this problem. In herds fed on a component-based diet, the rations not only may be too high in the concentrate component, but on the other hand also fibre-deficient (Nordlund *et al*., 1995; Garrett, 1996). Several reasons can lead to this undesired situation. First of all, the proportion of components may be miscalculated. There is chance that so the real weight of the forage, if only estimated by volume, does not meet the requirements due to falsely calculated dry-matter-base. This may result in an insufficient uptake of fibre or, inversely, a concentrate intake, which is relatively too high (Garrett, 1996). The time-schedule of feeding has a proven impact on ruminal pH (Yun and Han, 1989). Therefore a decision to feed smaller concentrate-meals more frequently will have a considerable impact on the arising of an acidotic ruminal environment. Also the time difference between concentrate and roughage feeding is of importance for regulation of the ruminal pH (Nordlund *et al*., 1995). The animals themselves may behave in such a way that they take up an imbalanced ration: In larger groups, with only limited access to the feedstuffs, the socially higher cows will eat first and longer, and, in case of component-feeding, take up relatively more concentrates and less fibre. Greediness of the animals housed in groups and sometime weather-changes are known to exacerbate this problem (Underwood, 1992; Nordlund *et al*., 1995). In TMR-fed herds an overmixing of the ration is documented (Garrett, 1996): Instead of letting the ration being mixed for some minutes, the mixing wagon may work up to an hour. This inevitably produces a diet very low in structure with high palatability. It will easily be taken up by the animals thus not providing the necessary buffering saliva-flow, giving less buffering-capacity of the feedstuffs within the reticulo-ruminal compartment. (Nordlund *et al.*, 1995; Garrett, 1996). The

fact that cattle tend to sort out concentrate even from a TMR has once again to be mentioned here (Leonardi and Armentato, 2003). Therefore it can be stated that SARA may occur in the mid-lactation also, in the phase of highest dry-matter-intake. More than in the early pre-partum period, it is almost exclusively related to management errors. Especially in high-producing dairy herds, where there is a narrow fibre to concentrate ratio, the impact on the remaining forage share may have severe consequences These factors may cause just a transient SARA-situation in the herd which usually will pass unnoticed. The depression of feed-intake going along with SARA, however, may cause clinically detectable acidosis, too (Garrett, 1996). Maekawa *et al*. (2002) reported that fresh animals are at an increased risk of developing SARA if component-fed compared with being fed a TMR. Due to the increased rate of consumption, less saliva is produced per unit of feed consumed when grain is fed separately from forages. Additionally, animals fed ingredients separately may consume all of the allotted grain and leave some of the forage. The net result is the consumption of a diet containing less forage than intended, increasing the risk of SARA.

Preventive Management of SARA

Two lines of applied SARA-prevention in dairy-herd management are visible. On one hand, the management of the different lactation-stage groups within the herd, on the other, the use of animal nutrition-guidelines applying to dairy cows. By both ways it is aimed at a proper building up of the ruminal mucosa after calving in order to ensure adaptation and resorptional capacity of SCFA.

Herd management

The adaptation of the ruminal mucosa to a concentrate-rich diet takes about four to six weeks to develop (Nordlund *et al*., 1995; Nocek, 1997). The bacteriological changes are said to take place within three weeks (Dirksen, 1985; Nordlund *et al*., 1995). This data deliver a guideline for the introduction of cows into lactation after the dry period or the first parturition. A gradual adaptation of dry cows to the lactational ration is necessary in order to ensure not to overtax the changes within the forestomach. In TMR-fed herds problems may arise in relatively small herds. In order to keep the labour preparing the rations low, the number of ration types may be reduced to one lactating and one dry cow ration. The switch from one type to another around partus is likely to overtax the ruminal adaptation resulting in SARA or even acute acidosis (Nordlund *et al.*, 1995). Especially in larger herds, a considerable factor is the personnel responsible for the feeding of the cows. Changes in the responsibility for feeding may lead to differences in feeding schedule, proportion of roughage and concentrate, having a considerable impact on an unstable ruminal environment. Although mainly described in feedlots (Elam, 1976), this also could apply to high-producing dairy herds and should therefore be regarded as a hazard in the herd health management. There are, of course, many other possibilities influencing the complex of adaptation and herd management. In any case, the veterinarian confronted with SARA suspected cows should be aware of this and analyse the herd management to define risks arising from here.

Feeding management

Best prevention of any fermentative disorder in the forestomach has to be seen in proper feeding management (Garry, 2000). Because SARA has to be viewed as a direct consequence from maximizing energy intake, a provision of components within the ration that are regulating physical and chemical components is desirable (Nocek, 1997). Therefore, in Germany for dairy cows a proportion from at least 18% crude fibre in dry matter is recommended, there from at least two-thirds being structured, not processed. This ensures the performance of a dairy herd, for the C2/C3 relation in ruminal fermentation pattern should at least be 2.0 with a butyrate proportion not greater than 15% (Dirksen, 1985). In the Netherlands and Belgium a system based on the structure value of feeding components is used since the 1990s (De Brabander *et al*., 2002). In component feeding, it should be taken care of the sequence of the components fed. It is recommended to have some roughage been fed before the concentrate component is given. This applies especially in the morning (Nordlund *et al*., 1995). In TMR using farms, a risk may arise from the overmixing of the ration. This may lead to a destruction of structure of the roughage components, predisposing the ration to lead to SARA. Moreover, a wrong estimation of the dry matter content of the

ration can lead to a miscalculation of roughage to concentrate ratio, giving way to the development of SARA due to higher concentrate intake as estimated (Nordlund *et al*., 1995).

Laminitis

One clinical sign regularly mentioned to be associated with SARA or subacute forms of
acidosis as well as ruminal acidosis in general is laminitis (Rossow, 1984; Dirksen, 1985; Gabler ,1990; Underwood, 1992; Nocek, 1997; Enemark *et al.* 1998; Garry, 2000; Oetzal, 2000; Ivany *et al*., 2002). There are little sound statistics about the prevalence of laminitis in dairy herds. Several predisposing factors and possibly causative agents have been defined. There is, however, still unclarity about the definitive etiology of bovine laminitis as well as about the link from acidosis to laminitis (Frankena *et al*., 1992; Lischer and Ossent, 1994; Nordlund *et al*., 1995). There is a strong influence of feeding regime on the prevalence of bovine laminitis and locomotive pathology in general, especially around parturition. Cattle fed on a diet high in concentrates are more prone to develop locomotive pathology (Livesey and Fleming, 1984; Manson and Leaver, 1988; Kelly and Leaver, 1990; Donovan *et al*., 2004). Also the amount and the frequency of the concentrate meals are having influence. The more concentrate in a short period is ingested, the more likely locomotive pathology is to develop (Bergsten, 1994). There is a certain relation to the body-condition of dairy cows and development of noninfectious laminitis. Gearhart *et al*., (1990) found cows being over conditioned at drying off were at greater risk to foot problems. However, no differentiation of the type of foot problems was made. Laminitis in SARA-affected cows is described as having a subacute and sometimes chronic character. Discoloration of the hoof, sole ulcers, abscesses and haemorrhages, misshapen hooves or double-walled soles are reported to occur in SARA-confirmed individuals (Nordlund *et al.*, 1995; Oetzal, 2000). Other factors related to the pathogenesis have also to be examined, for the pathogenesis of the laminitis still remains uncertain (Garrett, 1996). It is suspected that there are vasoactive substances entering the bloodstream from the rumen, leading to damage in the corium. The initial insult is thought to be metabolic in nature like a low ruminal pH. This allows a chain of pathological mechanisms to take place, eventually leading to ischemia of the distal limb and a clinically detectable form of laminitis, manifestating by blood imbibition of the sole during acute phases of the disease and classical picture of hoof deformation as the disease becomes chronic. Histamine, bacterial endotoxins and lactate are biological active agents suspected to interact in this complex (Lischer and Ossent, 1994; Nocek, 1997). Histamine has earlier been thought to be the main causative agent of bovine laminitis due to its vasodilatating activity (Dirksen, 1985). However, the idea is today that it plays a certain role in the pathogenesis but does not act solely. The fact that histamine is produced by decarboxilation of histidine in the rumen is long since known (Slyter, 1976). Bacterial endotoxins present in ruminal fluid also have been named as a possibly causative agent in the bovine laminitis complex. In an acidotic environment, the ruminal flora changes to a mainly gram-positive pattern. It has been shown that there is a detectable increase of endotoxins in the rumen, probably derived from the breakdown of the gram-negative bacteria (Aiumlamai *et al*., 1992; Andersen *et al*., 1993; Nocek, 1997). Lactate has been said to cause laminitis in sheep after being infused intraruminally. However, these effects could not be reproduced in cattle. In any case, the high levels of lactate reproduced and resorbed in ruminal acidosis may act as a co-agent in laminitis development (Lischer and Ossent, 1994; Dirksen, 1985). It strikes that these three substances (Histamine, Lactate, Endotoxins) are all products of an acidotic ruminal environment. The question remains how these substances are biologically active under circumstances as present in SARA, when there is a certain balance between adaptation and maladaptation providing stabilization of the ruminal environment. It has been proposed that the development of a non-acute laminitis in non-acute acidosis is the result of a repetitive metabolic insult (Dirksen, 1985). This fits to the idea of a ruminal pH, which drops in a certain period of time after the ingestion of concentrate. However, the initial mechanisms eventually leading to laminitis are yet to be identified. In any case, the correlation between concentrate-intake and the occurrence of locomotive pathology like laminitis supports the hypothesis of a common epidemiological background.

Milk fat depression

A depression of milk fat percentage in cows affected by SARA or generally non-acute forms of ruminal acidosis, respectively, has been documented (Dirksen, 1985, Nordlund *et al*., 1995, Chalupa *et al*., 2000; Oetzal, 2000). Because it usually occurs in individuals, the decrease of milk fat remains undetected in the bulk tank testing (Garrett 1996; Nocek, 1997). The fact that there are alterations in the ruminal fermentation patterns in SARA has been hold responsible by some authors for this depression (Rossow, 1984; Dirksen, 1985; Gabler, 1990). Also transient depression of the daily milk production has been reported in cases of SARA (Oetzal, 2000). The fact that the feeding largely influences the milk fat content is well known. The terms "low milk fat syndrome" and "milk fat depression" (MFD) are frequently used to describe a situation where there is a considerable depression in milk fat, largely due to mistakes in feeding strategy (Baumann *et al*., 2001). The following reasons for arising of low milk fat syndrome have been defined: 1) Feeding of a ration high in energy but deficient in roughage, 2) Feeding of processed roughage, e.g. by pelleting and 3) Supplementation of unsaturated fatty acids (Gurtler and Schweigert, 2000). A number of experiments showed the depression of milk fat being a sequel to a change in the ration.The increase of concentrates or the processing of roughage usually reduced milk fat content. In experiments the milk fat was decreased to 1.09–2.19% in specific situations (Van Breukelen *et al*., 1986). The milk fat depression was found being accompanied by a number of changes in the ruminal fermentation pattern. In one study the drop in milk fat was accompanied by an increase of milk yield and a body weight gain (Van Breukelen *et al*., 1985). Adding buffering substances to the high-concentrate-diet prevented the milk-fat content from dropping and re-established a higher ruminal pH, respectively (Rogers *et al*., 1982; Van Breukelen *et al*., 1985; Khorasani and Kennelly, 2001). It was concluded that the addition of buffering substances prevents the forming of trans-C 18:1 fatty acids that are suspected to inhibit the synthesis of milk fat in the mammary gland (Kennelly *et al*.,1999). There is no report about influence on milk-protein. There is a certain association between SARA and the reported milk-fat depression: Both arise in situations in which a diet high in concentrate, low in fibre or structured fibre, respectively, is fed to the dairy cows. The question remains, however, if milk-fat depression can be viewed as a symptom of SARA. Although the ruminal pH is reported to drop in experimental situations of inducing low milk-fat, this alone does not justify those situations to be characterized as being SARA. It seems justified to state that SARA may develop in the same situations where low milk fat syndrome is likely to occur, rather than to interpret milk fat depression being a sequel of SARA itself. It seems that milk fat depression also may occur in situations in which the adaptation of the ruminal flora prevents the development of SARA with its clinically detectable consequences.

Decreased DMI-Intake, loss of condition, ruminal hypomotility

In many papers related to the non-acute, non-clinical forms of acute ruminal acidosis, decrease of dry-matter-intake (DMI) is given as a consistent clinical sign, as a viable indicator of ruminal acidosis (Garrett, 1996; Garry, 2000; Stock ,2000). A Swedish study showed a lower feed-intake in dairy cows post-calving fed on a ration higher in concentrate, compared to cows fed on a low-concentrate-diet (Olsson *et al*., 1998). A recent study revealed a 25%-decrease in the intake of a TMR during SARA-periods induced, compared to normal. Moreover, the digestion of feedstuffs was generally impaired (Krajcarksi-Hunt *et al*., 2002).Many studies have been done on the feed intake in beef cattle with subacute acidosis.The reasons for the lower DMI have to be seen in weaker rumen motility, inhibited by certain mechanisms arising during low pH-phases within the rumino-reticular environment. It has been proposed that the high production of SCFA in ruminants fed on high-concentratediets leads to a reduction of rumen motility (Slyter, 1976; Furll *et al*., 1993). The idea that a metabolic acidosis leads to a decrease in the amplitude and frequency of ruminal contractions has been ruled out (Crichlow and Chaplin, 1985). Bacterial endotoxins have been related to the decrease of rumen motility. The principles of rumen hypomotility involving toxaemia in cases of coliform mastitis have been well described (Verheiden *et al*., 1981; Hoeben *et al*., 2000). A mechanism of inducing ruminal hypomotility has been related to endotoxaemia (Eades, 1997)..Another explanation for the

decrease of DMI has been given by Oetzal (2000). The described increase of osmolarity of ruminal content due to reduced absorption and increase of osmolarly active substances like glucose, SCFA or lactate leads to a flow of fluid into the rumen which in turn reduces the feed intake of the animal affected. A low body-condition has usually been associated with SARA (Nordlund *et al*., 1995; Nocek, 1997; Oetzal, 2000), on the other hand, some authors characterize animals affected by "chronic acidosis" as become obese due to a narrow C2/C3 relation in ruminal fermentation and associate it with fat-cow-syndrome (Dirksen, 1985; Gabler, 1990). This difference in description is the more apparent, as the other signs are characterized similarly by all authors. The question remains whether it is in fact dealt with different diseases or just stages of one disease. In beef-cattle, the narrow C2/C3 relation is to a certain extent desired in order to maintain the necessary body weight gain, but it has to be managed carefully for it is an unstable situation which may affect the DMI, negatively influencing the body weight (Stock, 2000). The same situation may arise in dairy cattle, in which, however, the narrow C2/C3 pattern leads to a milk-fat depression and an increase of body condition (Dirksen, 1985). The decrease in DMI described must not be confused with the decrease and the relatively lower increase of DMI in ongoing lactation of feed intake as associated with negative energy balance (NEB). This is reported to be the more dramatic the more the animals tend to be overfed in dry-period and therefore are in severer NEB (Rukkwamsuk, 1999). However,certain interaction seems to be possible. Both complexes, NEB and SARA, are associated with decrease of DMI and related to the post-partum-period. It could be thought of an interaction via the DMI exaggerating the one or the other problem. There is however, no literature available, especially because in both complexes yet it has to be researched. It is obvious that the described increase of body condition in dairy cows experiencing acute acidosis applies to the late-lactation and dry period, for an increase will rarely be achieved in the early lactational stadium. This overcondition of the animals later may exaggerate negative-energy balance and lead to the fat-cow syndrome. It appears also likely that those animals may develop a subacute acidosis leading to a further decrease in body condition as found at reduction of DMI and other factors. Therefore it appears to maybe useful differentiating a type of subacute acidosis, maybe better characterized as chronic, mainly in beef-cattle and late-lactation cows promoting a gain in body-condition from the subacute ruminal acidosis leading to the described loss in body condition. . Reasons for poor body-condition may be chronic inflammation (Oetzal 2000), leading to an impaired health status of the animal affected or the described decrease of DMI. A farmer may try to correct the possibly occurring loss of body condition, originating from SARA, by increasing the energy level of the ration, thus exaggerating the basic acidosis problem (Nordlund et al. 1995).

Alterations in faeces, diarrhea

The fact that faeces of cattle, affected by acute as well as subacute ruminal acidosis, change is well described. (Rossow 1984, Dirksen 1985, Nordlund et al. 1995, Garry 2000, Oetzal 2000). The structure and consistency of the faeces depend on rumination, activity of the ruminal flora and ruminal passage (Garry, 2000). The changes are described as alterations in colour, which appears brighter and yellowish. The pH of the faeces is lower than normal, usually slightly acidic (Dirksen, 1985). The smell of the faeces is said to be sweet-sour (Oetzal, 2000). The size of ingesta particles may be too large, being around 1 – 2 cm instead of less than 0.5 cm. Whole cereal grains may be present. The alterations are usually transient in nature (Garry, 2000). One explanation for this phenomenon is post-ruminal fermentation in the intestines due to a massive outflow of fermentable carbohydrates from the rumen (Oetzal, 2000). Another explanation could be the high osmolarity, which is described for the ingesta in SARA-affected animals, which could lead to soft faeces, due to binding of fluid in the intestinal lumen (Garry, 2000). Generally speaking, the impaired ruminal function as mentioned above in terms of rumination, bacterial breakdown and passage, leads to the alteration in faecal aspects. Kleen *et al*., (2003) reported that in SARA cases faces are bright, yellowish, have a sweet sour smell, appear foamy with gas bubbles, and contain more than normal amounts of undigested fibre or grain (Hall, 2002). Nordlund *et al*. (1995) reported on herds with loose faeces that contained substantial amounts of undigested feed particles. Intermittent diarrhoea and the presence

of undigested particles indicate inadequate digestion and fast passage of feed.

Use of buffers

Buffers are well advocated in therapy of acute ruminal acidosis, and they may also be beneficial in prevention against any form of acidosis (Rossow, 1984; Garry, 2000). Their use has been reviewed (Garry and Kallfelz, 1983) and, recently, a new technique, using a rumen model for evaluation of effect has been introduced (Zamarreno *et al*., 2003). For purpose of regulating the ruminal acidity, the use of bicarbonates seems to be suitable, for it can be considered as a replacement of the buffer provided by saliva-flow. In contrast, the benefit of hydroxides is said to be poor (Garry and Kallfelz, 1983). The osmotic effect, which is provided by sodium bentonit salt, may lead to a fluid flow into the reticulo-ruminal compartment, supporting a better turnover of the ingesta-flow. Moreover, some of the substances are said to have a corrective effect on the milk fat depression caused by low ruminal pH (Garry and Kallfelz ,1983; Rossow, 1984). However, there still is a lack of scientific evidence. The processing of feedstuffs with NaOH, done to increase palatability, also may contribute in rumen pH-stabilization (Dirksen, 1985). Generally, in a situation in which feeding high proportions of concentrate may cause a pH depression in the reticulo-ruminal compartment, the preventive use of buffers, especially bicarbonate, may prevent an overgrowth of acid-tolerant lactobacilli, thus preventing a further decrease (Garry, 2000). Downer and Cumming (1985) reported that addition of 150 g of sodium bicarbonate to the lactation feed per day had a positive effect on milk yield. Similarly a positive effect has been demonstrated on feed intake and milk fat percentage (Erdman, 1988). Hutjens (1991) reported that normally a single buffer is used but combination of several buffers are possible with a documented positive influence on milk yield, fat % and dry matter intake.

Ruminal pH and sub-clinical acidosis

Krajcarski-Hunt *et al*. (2002) found that induction of sub-clinical ruminal acidosis significantly reduced the mean daily rumen pH from 6.36 to 5.72, increased daily time below pH 6.0 from 155 to 938 minutes per day and increased the time where the rumen pH was <5.6 from 15 to 594 minutes per day. The risk of acidosis is increased when cattle are fed forages high in non-structural carbohydrates (sugars and starches) with low effective fibre content, such as clovers and young lucerne (alfalfa) and possibly ryegrass that may not stimulate adequate rumination and salivation, especially when fed in combination with concentrate feeding.

Clinical diagnosis of acidosis severity uses ruminal pH thresholds of 5.8 or less for SARA and 5.2 or less for acute acidosis (Cooper and Klopfenstein, 1996; Owens et al., 1998; Ghorbani et al., 2002). However, ruminal pH thresholds should be used as general guidelines as the actual ruminal pH where SARA becomes acute is difficult to determine as cattle vary in their ability to cope with dietary changes that induce acidosis and characterization of acidosis severity should be based more on symptoms (Stock and Britton, 1993, Schwartzkopf et al. 2004). Accumulation of VFA in the rumen, rather than lactic acid, is responsible for the decline in ruminal pH during SARA. Lactic acid is present in the rumen at very low concentrations during SARA (Mackie and Gilchrist, 1979; Britton and Stock, 1986; Owens et al., 1998; Ghorbani et al. 2002). Ghorbani et al. (2002) summarized ruminal pH data of steers fed a barley diet using pH thresholds of 5.8 (mild SARA) and 5.5 (severe SARA). et al. (2004) considered SARA to exist when ruminal pH fell below 5.8 for greater than a total of 12 h/d. Plaizer et al. (1999) reported dairy cows experienced SARA when ruminal pH was reduced below pH 6.0 and 5.6 for a substantial part of the day (5 and 1 h, respectively).

Blood pH and sub clinical acidosis:

Changes in systemic acid-base status are minimal during SARA (Horn *et al*., 1979; Goad *et al*., 1998; Brown *et al*., 2000). However, when ruminal lactate accumulation occurs, there is an elevated risk of metabolic acidosis whereby plasma lactate concentrations rise and blood pH is lowered (< 7.35) (Telle and Preston, 1971; Burrin and Britton 1986; Krehbiel *et al*., 1995; Owens *et al*., 1998; Brown *et al*., 2000). However, SARA can still lead to tissue damage, unstable microbial and bacterial populations and reduced protozoal numbers (Huntington, 1988). Prolonged effects can occur from one single bout of acidosis, attributing to cattle being “chronics” or “poor-doers”, where they have reduced feed intake, weight gain and feed ef-

ficiency (Huntington, 1988; Stock and Britton, 1993; Krehbiel *et al*., 1995). Clinical diagnosis of metabolic acidosis relies on blood pH levels equal to or less than 7.35 (Owens *et al*., 1998). Changes in systemic acid-base status have been reported to be minimal during SARA because the ruminal pH eventually recovers during the day and homeostasis of blood is maintained (Burrin and Britton, 1986; Goad *et al*., 1998; Brown *et al*., 2000).

Diaganosis

The definition of sub-clinical acidosis is controversial, with some authors suggesting that a pH of 5.5 detected by rumenocentesis be used as a cutpoint for detecting the disorder (Garrett *et al*., 1999). However, Bramley *et al*. (2007) used an approach of categorising cows on VFA, lactic acid and ammonia concentrations, and pH. One group of cows identified this way was consistent with acidosis, being characterised by high concentrations of valerate and propionate and low concentrations of ammonia. While the pH was low and the lactic acid concentrations high for this group, these were the least discriminatory variables. Using pH as the sole measure to detect acidotic cows was neither highly sensitive nor specific. Improved diagnostic measures to confirm diagnoses made on clinical signs need to be developed.

The current recommendations are to have your veterinarian perform rumenocentesis (sample rumen with a needle) on 12 animals per feeding group. Subacute acidosis is diagnosed if three or more have a pH of 5.5 or less. Samples should be collected at 4-8 hours after a TMR meal or 2-4 hours after the concentrate portion of a component-fed ration. Qualitative evaluation of manure can also be helpful.

Future thrust:

1.The understanding of the principles of ruminal acidosis, especially in its subclinical form requires certain clarity about the physiological conditions within the reticulo-ruminal compartment. The fermentation processes in the reticulo-ruminal complex of ruminants have attracted many researchers. Many techniques have been developed in order to gain insight into to the dynamic biochemical processes taking place in the fore stomach of ruminants (Van Soest *et al*., 1988). Many papers have thus been written related to the ruminal pH, although the value of the results is doubted by some researchers, because the individual circumstances and influences on the animal observed have always to be taken into account. These factors are e.g. the cow health status, the diets fed and feeding regime, diurnal variation or the different regions of the reticulo-ruminal complex where the samples are taken from. It has to be pointed out that the purpose of the biologically active and constantly altering ruminal environment is not to maintain a steady pH, but to develop circumstances most suitable for the digestion of a certain diet.

2.The sub acute ruminal acidosis (SARA) is believed to play an important role in high producing dairy farming. More emphasis should be given to low milk fat, low BCS, moreover laminitis and other kinds of pathology leading to high-culling rate on herd-level as it is directly linked to SARA. Although in particular in high producing dairy herds a high proportion of highly fermentable carbohydrates in the diet has to be expected in order to meet energy demand, that results in SARA.

3.SARA does occur also in relatively low producing herds, indicating that poor feeding management obviously is one of the key factors responsible for SARA. SARA has to be expected in situations of severe negative energy balance and might be traced in herds with low body condition scores and heavy condition loss after calving. Milk fat depression is observed in herds showing SARA, thus is also to be considered being present in herds with apparently normal fat percentage. Ruminocentesis proved to be a valuable diagnostic tool in diagnosing SARA

References

Aiumlamai, S., Kindhal, H., Fredriksson, G., Edqvist, L.E., Kulander, L., Eriksson, O., 1992. The role of endotoxins in induced ruminal acidosis in calves. Acta Veterinaria Scandinavica 33, 117–127.

Andersen, P.H., Bergelin, B., Chirstensen, K.A., 1993. Effect of feeding regimen on concentration of free endotoxin in ruminal fluid of cattle. Journal of Animal Science 72, 487–491.

Baumann, D.E., Grinari, J.M., Baldi, A., Stelwagen, K., 2001. Regulation and nutritional manipulation of milk fat: Low milk-fat syndrome. Livestock Production Science 70,

15–29.

Bergsten, C., 1994. Haemorrhages of the sole horn of dairy cows as a retrospective indicator of laminitis. An epidemiological study. Acta Veterinaria Scandinavica 35, 55–66.

Bramley, E., Lean, I.J., Costa, N.D., 2007. Clinical acidosis in a Gippsland dairy herd during grain-induced subacute ruminal acidosis in dairy cows. Bovine Practioner 90 , 856-866.

Brand, A., Warner, M., 1996. Monitoring reproductive performance: Execution. In: Brand, A., Noordhuizen, J.P.T.M., Schukken Y.H., eds., Herd Health and Production Management in Dairy Practice Wageningen Pers, Wageningen, pp. 293–312.

Brown, M.S., Krehbiel, C.R., Galyean, M.L., Remmenga, M.D., Peters, J.P., Hibbard, B., Robinson, J., Moseley, W.M., 2000. Evaluation of models of acute and subacute acidosis on dry matter intake, ruminal fermentation, blood chemistry, and endocrine profiles of beef steers. Journal of Animal Science 78, 3155-3168.

Burrin, D.G., Britton, R.A., 1986. Response to monensin in cattle during subacute acidosis. Journal of Animal Science 63, 888-893.

Chalupa, W., Sniffen, C.J., Stone, G.M., 2000. Balancing rations for milk components. Asian-Australasian Journal of Animal Science 13, Suppl. 388 – 396.

Cooper, R., Klopfenstein, T., 1996. Effect of rumensin and feed intake variation on ruminal pH. Scientific update of on rumensin/ tylan/ micotil for the professional feedlot consultant. Elanco animal health, Greenfield, IN. A1-A14.

Crichlow, E.C., Chaplin, R.K., 1985. Ruminal lactic acidosis: Relationship of forestomach motility to non-dissociated VFA-levels. American Journal of Veterinary Research 46, 1908–1911.

Dawson, K.A., Allison, M.J., 1988. Digestive disorders and nutritional toxicity. In: Hobson, P.N. ed.. The rumen microbial ecosystem. Elsevier Science, Barking, pp. 445 – 459.

De Brabander, D.L., De Boever, J.L., Vanacker, J.M., Geerts, N.E., 2002. Evaluation and effects of physical structure in dairy cattle nutrition. In: Kaske, M., Scholz, H., Holtershinken, M., eds.. Recent developments and perspectives in bovine medicine, Keynote lectures from the XXII World Buiatrics Congress, Hannover, pp. 182–197.

Dirksen, G., 1985. Der Pansenazidose-Komplex – Neuere Erkenntnisse und Erfahrungen. Tierärztl. Prax. 13, 501–512.

Dirksen, G., 1990. Erkrankungen desVerdauungsapparates. in: Rosenberger, G. ed., Die klinische Untersuchung des Rindes. Verlag Parey, Berlin and Hamburg, pp. 288–400.

Dirksen, G., Liebich, H.G., Mayer, W., 1985. Adaptive changes of the ruminal mucosa and their functional and clinical significance. Bovine Practitioner 20:116–120.

Dirksen, G., Liebich, H.G., Brosi, G., Hagemeister, H., Mayer, E., 1984. Morphologie der Pansenschleimhaut und Fettsäureresorption beim Rind – bedeutende Faktoren für Gesundheit und Leistung. Journal of Veterinary Medicine Series A 31, 414–430.

Donovan, G. A.,. Risco, C.A., De Chant Temple, G.M., Tran, T.Q., Van Horn, H.H., 2004. Influence of transition diets on occurrence of subclinical laminitis in Holstein dairy cows. Journal of Dairy Science 87, 73-84.

Downer, J.V., Cumming, K.R., 1985. A ten year review of lactation study. Journal of Dairy Science 68, 191-201.

Dunlop, R.H., 1961. A study of factors related to the functional impairment resulting from loading the rumen of cattle with high carbohydrate feeds. University of Minnesota, St. Paul, PhD

Eades, S.C., 1997. Endotoxaemia in dairy cattle: mechanism of reticulo-ruminal stasis. The Veterinary Journal 153, 321–327.

Elam, C.J., 1976. Acidosis in feedlot cattle: Practical observations. Journal of Animal Science 43, 898–901.

Enamark, J.M.D., Jorgenssen, R.S., Enemark, P.S., 1998. Rumen acidosis, with special emphasis on diagnostic aspects in subclinical rumen acidosis. Dan. Veterinaertidsskri. 81, 886–897.

Erdman, R. A., 1988. Dietary buffering requirements of the lactating dairy cow: a review. Journal of Dairy Science 71, 32-46.

Frankena, K., Van Keulen, K.A.S., Noordhuizen, J.P.T.M., Noordhuizenstrassen, E.N., Gundelach, J., De Jong, D.J., Saedt, I., 1992. A cross sectional study into prevalence and risk indicators of digital haemorrhages in female

dairy calves. Preventive Veterinary Medicine 14, 1–12.

Furll, M., Schafer, M., Dabbagh, M.N., 1993. Einfluß subakuter Buttersäurebelastung aauf die Pansenfunktion bei Rindern unterschiedlichen Alters. Mh Veterinary Medicine 48, 189–195.

Gabler, G., 1990. Pansenazidose: Interaktionen zwischen den Veränderungen im Lumen und in der Wand des Pansens Übers Tierernährg 18, 1–38.

Garret, E., 1996. Sub acute rumen acidosis- clinical signs and diaganosis in dairy herds. Large Animal Veterinarian 11, 6-10.

Garret, E.F., Nordlund, K.V., Goodger, W.J., Oetzel, G.R., 1997. A cross sectional field study investigating the effect of periparturient dietary management of ruminal pH in early lactation dairy cows. Journal of Dairy Science 80, 169

Garrett, E.F., Perreira, M.N., Nordlund, K.V., Armentano, L.E., Goodger, W.J., Oetzal, G.R. , 1999. Diagnostic methods for the detection of subacute ruminal acidosis in dairy cows. Journal of Dairy Science 82, 1170–1178.

Garry, F.B., 2002. Indigestion in ruminants. In: SMITH B.P., ed.. Large Animal Internal Medicine, 2nd ed. Mosby, St. Louis and Baltimore, pp.722–747.

Garry, F.B., Kallfelz, F.A., 1983. Clinical aspects of dietary buffers for dairy cattle. Compend. Contin. Educ. Pract. Vet. 5, 159–166.

Gearhart, M.A., Curtis, C.R., Erb, H.N., Smith, R.D., Sniffen, C.J., Chase, L.E., Cooper, M.D., 1990. Relationship of changes in condition score to cow health in Holsteins. Journal of Dairy Science 73, 3132–3140.

Ghorbani, G. R., Morgavi, D.P., Beauchemin, K.A., Leedle, J.A.Z., 2002. Effects of bacterial direct-fed microbials on ruminal fermentation, blood variables, and the microbial population of feedlot cattle. Journal of Animal Science 80, 1977-1986.

Goad, D. W., Goad, C. L., Nagaraja, T.G., 1998. Ruminal microbial and fermentative changes associated with experimentally induced subacute acidosis in steers. Journal of Animal Science 76, 234–241.

Gurtler, H., Schweigert, F.J., 2000. Physiologie der Laktation. In: Engelhardt W., Breves, G. eds., Physiologie der Haustiere Enke-Verlag, Stuttgart, pp. 572–593

Gurtler, H., 1989. Die Physiologie der Verdauung und Resorption – Die Verdauung des Wiederkäuermagens In: Kolb H., ed., Lehrbuch der Physiologie der Haustiere, Band 15th Ed. Verlag Fischer, Jena, pp. 234–297.

Hall, M.B.,, 2002. Characteristics of manure. In: Proceedings of the tristate dairy nutrition conference, Fort Wayne, Indiana, US, pp.141-147.

Hoeben, D., Burvenich, C., Trevisi, E., Bertoni, G., Hamann, J., Buckmaier, R., Blum, J.W., 2000. Role of endotoxins and TNF-alpha in the pathogenesis of experimentally induced coliform mastitis in periparturient cows. Journal of Dairy Science 67, 503–514.

Hoover, W.H., Miller, T.K., 1991. Rumen digestive physiology and microbial ecology. The Veterinary clinics of North America Large animal practice 7, 311-325.

Horn, G.W., Gordon, J.L., Prigge E.C., Owens F.N., 1979. Dietary buffers and ruminal and blood parameters of subclinical lactic acidosis in steers. Journal of Animal Science 48, 683-691.

Huntington, G. B., 1988. Acidosis. In: The ruminant animal – digestive physiology and nutrition. Editor D.C. Church, Prentice Hall, Englewood Cliffs, New Jersey, pp. 474-480.

Hutgens, M.F., Overton, R., Brand, A., 1996. Monitoring milk production: Optimizing rumen digestion in the dairy cow. In: Brand A., Noordhuizen J.P.T.M., Schukken Y.H., eds., Herd Health and Production Management in Dairy Practice, pp. 203–222.

Hutjens, M.E., 1991. Feed additives. Vetrinary clinics of North America. Food Animal Practice 7, 525-540.

Ivany, J.M., Rings, D.M., Anderson, D.E., 2002. Reticuloruminal disturbances in the bovine. The Bovine Practitioner 36, 56–64.

Kaufmann, W., 1979. Influence of the composition of the ration and the feeding frequency on pH-regulation in the rumen and on feed-intake in ruminants. Livestock Production Science 3, 103-114.

Kelly, E.F., Leaver, J.D., 1990. Lameness in dairy cattle and the type of concentrate given. Journal of Animal Production 51, 222–227.

Kennelly, J.J., Robinson, B., Khorasani, G.R.,

1999. Influence of carbohydrate source and buffer on rumen fermentation characteristics, milk yield and milk composition in early lactation Holstein cows. Journal of Dairy Science 82, 2486–2496.

Khorasani, G.R., Kennelly, J.J., 2001. Influence of carbohydrate source and buffer on rumen fermentation characteristics, milk yield, and milk composition in late-lactate Holstein cows. Journal of Dairy Science 84, 1707–1716

Kleen, J.L., Hooijer, G.A., Rehage, J., Noordhuizen, J.P.T.M., 2003. Sub acute ruminal acidosis , SARA. a review. Journal of veterinary medicine A. 50, 406-414.

Krajcarski-Hunt, H., Plaizer, J.C., Walton, J.P., Spratt, R., McBride, B.W., 2002. Effect of subacute ruminal acidosis on in situ fibre digestion in lactating dairy cows. Journal of Dairy Science 85, 570–573.

Krehbiel, C.R., Britton, R.A., Harmon, D.L., Wester, T.J., Stock, R. A., 1995. The effects of ruminal acidosis on volatile fatty acid absorption and plasma activities of pancreatic enzymes in lambs. Journal of Animal Science 73, 3111-3121.

Leek, B.F., 1993. Clinical diseases in the rumen. Veterinary Record 113, 10–14.

Leonardi, C., Armentano, W.J., 2003. Effect of quantity, quality, and length of alfalfa hay on selective consumption by dairy cows Journal of Dairy Science 86, 557-564.

Lischer, C., Ossent, P., 1994. Klauenrehe beim Rind: Eine Literaturübersicht Tierärztl Prax 22, 424–423.

Livesey, C.T., Fleming, F.L., 1984. Nutritional influences on laminitis, sole ulcer and bruised sole in Friesian cows. Veterinary Record 114, 510–512.

Mackie, R.I., White, B.A., 1989. Recent advances in rumen microbial ecology and metabolism Potential impact on nutritient output Journal of Dairy Science 73, 2971-2995.

Maekawa, M., Beachemin, K.A., Christensen, D.A., 2002. Effect of concentrate level and feeding management on chewing activities, saliva production and ruminal pH of lactating dairy cows. Journal of Dairy Science 85, 1165-1175.

Manson, F.J., Leaver, J.D., 1988. The influence of concentrate amount on locomotion and clinical lameness in dairy cattle. Animal Production 47, 185–190.

Martens, H. , 2000. Funktionen der Vormägen. In: Engelhardt W.V., Breves G. eds., Physiologie der Haustiere Enke-Verlag, Stuttgart 344 – 362.

Moller, P.D., 1993. Acidosis in dairy cows. Acta Veterinaria Scandinavica 89, 111–112.

Murphy, M., 1993. The pH fluctuation in the rumen of lactating cows. Acta Veterinaria Scandinavica 89, 109–110.

Nocek, J.E., 1997. Bovine Acidosis: Implications on laminitis. Journal of Dairy Science 80, 1005–1028.

Nordlund, K.V., Garrett, E.F., Oetzal, G.R., 1995. Herd-based rumenocentesis - a clinical approach to the diagnosis of subacute rumen acidosis. Compendium Contin Educ Pract Vet 17, 48–56.

Oetzel, G. R., 2000. Clinical aspects of ruminal acidosis in dairy cattle. Proceedings of the Thirty-Third Annual Conference, American Association of Bovine Practitioners, Rapid City, SD. pp 46–53

Olsson, G., Bergsten, C., Wiktorsson, H., 1998. The influence of diet before and after calving on the food intake, production and health of primiparous cows, with special reference to sole haemorrhages. Journal of Animal Science 66, 75–86.

Owens, F.N., Secrist, D.S., Hill, W.J., Gill, D.R., 1998. Acidosis in cattle: a review. Journal of Animal Science 76, 275-286.

Owens, F.N., Goetsch, A.L., 1988. Ruminal fermentation. In: Church D.C., ed., The Ruminant animal Verlag Prentice Hall , Englewood Cliffs, pp. 145–171.

Owens, F.N., Secrist, D.S., Hill, W.J., Gill, D.R., 1996. Acidosis in cattle: A review. Journal of Animal Science 76, 275–286.

Pfeffer, E., 1987. Verdauung. In: A. Scheunert and A. Trautmann, eds.. Lehrbuch der Veterinär-Physiologie 7. Auflg. Verlag Parey, Berlin u. Hamburg, 29–57.

Plaizer, J.C., Martin, A., Duffield, T., Bagg, R., Dick, P., McBride, B.W., 1999. Monitoring acidosis in the transition cow. Journal of Dairy Science 82, 110

Rogers, J.A., Davies, C.L., Clark, J.H., 1982. Alteration of rumen fermentation, milk fat synthesis, and nutrient utilization with mineral

salts in dairy cows. Journal of Dairy Science 65, 577-586.

Rossow, N., 1984. Erkrankungen der Vormägen und des Labmagens. In: Rossow, N. ed., Innere Krankheiten der landwirtschaftlichen Nutztiere Fischer Verlag, Jena, pp. 224–259.

Rukkwamsuk, T., 1999. Negative energy balance in postparturient dairy cows: Consequences and adaptations. , PhD Thesis, Utrecht University, pp.17.

Schwartzkopf, G., K.S., Beauchemin, K.A., McAllister, T.A., Gibb, D.J., Streeter, M., Kennedy, A.D., 2004. Effect of feed delivery fluctuations and feeding time on ruminal acidosis, growth performance, and feeding behavior of feedlot cattle. Journal of Animal Science 82, 3357-3365.

Slyter, L.L., 1976. Influence of acidosis on rumen function. Journal of Animal Science 43, 910 –924.

Stock, R., 2000. Acidosis in cattle: An overview. Proc. 33rd Annual Conv. Americ. Assoc. Bov. Pract, Rapid City, pp. 30–37.

Stock, R.A., Britton, R., 1993. Acidosis in feedlot cattle. In: Scientific Update on Rumensin/Tylan for the Professional Feedlot Consultant. Elanco Animal Health, Indianapolis, pp. A1-A16.

Telle, P.P., Preston, R.L., 1971. Ovine lactic acidosis: intraruminal and systemic. Journal of Animal Science 33, 698-705.

Underwood, W.J., 1992. Rumen lactic acidosis, Part I. Compend Contin Educ Pract Vet 14, 1127-1133.

Van Breukelen, P., Wensing, T., Breukink, H.J., 1986. The reliability of some ruminal parameters in predicting milk fat production. In: 4th International symposium of veterinary laboratory diagnosticians Amsterdam, The Netherlands, pp.338 –341.

Van Breukelen, P., Wensing, T., Breukink, H.J., 1985. Some experiments with the feeding of chopped roughage to high producing dairy cows. Zeitschr. Tierphys., Tierern., Futtermittelk 53, 19 –34.

Van Soest, P.J., Sniffen, C.J., Allen, M.S., 1988. Rumen Dynamics. In: Dobson A., Dobson M.J., eds. Aspects of Digestive Physiology in Ruminants, pp. 21–42.

Verheiden, J.H.M., Van Miert, A.S.J.P.A.M., Schotman, A.J.H., Van Duin, C.T.M., 1981. De pathogenese van coliforme mastitis. , Die Pathogenese der Colimastitis. Tijdschr Diergeneesk 106, 501–507.

Yun, S.K. and Han, J.D., 1989. Effect of feeding frequency of concentrate to milking cows in early lactation on pH and VFAconcentration in rumen fluid and on milk composition and milk yield. American Journal of Animal Science 2, 418 – 420.

Zamarreno A.M., Garacia-Mina, J.M., Cantera, R.G., 2003. A new methodology for studying the performance of products against ruminal acidosis. Journal of the Science of Food and Agriculture 83, 1607-1612.