CHANGES IN THE PROFILE OF LIVER ENZYMES IN NEWBORN CALVES INDUCED BY EXPERIMENTAL, SUBCLINICAL ACIDOSIS IN PREGNANT COWS AND OSMOTIC DIARRHOEA

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ABSTRACT
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Experimental, subclinical acidosis was induced by oral administration of saccharose during the last 2 months of pregnancy in 15 cows. Seven cows and their newborn calves were used as a control group. The liver enzyme activities in the serum and the blood acid-base status were determined in the 15 calves from the cows in the experimental group. Mannitol was administered orally to 8 calves from the experimental group to induce osmotic diarrhoea. It was concluded that subclinical acidosis in pregnant cows alters the biochemical liver profile of their newborn calves, affecting the aspartate aminotransferase, alanine transaminase, beta glucuronidase, glutamate dehydrogenase and bilirubin activities in the serum, which are associated with oedematous changes to the hepatocytes. Diarrhoea was accompanied by an increase in the alkaline phosphatase and gammaglutamyl transferase activities and a decrease in the total protein concentration in the serum. These changes were apparently related to the numerous necrotic foci in the liver and the proliferation of the Kupffer cells. It would appear from these results that the liver damage in the newborn calves was associated with the subclinical, metabolic acidosis in their dams and that osmotic diarrhoea occurring in the neonatal period additionally impaired the liver function.

Keywords: acidosis, cattle, diarrhoea, enzymes, liver, neonates

Abbreviations: ALP, alkaline phosphatase; ALT, alanine transaminase; AST, aspartate aminotransferase; βGR, betaglucuronidase; bw, body weight; ELISA, enzyme-linked immunosorbent assay; GD, glutamate dehydrogenase; GGT, gammaglutamyl transferase; IU, international unit

INTRODUCTION
Acidosis in dairy cows most often results from feeding large amounts of readily digestible carbohydrates. As a result of such an excessive supply of carbohydrates, an increased concentration of volatile fatty acids appears in the rumen. Changes in fermentation then lead to a decrease in acetoacetic acid synthesis and an increase in the propionic and then in butyric acid concentrations. A high concentration of butyric acid in the ruminal fluid results in an increase in its concentration in the blood serum, thus leading to acidosis (Moller, 1993). Most animals adapt to the altered feeding regime but, if such a diet is continued for a period of several weeks, it can lead to
metabolic disorders (Erdman, 1993).

Dairy cows are most susceptible to a subclinical form of metabolic acidosis, which manifests itself by a decreased concentration of fat in the milk (Glazer et al., 1992), laminitis, displaced abomasum and poor feed utilization (Nøgaard, 1993). Metabolic acidosis is also a cause of dystocia (Ivanov et al., 1987).

Subclinical chronic acidosis results in pathological changes in the ruminal wall, such as chronic inflammation, ulceration and/or hyperkeratosis (Gabel, 1990). A damaged ruminal wall facilitates the absorption of substances (Erdman, 1993) such as endotoxins, which then pass to the liver via the portal route, so causing damage (Ainmalamali et al., 1992). This has been confirmed by Adamski (1992), Bieniek (1981) and Das and Misra (1992), who observed an increased activity of the liver enzymes in the serum and necrotic lesions in the liver in the course of acidosis in sheep, cows and goats.

Feeding pregnant cows a diet which leads to the development of acidosis also affects the health of their newborn calves (Hejlasz et al., 1987; Janiak et al., 1987).

Some authors have suggested that subclinical metabolic acidosis in pregnant cows is the main cause of the weak calf syndrome (Torres et al., 1987) and of gastrointestinal problems, most commonly diarrhoea, in calves during the first days of life (Fejardo et al., 1988; Torres et al., 1987).

Hejlasz and colleagues (1987) observed that calves born to cows with metabolic acidosis are born in a state of metabolic acidosis and that diarrhoea appears on the second or third day of life. This observation would indicate that the acid–base balance in pregnant cows is an important factor in the aetiology of diarrhoea in newborn calves.

The status of the liver in newborn calves has been the subject of investigations by many authors, who have observed the damage which arises during the course of diarrhoea (Batmaz and Kennerman, 1992; Szulc and Sroka, 1991). However, the relationship between subclinical acidosis in pregnant cows and the status of the liver in their calves has not yet been fully studied. Janiak and colleagues (1987) suggested that feeding pregnant cows an unbalanced diet containing large amounts of readily digestible carbohydrates led to lipid and protein metabolic disorders in their calves which were probably connected with the liver dysfunction.

Our earlier investigations showed that the pathological changes in the liver of diarrhoecic calves, especially those which were 1 day old, were so massive that they had probably begun during intrauterine life (Grodzki et al., 1991). However, the effect of differences in acid–base balance in pregnant cows on the biochemical profile in livers of their newborn calves has not yet been directly investigated.

It was therefore decided to investigate the effect of a 4–5 week period of acidosis prior to parturition in pregnant cows on the biochemical profile in the livers of their newborn calves and also the changes in that profile caused by experimentally induced osmotic diarrhoea.