Trace element deficiencies in the pathogenesis of respiratory distress syndrome in the mature newborn calf

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In Belgium, respiratory distress syndrome (RDS) is one of the leading causes of neonatal death in the mature hypermuscled Belgian Blue calf (BB) but also occurs in other cattle breeds. Major clinical signs (tachypnea, tachycardia and sometimes depression) develop in the first hours after birth and are due to insufficiency of functional surfactant. Knowing that trace elements deficiencies can slow pulmonary maturation, the aim of this study was to investigate trace elements status in 10 RDS affected BB herds in comparison with 6 reference herds without any evidence of RDS.

In each herd, blood was sampled from 10 pregnant or freshly calved healthy cows. In each blood sample, the plasmatic zinc (Zn) and copper (Cu) contents and erythrocytic glutathion peroxydase activity (GSH-pxe) were measured and considered normal when above 15 µmol/L, 14 µmol/L and 250 IU/gHb, respectively. A herd was deficient in one element if at least 30 % of sampled animals were out of normal range for this element. Milk was also sampled and pooled from 10 other cows or, when possible, bulk milk was taken. Milk iodine (I) content was considered normal when above 80 µg/L. Results were compared between groups by Chi-square test.

All RDS affected herds had low Zn and Cu concentrations and low GSH-pxe activity. Eight out of 10 had low I in milk. In the non-RDS affected herds, only 1 herd was deficient in I, Zn, Cu and had low GSH-pxe activity, 2 herds were deficient in Zn and Cu and 1 herd was deficient in Cu. RDS affected herds were significantly more often deficient in I and GSH-pxe than non-affected herds.

It seems that the trace elements selenium (Se), Zn and Cu play an essential role in the development of RDS. The same applies for I, although not deficient in all herds. The reason for this might be that milk samples were taken during lactation, when cows' nutrition was different and, in any case, supplemented in I. In mature babies, the same RDS is observed but its etiology is still unclear, although I deficiency in the mother is suspected to play a major role in pathogenesis. Furthermore, it has been demonstrated that a Se-dependent deiodinase is responsible for transformation of thyroxine (T4) into tri-iodothyronine (T3), which is essential for effective surfactant production.

In conclusion, results suggest an association between RDS in mature newborn calves and trace elements deficiencies, especially Se and I, that can be responsible for primary surfactant insufficiency.