Hereford bull face bone structure change

Company/Ranch Name Name Email Address Address City City State Zip Headcount Weight Cattle Purchase Purchase Date Cattle Origin Cattle Location Cattle Location Current Livestock Location Status Frame Status Frame Score Body Condition Score Quality Weight Variation Of Primary Breed Calves Are 100% Hereford of Calves are 100% Hereford x Angus Red of Calves are 100% Hereford x Angus Calves are other Start Calvin Race Bull) Genetic Comments Genetic Comments Feed/Nutrition Program De-Wormed Used Weaned Vaccinations Date Bocos Bangs Vaccinated Bangs Vaccinated Implanted Implanted ImplantEd Implant Kind Implant Date Bunk Broke Castration Spayed Open Management Comments Auction Market Market Market Auction Lot Number(s) : Weighing time : % Shrink Slide ($/cwt) Marketing Comments Marketing Representative: Roger W. Blowey BSc BVSC FRCVS FRAgS, A. David Weaver BSc DR MED VET PHD FRCVS, in Color Atlas of Diseases and Disorders of Cattle (Third Edition), 2011a cartilaginous growth failure generally as an inherited defect.the hereford calf (1.6) demonstrates brachycephalic enanism. The head is short and abnormally wide, the lower jaw is overtaken, and the legs are very short. The abdomen also enlarged. The calf had difficulty standing, was dysrenic as a result of the deformity of the skull (snoring dwarf), and there was also a cleft palate. A 2-week-old Simmental cross knocker calf (1.7) shows a strong inclination of all four legs, especially the forelegs, stunted growth and a slightly scruffy face, and euthanasia was indicated. Born in May from a silage-only fed prey, additional feeding seemed to reduce the incidence of achondroplasia from 40/200 to 5/200 offspring in successive years. Bulldog calves are often stillborn (1.8). This Ayrshire has a large head and short legs, but also has extensive subcutaneous oedema (anasarca). Enanism is inherited in several races, including Hereford and Angus.A related condition is congenital joint laxity and enanism (CJLD), which is a distinct congenital anomaly in Canada and the United Kingdom. A severe case of CJLD from Canada, the newborn calf (1.9) has a crouching appearance, short legs, metacarpophalengic hyperextension and sickle-shaped hind legs. Many calves are disproportionate dwarves. The joints become stable within 2 weeks and calves can walk normally. No other anomalies are observed. In the UK in 2009/10, 70 of a group of 85 South Devon x Angus calves showed shortened limbs, joint laxity (especially fetlocks), dyspnoea in the first few days of life, and in some cases braquignathia. The had been fed straw after housing, and later straw and silage. Meliton N. Heifer, in Reproductive toxicology and Development, 2011Fifteen Hereford crossbars and heifers with body weight range of 272 to 338 kg were assigned 5/treatment group and micelial monensin in suspension of aqueous acacia was administered to 5% suspension of aqueous acacia at doses of 12.5, 22.4 or 39.8 mg/kg body weight per gavage. Another group of four similar animals were gavaged with solvent extracted mycelium, free of monensin, equal in total mycelial weight to the high dose of monensin. Fourteen-day losses to death were 0/5, 3/5 and 2/5, respectively, for groups of 12.5, 22.4 and 39.8 mg/kg. No cattle treated with mycelium without monensin died. In this study, the median lethal dose (LD50) of monensin was calculated to be 35.8 mg/kg ± 13.5 mg/kg. Clinical signs of poisoning consisting of anorexia, diarrhea and mild depression occurred as early as day 1 post-treatment (PT). By day 5 PT, laborious breathing and moderate to severe depression were observed, but by day 10 animals were consuming food normally. All surviving cattle were apparently normal by day 12 PT. There were no pathological findings in control animals to which mycelium without monensin was administered. Striated muscle lesions of necrosis and fibroplasia were found in the heart and necrosis and regeneration in skeletal muscles in several animals treated with monensin. Roger W. Blowey BSc BVSC FRCVS FRAgS, A. David Weaver BSc DR MED VET PHD FRCVS, in Color Atlas of Diseases and Disorders of Cattle (Third Edition), 2011the hereford heifer crossed in 7,167 is anti-year old and has enlarged lockfets and a characteristic brown sheath to the hair layer. (Hereford also has lice.) Hair loss and hair pigment (7,168) can produce a spectacular appearance, as seen in the cross calf calf at 7,169, which also shows the thick hair typical of copper deficiency. Bone fragility and anemia are other clinical features. Brazilian cows at 7,170 show poor growth, poor hair layer and pigment loss. The enlargement of the fetlock joint (7,171) is due to the widening and irregularity of distal metacarpian physiasias, as seen on X-rays (7,172) of an affected animal (left) compared to a normal animal (right). Similar radiographic changes are seen in digits. Other animals may atrophy, develop sloping legs, contracted tendons and kyphosis. Excluding phosphorus deficiency, a copper deficiency may be the most severe mineral limitation for cattle grazing in large tropical regions. In Veterinary Medicine (Eleventh Edition), 2017The death at the age of 2017 of Poll Hereford and hereford calves with horns up to 3 months of age can be caused by hereditary cardiomyopathy. Calves are identifiable before death by their very fast growth rate, short curly layer and moderate bilateral exophthalm. Death is usually precipitated by stress or exercise and is characterized by dyspnoea, the passage of bleeding foam from the nose, and a course of a few minutes to a few hours. Less acute cases have congestive heart failure syndrome days before death. Life expectancy is less than 6 months. In necropsy there is an obvious spot of the myocardium, reminiscent of a bad case of white muscle disease. The disease appears to be conditioned by a single autosomal recessive gene. JULIE A. DENNIS, ... PETER J. HEALY, in Animal Models of Movement Disorders, 2005Hyperekplexia in newborn Hereford calves was first identified in the United States of America and published as Hereditary Neuraxial Oedema (HNO) (Cordy et al., 1969). Subsequently, similar clinical syndrome was observed in Hereford calves surveyed in newborns in Australia (Blood and Gay, 1971) and England (Weaver, 1974), and in Hereford cross calves polled in New Zealand (Davis et al., 1975). The breeding records of American herds were considered consistent with the autosomal recessive inheritance of the condition. No data were submitted with the Australian and English cases to corroborate this conclusion, although New Zealand cases were born for Hereford chicken cross of Holstein cows paired with a Poll Hereford bull. Subsequent research confirmed that the hyperekplexic component of HNO syndrome was inherited autosomal recessively (Healy et al., 1985) and resolved disparities in the association between hyperekplexia and pneuraxis edema in some reports of HNO syndrome (Healy et al., 1986; Harper et al., 1986a). In the initial US report (Cordy et al., 1969) three of the twelve affected calves examined had severe spongy vacuo relaxation of the white matter of the central nervous system (CNS). The degree of vacuolation in the remaining nine cases was lower and was recorded as microvacuolation. Doctors recorded that a calf had fractures of the epiphysary plaques of both femurs. Blood and Gay (1971) did not notice significant microscopic lesions in the CNS of nine cases they studied. Studies of thirteen HNO-affected calves born in Tasmania (Donaldson and Mason, 1984) revealed five significant hip injuries, and ten with HNO-consistent histological injuries as described by Cordy and his colleagues (1969). In examining records of the laboratory component of the veterinary diagnostic service in New South Wales, the researchers established that during the 1970s and early 1980s HNO was the most common diagnosis offered to field veterinarians investigating suspicious inherited diseases in newborn calves. In response, the researchers established an experimental breeding herd by buying cows and bulls reputed to be parents of calves who showed myoclonic spasms sensitive to stimulus and could not stand. During 1981, 1982 and 1983, fourteen fifty-six calves born in the herd on display, birth, myoclonic spasms of the whole body with stimulus response similar to those described by Cordy and his colleagues (1969), Blood and Gay (1971), Davis and his colleagues (1974), and Donaldson and Mason (1984). (1984). of the fourteen calves born in this experimental breeding herd could rise from birth unanstioned, however, they all showed normal minting, with no severe Spongy spongy cnS vacuolation. Thirteen of the fourteen calves had injuries to the hip joints, the most common being the flattening of the acetabular pit and the burn of the femoral head. Researchers suggested that a separate disorder was more likely to be present in the surveyed population of Hereford to account for cases of calves with neuraxis oedema (Harper et al., 1986a; Healy et al., 1986). Researchers established a smaller reproductive herd composed of calves' parents who had severe spongy CNS vacuolation. The twenty-one calves born in this herd could increase unanstioned after birth, but six developed clinical signs of progressive CNS dysfunction within thirty-six hours of birth. These six calves were euthanasiated between three and five days of age due to progressive upper central nerve dysfunction, and all had severe spongy CNS vacuolation. Estimates of amino acid and ketoacid concentrations in bodily fluids and tissues indicated extreme elevations of branched-chain amino acids, valine, leucine and isoleucine, and their derived ketoacids. This finding was consistent with the calves affected with branched deficiency of the ketoacid chain dehydrogenase, or maple syrup urine disease (MSUD) (Healy et al., 1986). The two breeding experiments, and the results of clinical, biochemical and pathological examinations, clearly confirmed that the syndrome formerly known as HNO comprised two distinct entities, which are inherited as autosomal recessives. The condition, characterized by spontaneous and stimulus-sensitive myoclonic spasms that were evident from birth, was given the descriptive title, inherited congenital myoclonus (ICM) (Harper et al., 1986a). The disease characterized by progressive CNS dysfunction, with high concentrations of branched-chain keto and amino acids, is clearly bovine MSUD (Harper et al., 1986b). Roger W. Blowey BSc BVSC FRCVS FRAgS, A. David Weaver BSc DR MED VET PHD FRCVS, in Color Atlas of Diseases and Disorders of Cattle (Third Edition), 2011a 1-week-old Hereford crossed calf (2.22) is dying and passing dying dying and passing dying mucous membranes, a mixture of blood, mucus and intestinal lining. Classically, necropsy revealed diphtheria enteritis (2.23), with thickening of the mucosa. However, not all calves are affected so severely. Although Salmonella enterica, serovar Typhimurium was isolated from the dysenteric faeces of the affected 3-week-old Frisian calf I was just slightly ill. Other cases show slight intestinal inflammation, the main changes being pulmonary congestion and epicardial and renal hemorrhages. Animals recovering from Peruvian sepsis (especially S. dublin) may occasionally develop necrosis of the limbs, limbs, ear tips, tail and legs. Frison of 4 months (2.25) was recovering from nonspecific pyrexia that had affected her about 6 weeks earlier. No enteritis had been observed, and is often not involved, but later ear tip necrosis resulted in a bilateral detachment of more than half of the pinna. S. dublin was isolated from the faeces. In the 4-month-old crossed Hereford (2.26), circumferential skin necrosis immediately above the rear fetishes has produced gangrene and limb necrosis. The over-extension in the fetlocks is probably due to the rupture of the flexor tendon. Salivation is an answer to pain. Thomas J. Divers, Alexander of Lahunta, in Rebhun's Diseases of Dairy Cattle (Third Edition), 2018 Inherited forms are described in numerous races, including Hereford, Charolais, Dexter, Ayrshire, and Holstein. Obstructive hydrocephalus is often accompanied by other brain malformations, which will influence the character of clinical signs. A common cause of obstruction is the failure of the mesencephalic aqueduct to develop normally. The latter may be associated with the presence of a single structure representing the colliculi rostrales. The cause of this mesencephalic malformation is unknown in cattle, but is inherited in laboratory rodents. Clinical signs will be prosecephalic, but the brainstem and cerebellar signs may be present if there is a significant increase in intracranial pressure. Roger W. Blowey BSc BVSC FRCVS FRAgS, A. David Weaver BSc DR MED VET PHD FRCVS, in Color Atlas of Diseases and Disorders of Cattle (Third Edition), the 2011 eyes of the 4-day Hereford cross-grill in 8.4 were affected and the animal was totally blind. In other animals, only one eye may be affected, or cataract may not cause total vision loss. Congenital cataract is not normally progressive. Cattle cope with significantly good blindness and can be raised in confinement systems. They quickly learn to stay within the group, although handling can be difficult. Blind dairy cows will learn to follow the herd to and from the pastures. Congenital cataract may be inherited, or it may be the result of the teratogenic effects of maternal REN DISEASE infection during early/medium pregnancy. 8.5 shows a congenital nuclear cataract in a young calf in Friessia. Note in the acquired cataract the two large synechiaas (adhesions of the iris to the cornea), and the opacity and wrinkles of the lens in the cow Guernsey in 8.6. Cataracts may be secondary to inflammatory processes within the eye, when they can be progressive. In contrast, congenital cataracts (8.4) are rarely progressive. In Veterinary Medicine Edition), 2017 Secondary bullous epidermolysis (JEB) is inherited in Belgian foals; Danish Hereford, Belgian Blue, Charolais, Angus and Simmentales Calves; and Suffolk, Churra, and lambs from south Dorset Down.1-4 The disease is inherited in an autosomal-recessive pattern. JEB on horses is a trait affecting Belgians, other draft breeds, and American horses of the saddle.5,6 Heterozygote haplototype is common in barrel breeds, with 17% of Belgian horses being carriers in North America and 8% to 27% of Breton horses, Comtois, Vlaams Paard and Belgische Koudbloed Flander breed in Europe.The genetic defect responsible for JEB in the Belgian and European draft breeds is an insertion of cytosine (1368insC) creating a premature stop codon in the , which encodes the laminin subunit chain No. 2. Truncated subunit strings of laminin n.o 2 lack the C-terminal domain, so it cannot interact with the other two subunits, thus avoiding the formation of laminin 5. The defect in Belgian blue cattle is in the LAMA3 gene, which encodes the alpha 3 subunit of laminin-332.4 Laminin is widely distributed in the basement membrane of epithelial tissues, and the lack of this family of proteins results in loss of cellular adhesion between dermis and epidermis.4 Disease in Charolais calves is the result of a mutation in the beta 4 deinegrine gene (in terms of disease in humans) and loss of function of this protein.2 Disease in hereford cattle is the result of a mutation in the LAMC2 gene, encoding the laminin gamma 2 protein, which results in loss of gene function and lack of laminin gamma protein 2.3The parts are usually born alive, but irregular and red erosions and ulcerations develop in the skin and mouth above pressure points or after mild trauma. There are often extensive erosions along the coronary bands, with the detachment of hooves, and in the mucocutaneous joints of the mouth, rectum and vulva. Dystrophy teeth that are visible at birth are white with irregular serrated edges and chopped enamel. Definitive diagnosis in shooting horses requires DNA testing for JEB. There is no treatment for the affected foals, lambs or calves, and they eventually succumb to secondary infections or a complete detachment of hooves. Hooves.