and cobalt sulphate, 1.5 g) has been reported to be effective in the treatment when administered at the rate of 15-40 g/day along with vitamin B_1 (10-20 mg/kg BW) and ascorbic acid; the affected animal should be fed high protein diet.

Deficiency of Selenium

Deficiency is becoming much more common than selenium toxicity because of its presence in glutathione peroxidase ($GSH\text{-}P_X$), a selenoenzyme. Selenium is indispensable for protection of haemoglobin and the erythrocyte membrane. Erythrocyte glutathione peroxidase activity is an important indicator of the selenium status of livestock.

Oxalate Poisoning

Oxalic acid is an organic acid occurring in a range of plant species commonly consumed by ruminants. It is toxicologically antagonistic to divalent cations such as calcium, magnesium, etc. The dietary intake of oxalic acid is dependant on the feeds and fodder ingested by the animals. For example, wheat straw contained negligible amount of total oxalate (0.17%) while paddy straw has 1.54%. This acid in ruminant diet has an adverse effect on calcium assimilation (Talapatra et al., 1948).

Oxalate poisoning in livestock occurs primarily from the ingestion of the oxalate-containing plants of the wood-sorrel family (*Oxalidaceae*) in Australia and of the chenopod or goosefoot family (*Chenopodaceae*) in North America (Greasewood plant) and in central Asia (Halogeton plant). The oxalate in the plants of *Oxalis spp.* and some species of *Rumex* exists chiefly as acid potassium oxalate while that in plants of *Chenopodaceae*, it exists chiefly as soluble sodium oxalate and insoluble calcium and magnesium oxalate. Grasses may have ammonium oxalate. Oxalic acid also forms on moist straw infected with the fungi, *Aspergillus niger* and *A. flavus*. Oxalates present in certain tropical grasses such as those in the genera *Cenchrus, Setaria, Pennisetum, Digitaria* and *Brachiaria* may cause chronic renal failure in grazing ruminants due to formation of oxalate crystals and urinary calculi.

Oxalate Content in Green Fodder

The oxalate content varied from 2.10 to 3.92% in both the hybrid napier cultivars (PBN-231, PBN-83). In multi-cut grasses, total oxalate content increased linearly with the successive cut (Ahuja et al., 1998) and

continuous feeding of such fodder could result in ruminal alkalosis. The oxalate content can be reduced by ensiling, since anaerobic microbes degrade oxalates to carbonates and finally to carbon dioxide which increase the silage pH. In single-cut grasses, total oxalates decreased with increasing age of plant (Middleton and Barry, 1978). Bajra fodder contained more amount of oxalate in the early stage than in the late stage of growth. Similarly leaves had higher amount of oxalate than the stem. Bajra fodder plant has 2.12% oxalic acid at preflowering stage. Soaking in water reduce the oxalic acid content (Parveen et al., 1988).

Oxalate content %

Plant height (cm)	*Oxalic acid content %*
144	2.78
155	2.70
182	2.62
210	2.52
230	2.52
242	2.41
290	2.30
300	1.93
330	1.03

Plant part	*Bajra fodder*	*Napier grass*	*Bajra straw*
Leaves	3.19	2.21	2.9
Stem	1.36	1.46	1.09
Whole plant (300 cm height)	1.93	1.60	1.88

Ajaib Singh (2002) reported the seasonal variations in oxalate content of Napier Bajra Hybrid (PBN-233). The total oxalate contents increased linearly and varied from 2.20 to 3.60% during the successive cuts from April to August. The level of oxalate was significantly more in the month of June and July, which might be due to the peak growth in summer and rainy seasons. Roughly one third of the oxalates was found to be present in soluble form and the relative proportion of soluble and insoluble oxalate remained constant during this growth period. When the fodder was harvested at one and two metre heights, the total oxalate contents were found to be 2.80 and 2.30%, respectively. However, the proportion of soluble oxalates increased by 9.7% at two metre height.

Oxalate Metabolism

The presence of oxalate degraders is widespread among the gastrointestinal tract of many species, including rodents, rabbits, guinea pigs, swine, horses, ruminants and human beings. However, not all individuals within a species possess oxalate-degrading bacteria. These bacteria (*Oxalobacter formigenes*) are inhabitants of ovine and bovine rumens as well as the large intestine of human beings and other nonruminant animals. Adapted animals can tolerate levels of oxalates which would be lethal to nonadapted animals.

In ruminants, Talapatra et al. (1948) studied the dynamics of ruminal metabolism of oxalate using the plant *Halogeton glomeratus* (Halogeton is a branched annual herbaceous plant native to arid alkaline soils, barren soils. It contains high concentration of oxalate. Sheep die in a sleeping position, characteristic of effects of hypocalcaemia, from halgeton poisoning). When oxalate is consumed by a ruminant animal, it may be degraded by certain rumen bacteria (*Oxalobacter formigenes*) to a nontoxic form (formic acid and carbon dioxide), or it may combine with calcium or magnesium to form insoluble salt, or it may be absorbed from the rumen into the bloodstream where it may combine with calcium eventually to produce hypocalcaemia or oxalate may interfere with other body processes and / or be excreted. Soluble oxalates are degraded in the rumen to carbonate and bicarbonate, which raises rumen pH (eventually leading to severe ruminal alkalosis) and slows down the microbial activity. Of course, this depends on the nutritional status of the animal and the functioning of the ruminal microflora. The insoluble oxalate salts may accumulate in various tissues, especially the rumen wall and kidneys. In certain cases calcium oxalates paralyse the brain. They may also cause destruction of the RBC.

The studies of Panda and Sahu (2002) showed that soluble oxalates were completely degraded and about 50% of insoluble oxalates were broken down in the gastrointestinal tract of the bull. It can be concluded that the total oxalate intake at the level of 0.58% of the DM intake may be harmless with the calcium intake of 7.8 g/100kg body weight. While delineating oxalate toxicosis, James (1972) attributed death of the animal to the following:

1. Hypocalcaemia
2. Uremia resulting from the damage of the kidneys by the oxalate crystals
3. Interference with energy metabolism since oxalate interferes with succinic dehydrogenase and lactic dehydrogenase enzymes.