

**Genesis™ Injection Abamectin Plus Vitamin B12**  
**Antiparasitic for cattle & sheep**

## Vitamin B12 Deficiency in Sheep and Cattle

Vitamin B12 deficiency in animals is the end result of insufficient Cobalt intake. Cobalt is acquired from the pasture and soil as animals feed and is used to make Vitamin B12 by micro-organisms within the rumen (2nd stomach). Vitamin B12 is then absorbed in the small intestine and transported throughout the body by the blood, with excess being stored in the liver. A female animal will provide B12 to a growing foetus, but will not supply any of the vitamin in its milk.

The amount of Cobalt available to animals varies as a result of several different factors. The amount of Cobalt occurring naturally in the soil varies according to the type of rock from which the soil is derived. The amount then available for uptake by the pasture is influenced by the presence of other minerals (e.g. Manganese) and supplements (e.g. lime), which can bind the Cobalt in the soil and prevent its absorption by plants.

Rainfall can also play a major role, with high rainfall leading to leaching of Cobalt from the topsoil into the lower soil layers. This can be seen as a seasonal effect where rainfall is concentrated at one time of the year.

The type and condition of the pasture can also play a major role. In general, grasses take up less Cobalt than legumes and lush, fast-growing pastures absorb Cobalt at a lower rate than slower growing pastures.

These factors mean that coastal areas are particularly prone to Cobalt deficiency and the deficiency has been well documented in coastal southern Australia. Cobalt deficiency has, however, also been identified in many inland parts of Australia.

Vitamin B12 is essential for cell growth and maturation, energy production and wool growth. In general, sheep are more susceptible to Vitamin B12 deficiency than cattle are. Rapidly growing animals (i.e. lambs/calves and weaners) are most likely to develop a B12 deficiency and will suffer most badly when a deficiency develops. Pregnant animals, particularly when young, are also vulnerable since they must provide sufficient B12 for a rapidly growing foetus as well as themselves. Other classes of animal are less likely to develop a B12 deficiency, but all will be affected if a deficiency develops.

Clinical signs of Vitamin B12 deficiency include weight loss, suppressed appetite, decreased feed efficiency, anaemia, diarrhoea, rough coat, scaly ears and weepy eyes. These signs may occur in different combinations and the clinical picture is often not very clear. In many cases B12 deficiency manifests as a wasting disease and, in severe cases, the mortality rate may be high. Like most diseases, however, far greater economic losses result from undetected subclinical disease causing reduced production than from clinical outbreaks. Vitamin B12 deficiency can lead to reduced fertility, lower birth rates and birth weights, reduced growth rates, and reduced wool growth and wool quality.

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Vitamin B12 deficiency also impairs the immune function of animals so that they are more likely to acquire infectious diseases and will suffer worse when infected. Animals that are deficient in B12 are likely to carry a higher worm burden than they otherwise would. The high worm burden causes scours and damage to the wall of the intestine so that less B12 is absorbed in the gut and the immune system is suppressed even further. Thus, a vicious circle leading to severe disease is created.

It is important to remember that the animals that are most susceptible to Vitamin B12 deficiency are also those that are most susceptible to infection with worms. Good worm control allows animals to make best use of the Cobalt that is available in the environment and prevents worm populations from taking advantage of an animal's depressed immune system. Prevention of Vitamin B12 deficiency allows animals to withstand worms better and minimises the required frequency of drenching. B12 also helps in the repair of damage after a worm burden has been removed. It is therefore good practice to consider treatment programmes for the two diseases together rather than in isolation.

Prevention or treatment of B12 deficiency is best achieved by providing a Vitamin B12 supplement directly to the animals. It is important to know the level of deficiency to determine the frequency with which animals may require supplementation. The level of Vitamin B12 deficiency can be assessed easily using a blood test. In cases where subclinical deficiencies are suspected, at least 10 individuals from the most susceptible class of animal should be tested at the most critical time of the year.

In areas where the Vitamin B12 deficiency is marginal, treatment of the susceptible classes of animals (i.e. lambs, calves and pregnant animals) may be sufficient to prevent problems and minimise production losses. In areas with severe deficiency problems, treatment of all animals may be required on an ongoing basis.

It is good practice to provide all animals entering a feedlot with a B12 supplement as part of their induction programme. In cases of even marginal deficiency, the B12 will enhance the animals' appetite, getting them onto feed quicker and providing better feed efficiency and improved weight gains (as demonstrated by the 17kg weight advantage in the graph below).

## Weight gains

