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IN BRIEF

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DairyNZ

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Getting plantain into your system

New Zealand-bred plantain (*Plantago lanceolata* L.) cultivars are useful forages for dairy cows, not only for feed quality and good summer-dry forage production, but some are now recognised for their environmental benefits by reducing urinary nitrogen (N) concentration.

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Here we will review the benefits and challenges around sucessful establishment and management of plantain in general, recognising that, as yet, there is limited published information on cultivar differences.

Plantain is an upright perennial dicotyledon herb with a coarse, fibrous root system. The species grows throughout New Zealand on a wide range of soils, including those of low to medium fertility and a wide pH range, but it is not suited to water-logged or saline soils[1].

Modern forage plantain can respond to good management by expressing a production potential similar to perennial ryegrass when newly established[2,3]. As plantain has a moderate to high tolerance of summer heat, in warmer and drier regions it can provide valuable forage and improve milk production during summer and autumn[4,5,6].

Key findings

- Plantain can be established as a pure crop, in a herb-based mix or in a grass-based mix.
- Plantain establishes well in most regions and soils, and late spring sowing by direct drilling generally results in the best first-year yield.
- Frequent grazing and treading damage in winter will reduce plant density. Re-sowing after two to four years may be required to maintain a high proportion of plantain in the pasture.
- Two herbicides are now registered in New Zealand for use with plantain – Dynamo in mixed pastures and T-Max in forages without clover.

In the last five years, new impetus for plantain use has come from a number of studies that have consistently shown the cultivar Ceres Tonic to significantly reduce N concentrations in cattle urine[4,5]. This has important benefits for reducing nitrate leaching risk in dairy systems, where sufficient plantain is included in the animal diet.

The Forages for Reduced Nitrate Leaching (dairynz.co.nz/frnl) and Greener Pastures (http://nsentinel4.co.nz/collaboration/) research programmes are now determining the optimum dietary levels and how to achieve them in farm systems. Good establishment and management of forages containing plantain will be critical to getting the maximum benefit from plantain.

Establishing plantain

Plantain can be sown in a variety of ways – as a pure sward, mixed with clovers and other herb species ('herb-based mix') or mixed with grass and clover ('grass-based mix'). The different forage types have their own advantages and disadvantages (Table 1).

Which type to use depends on the intended purpose of plantain on-farm. In all situations, plantain is best established by sowing at 10 mm depth, into a cultivated seedbed or direct drilling seed into herbicide-treated pasture, when the soil temperature is above 10°C. A range of establishment methods have been used in various field trials.

The only trial directly comparing methods showed that the more expensive direct drilling, compared with broadcasting seed, was more than compensated for by more dense plant populations, lower weed content and increased yield[7].

As with any forage species, sowing date will depend on the temperature and moisture conditions dictated by region and soil type. For plantain, there have not been any direct comparisons of sowing date, but both autumn and spring sowing has been successful in North and South Island regions, with summer sowing an option where irrigation is available.

For spring sowing, planting too early risks a late frost damaging young plants, while planting too late risks dry conditions reducing plant establishment and survival[9]. On winter-wet sites, a spring sowing after spraying with glyphosate, light cultivation and drilling is likely to be most successful[6,7].

For autumn sowing, planting too early risks poor germination in summer-dry conditions, while planting too late may not ensure sufficient leaf growth and root development before cooler winter temperatures arrive, and risks the first grazing being in wet and cold conditions[8].

Plantain as a pure sward

The main advantage of a pure plantain sward is that it can be managed to meet the requirements of the plantain cultivar. Optimal sowing rates for pure swards are 7-10 kg/ha[11].

Table 1: advantages and disadvantages of plantain forage options

Forage types	Advantages	Disadvantages
Pure plantain	• Dedicated grazing management (greater plantain production and persistence). • Maximum benefit from plantain (e.g. minerals and reducing N leaching).	• Slow winter growth of some cultivars compared with ryegrass. • Difficult to utilise well in wet winter conditions on heavy soils.
Plantain with clover and/or chicory ('herb-based mix')	• Dedicated grazing management (greater plantain production and persistence). • High benefit from herbs (e.g. minerals and reducing N leaching). • Clover provides nitrogen. • Clover fills in gaps in sward rather than weeds.	• Slow winter growth of some cultivars compared with ryegrass. • Difficult to utilise well in wet winter conditions on heavy soils. • Potential bloat risk if swards become clover-dominant.
Plantain in diverse pasture mix ('grass-based mix')	• Better pasture production in summer/autumn. • Higher nutritive value in summer/autumn. • Longer growing season. • Less N leaching if plantain present in sufficient proportion.	• Animals may selectively graze. • Feeding value of herbs diluted. • Plantain plants may not persist. • Few herbicides available for control of broad-leaf weeds

Plants should have a minimum of six fully developed leaves before they are ready for the first grazing, to ensure that plants have a well-developed root system[8].

Grazing management strategies designed to maximise quality and quantity of plantain crops aim to maximise leaf growth and minimise stem growth. Grazing at ~25 cm height down to a residual of ~8 cm is recommended, because as plantain leaves age they become more fibrous, less digestible and the quality declines regardless of stem content[9]. The main disadvantages of a pure plantain sward are poor winter growth compared with perennial ryegrass (although this varies with cultivar) and the relative sensitivity to pugging and winter damage due to the open crown growth of plantain.

Plantain in a herb-based mix

The main advantage of herb-based mixes is the high forage quality from combining plantain with chicory and/or clovers[3]. Optimal sowing rates are 6-8 kg/ha for the plantain component, with the other species making up an additional 10-12 kg/ha (e.g. chicory and clover).

The guideline of first grazing at the six-leaf stage also applies to herb mixes[8] and, subsequently, it is recommended that swards be grazed to ~8 cm residual heights with four-week intervals[3]. For dairy cows, the main disadvantage of this forage type is the risk of bloat, which has been anecdotally reported.

Plantain in a grass-based mix

Plantain improves the summer quality and autumn recovery of perennial ryegrass pastures, especially in summer-dry environments[6]. Optimal sowing rates are 2-4 kg/ha, any lower has a minimal benefit for herbage production[10]. Plantain in a ryegrass pasture mix is necessarily managed as with a ryegrass-white clover pasture (grazing to residuals of 1500-1600 kg DM/ha), which is within the tolerance of plantain[9,10].

Plantain can be drilled or broadcast into already established pasture, although establishment is slower and plant populations may not reach the density required to affect urinary nitrogen concentration. The recommendation to graze at six developed leaves does not apply, as this will be too long a rotation for the grass and will shade out the plantain seedlings. A normal ryegrass rotation will allow the plantain to emerge within the established pasture.

Figure 1: one-year-old perennial ryegrass/plantain/clover mix near Dannevirke. The pasture was sprayed, drilled and rolled in March 2017. The seed sown comprised 22 kg perennial ryegrass + 3 kg plantain + 2 kg sub clover + 2 kg white clover.

Challenges for management

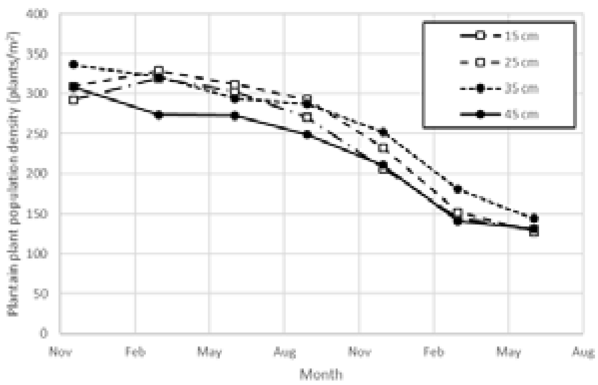
The main challenges for managing plantain are maintaining plant population density, control of broad leaf weeds and sporadic palatability issues.

A substantial reduction in plant numbers can occur after 2-4 years and thus plantain is often regarded as a short-lived perennial[11]. Only one study (in Northland) has reported contributions of plantain in mixed swards greater than 15 percent of total forage dry matter after four years[6].

When plant populations have been measured, the decline has often occurred in the second summer-autumn[7,9] (e.g. Figure 2) and is often associated with insect pests. Severe outbreaks of plantain moth infestation can occur in pure plantain swards, which can be controlled with the registered insecticides Exirel or Minecto Star. In herb mixes and grass mixes, the moth damage is generally not substantial enough to warrant action.

Instead, plants appear to be mainly lost through competition from other species, by short grazing rotations and from treading damage on wet soils. While plantain appears tolerant of low grazing residuals, it must be allowed to recover to six leaves (or approximately 25 cm in height) for critical root reserves to be replenished[9].

Figure 2: typical loss of plantain populations during a Waikato study of pure plantain swards harvested at four pre-graze heights: 15, 25, 35 and 45 cm[9].



Plantain is a prolific seeding plant and new plants can establish from natural reseeding under rotational grazing with dairy cows[1]. A detailed Manawatu study showed new plants making up 14% of growing shoots from natural establishment over winter and spring[12].

Stock will avoid eating seed heads and germination occurs quickly with sufficient moisture and warmth, though seedling survival is best where there is bare ground and competition is limited[1,12]. However, to maintain plant populations at a level sufficient to ensure plantain is a high proportion of the cow's diet, under-sowing by drilling or broadcasting plantain seed in spring may be necessary.

It is wise to eliminate weeds as thoroughly as possible before sowing plantain, as there are few post-establishment herbicides available that do not harm plantain[13]. Only two products are currently registered, Dynamo (a.i. bentazone) can be used in grass-based mixtures and T-Max (a.i. aminopyralid) can be used in pure plantain crops or mixtures without clover.

Plantain cultivars

Several plantain cultivars are now available for dairy farmers. They largely differ in their flowering dates and seasonal growth rates (with implications for seasonal growth habit and palatability) but there is currently no published information on their relative efficacy in reducing urinary nitrogen concentration.

There is little evidence that establishment requirements differ substantially between cultivars, although late autumn sowing of winter-dormant cultivars should be avoided.

Resources

- DairyNZ Farm Fact Plantain Establishment 1-78a

  DairyNZ Farm Fact Plantain Management 1-78b

  dairynz.co.nz/farmfacts (>farm management >forages)
- The proceedings of the New Zealand Grassland Association has over 40 papers covering various aspects of plantain forage management, of which 15 relate to dairy systems. https://www.grassland.org.nz/

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Is failure of passive transfer affecting your heifers?

Rearing healthy, high genetic merit replacement stock is key to the future productivity of a herd and management strategies for stock health start from day zero. This is because calves are born with a poorly developed immune system.

Emma Cuttance, **VetEnt**

Unlike humans, the bovine placenta prevents the transfer of large immunoglobulin molecules (antibodies), which are essential for immunity, from crossing the placental-blood barrier and into the foetal calf.

Instead, newborn calves must ingest colostrum to absorb immunoglobulins across their intestinal wall and obtain sufficient immunity until their own immune system becomes functional. This transfer of immunity via colostrum is most effective in the first 24 hours after birth and is often referred to as 'passive transfer'.

When calves receive sufficient quantities of good quality colostrum there are measurable, high concentrations of immunoglobulin in their blood. Calves that fail to absorb sufficient quantities of immunoglobulin in those first 24 hours suffer from failure of passive transfer (FPT) of immunity.

FPT can be identified by analysing blood samples for concentrations of immunoglobulin G (IgG) or total protein (TP).

Key findings

- Failure of passive transfer (FPT) occurs when calves fail to absorb sufficient quantities of immunoglobulin from colostrum in the first 24 hours of life.
- A recent New Zealand study indicates that, on average, about a third of calves have FPT, but this prevalence varies widely between farms from 5 to 80 percent of calves.
- Calves with FPT are immune compromised and are, therefore, at a greater risk of disease and mortality. This effect is still evident at 12 months of age.
- Strategies to improve quality and timely intake of colostrum will result in improved health and survival.

IgG concentrations less than 10 g/L or TP concentrations less than or equal to 52 g/L indicate FPT. To assess herd prevalence of FPT, samples should be collected from healthy calves between 24 hours and seven days old.

In overseas studies, FPT has been reported to affect, on average, 19 to 40 percent of calves[1, 2]. A similar prevalence has been recently reported in New Zealand. In a 2015 study of 4000 dairy calves from 106 seasonal pasture-based farms across nine different regions, 33 percent of calves had FPT, but the prevalence ranged between 5 and 80 percent on individual farms[3].

This study indicated many calves are not getting enough good quality colostrum immediately after birth to ensure passive immunity.

What are the effects of FPT?

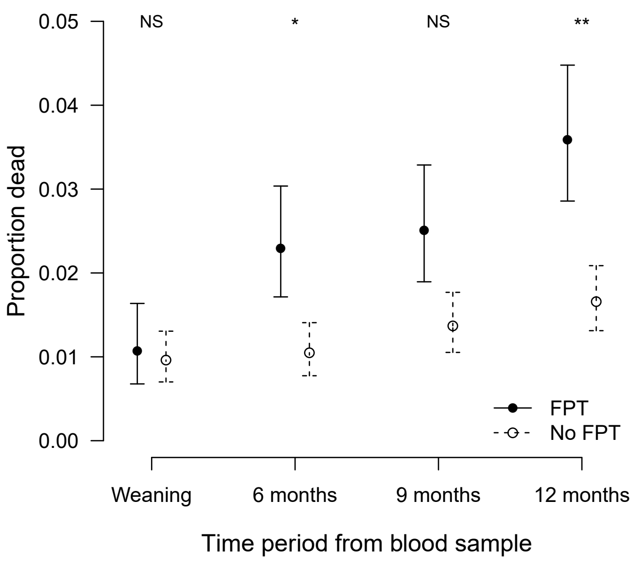
Internationally, FPT has been reported to increase the risk of mortality, disease and ill-thrift in dairy calves, and has been associated with long-term reductions in animal productivity[4].

Calves with FPT are more susceptible to diarrhoea, respiratory disease, septicaemia and enteritis[5,6]. FPT can also lead to under-development of the digestive tract and lower feed intake, resulting in poor growth and subsequent milk production[7].

In the recent New Zealand study, the effects of FPT (serum total protein [STP] less than or equal to 52 g/L at 1-7 days of age) on mortality, morbidity and body weight were investigated from birth until weaning. Of the 4000 calves, a subset of 2000 (representing 35 farms in the Waikato and Canterbury regions) were monitored until 12 months of age to ascertain the longer-term effects of FPT.

Animal health

Figure 1. The difference in the mortality between calves with failure of passive transfer (FPT; serum total protein less than or equal to 52 g/L) and calves without FPT at different ages. Vertical bars represent the 95 percent confidence limits.



Calves with FPT were twice as likely to have a farmer-recorded animal health event (e.g. diarrhoea) compared with calves without FPT. While specific diseases were not analysed separately, this relationship is similar to international findings[6,8,9]. In the Netherlands, calves with IgG concentrations less than 7.5 g/L were twice as likely to develop respiratory disease[9]. In North America[6], the odds of having a veterinary-diagnosed respiratory disease were two-fold greater in calves with serum IgG between 8.0 and 13.0 g/L compared with calves with serum IgG greater than 13 g/L. Therefore, if calf immunity via passive transfer can be improved, the risk of disease during the period from birth to weaning will be reduced.

Mortality

Surprisingly, mortality did not significantly differ pre-weaning. Rather, the heightened mortality risk for FPT-affected calves occurred between six to 12 months of age (Figure 1). By 12 months, 3.5 percent of heifers that had FPT as calves had died, whereas those without FPT had a mortality rate of 1.8 percent.

The increased risk of mortality in calves with FPT is consistent with findings from international studies[7]. In a 10-year study in North America, researchers measured the mortality risk of 3500 holstein replacement calves up to four months of age on a farm with endemic salmonellosis[5]. They reported that survival rates increased for each 5 g/L increase in TP concentration up to 60-64 g/L.

In particular, calves with TP concentration less than 40 g/L had a 4.6 times greater risk of mortality than calves with TP concentration of 60 g/L. Another North American study of 3300 Holstein heifer calves[7] indicated mortality risk between 0 and 6 months of age decreased when TP concentration increased from 40 g/L to 60 g/L, but there was no further improvement above 60 g/L.

It is logical that a young calf affected by FPT is more prone to succumbing to a serious disease. Yet the results from the New Zealand study indicate that calves with FPT had approximately double the odds of mortality from weaning up to 12 months of age but not before weaning.

A possible explanation is that the relationship between FPT and mortality may become more apparent post-weaning when heifers are often reared off the milking platform and, therefore, observed less frequently for detection and treatment of disease than during the pre-weaning calf rearing phase.

Body weights

FPT was associated with a significantly lower body weight at weaning, six, nine and 12 months of age. However, these differences were minor, ranging from 0.93 kg at weaning (88.77 kg v 89.70 kg) to 3.30 kg at 12 months (240.92 kg vs 244.22 kg). Possibly these results were influenced by the separation and preferential feeding of under-performing animals identified by the farmer or grazier.

Therefore, if the slower growing calves were those with FPT they would have been unknowingly preferentially fed by the farmer or grazier. This may mean that the effect of FPT on body weight may be larger than measured in this study.

Nevertheless, several international studies also indicate that FPT has only a small negative effect on body weight. In America, 2900 heifer calves were monitored from birth to three months of age on 19 commercial dairy farms[10]. Only a small (0.76 kg) decrease in body weight was reported for calves with FPT (STP concentration of less than or equal to 52 g/L)[10].

Researchers in Belgium observed that calves with STP concentration less than 7.5 g/L grew, on average, 79 g/day less than calves with STP concentration greater than 7.5 g/L, which equated to a difference of 1.8 kg from approximately three to six weeks of age. In a study conducted in Poland, calves with different serum immunoglobulin concentrations at 30 to 60 hours of life (less than five, 5-10, 10-15, or greater than 15 g/L) were monitored until one-year-old[8], with no difference in body weight gain detected for the first six months. There were some growth differences between certain groups and certain time points after six months but these were limited.

Conclusion

Failure of passive transfer in dairy calves is associated with greater susceptibility to disease and a higher mortality. The high prevalence of FPT detected in the New Zealand study indicates significant improvement is needed on some farms.

Management practices to ensure sufficient quality colostrum is consumed within the first 24 hours will lead to improved health and survival outcomes for replacement heifers.

For more information on colostrum management, visit dairynz.co.nz/animal/calves/calf-care/

Steps to prevent FPT

- Do you have a problem? Blood test 12 calves this season and find out.
- Newborn calves should only be fed first milking colostrum, no later than six to 12 hours after birth.
- Clean is key – do not leave colostrum without a preservative or refrigeration and a lid! If you would not drink it, do not feed it to the calves. The bacteria inhibit absorption of antibodies.

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Responses to Rumensin under pasture systems

The active ingredient in Rumensin is Sodium Monensin, a compound that alters the populations of specific micro-organisms in the rumen. These micro-organisms ferment carbohydrates and produce waste products known as volatile fatty acids (VFA). Therefore, adding Rumensin to the diet may alter the proportion of VFA produced[1] in the rumen.

John Roche, Jane Kay, **DairyNZ**

Recent questions on how Rumensin fits into a pasture-based system led us to revisit this review that was published in 2011. Implications of the research are still relevant today.

VFA are used by the cow for energy and the main VFAs produced in the rumen are acetate, propionate and butyrate. Rumensin targets the micro-organisms that ferment carbohydrates to acetate and butyrate, with the objective of increasing the micro-organisms that produce propionate[2].

This effect has been reported in multiple studies in laboratory analyses and in cattle[1], but the extent of the effect appears to be diet dependent, and results in grazing cows are inconsistent[2]. Because:

1. propionate results in greater glucose production by the liver and insulin by the pancreas,
2. acetate and butyrate are precursors for milk fat, and
3. Rumensin alters fatty acid and protein metabolism in the rumen.

Rumensin should, in theory, reduce milk fat, and increase milk protein and milk volume[1].

Key findings

- Rumensin alters the populations of certain rumen micro-organisms to favour increased propionate production. In theory, this should increase milk protein yield.
- However, milk production responses to Rumensin have been inconsistent and difficult to predict. An international review and New Zealand pasture-based studies indicate that average responses are approximately 15-25 g milk protein/cow/day, with responses expected to be greater as the digestibility of the diet declines (e.g. expected to be higher with summer pastures than with spring pastures).
- Research results indicate that Rumensin does not improve fertility, but reduces the incidence of bloat, and may reduce the risk of ketosis in some circumstances.

As methane production is a by-product of acetate production, Rumensin would also be expected to reduce methane production and increase the efficiency with which feed energy is converted to milk and meat.

Rumensin also interferes with ruminal protein degradation, a key factor in the production of the stable foam that causes bloat in ruminants. It is, therefore, reported to be an effective bloat preventative.

Although there is a sound basis for all these claims, there are inconsistencies in the reported effect of Rumensin on these important factors. Previous reviews[2,3,4,5] summarised the effect of Rumensin on dry matter intake (DMI), milk production, reproduction, body condition score and cow health, particularly during the transition period and early lactation. The likely implications of Rumensin use for New Zealand pasture-based systems are presented here.

Rumen fermentation

Rumensin alters the populations of micro-organisms in the rumen to increase the production of propionate and reduce the breakdown of protein.

Rumen micro-organisms ferment carbohydrates from the cow's diet to grow and they produce volatile fatty acids (VFA) as waste products. The cow has evolved to use these VFA as her primary sources of energy[1].

There are three main VFA: acetate, butyrate and propionate, and the ratio of these depend on the micro-organism populations in the rumen. Many factors can cause changes in rumen micro-organism populations and, as a result, the proportion of different VFA[1] produced.

1. Individual cows can have very different populations of micro-organisms[2] and, therefore, there is cow-to-cow variation (genetic effect) in rumen VFA production.
2. Diet influences the growth of different micro-organisms and can, therefore, influence VFA production. For example, fibre-based diets tend to direct rumen fermentation towards acetate production, while starch-based diets increase the proportion of propionate produced[1].
3. Feed additives (e.g. antibiotics[1], yeast cultures[6]) can selectively target certain types of micro-organisms, thereby altering rumen fermentation. For example, Rumensin selectively targets the micro-organisms that ferment carbohydrates to acetate, therefore favouring growth of micro-organisms that ferment carbohydrates to propionate.

In addition to fermenting carbohydrates, rumen micro-organisms break down dietary protein. This can result in wastage of dietary protein, with a requirement for more protein in the diet to offset this inefficiency.

Some of the micro-organisms that rapidly break down protein are sensitive to Rumensin. Therefore, supplementation with Rumensin can result in more high-quality dietary protein reaching the small intestine[1]. This is unlikely to be a benefit in pasture-based systems where protein rarely limits milk production.

However, its role in systems with greater use of low protein supplements (e.g. cereal grains, maize silage) or when pasture metabolisable energy or protein levels are low (e.g. drought) requires further evaluation.

Milk production

Milk production responses to Rumensin supplementation are variable and impossible to predict. On average, under New Zealand dairy farming conditions, cows supplemented with Rumensin produce 15-25 g more milk protein/cow/day than unsupplemented cows. This effect could be more when feeding poor quality forages and less when pasture quality is highest.

Milk production responses to Rumensin supplementation in pasture-based systems have been inconsistent. In an international review[2] of monensin supplementation, cows supplemented with Rumensin produced 2 percent more milk and 2 percent more milk protein than unsupplemented cows, with no effect, on average, on milk fat production. This is approximately equivalent to an additional:

- 20 g milk protein from cows producing 2.4 kg MS
- 18 g milk protein from cows producing 2.1 kg MS
- 15 g milk protein from cows producing 1.75 kg MS
- 12 g milk protein from cows producing 1.4 kg MS or
- 9 g milk protein from cows producing 1.0 kg MS.

At $8.70/kg milk protein ($6.50/kg MS), such increases would result in a milk revenue increase of $0.08-0.17/cow/day depending on MS yield/cow.

Results indicated that diet and stage of lactation had an effect on the response to Rumensin, with greater responses evident in pasture-based herds. Waghorn and co-workers[5] reviewed the research undertaken in pasture-based systems in Australia and New Zealand and, again, reported variable results.

Australian studies[7] reported a 30 g/cow/day increase in milk protein but these results were not achieved in subsequent trials on 18 dairy farms in Australia[8, 9], wherein Rumensin increased milk yield but did not increase milk protein yield. Waghorn and co-workers[5] quoted unpublished experiments in New Zealand and Australia that reported an average increase of 40 g MS/cow/day across the dataset, but only an increase of 23 g MS/cow/day in those studies undertaken in New Zealand.

The reason for the inconsistency in milk production responses to Rumensin is not clear, but it may reflect a negative effect of Rumensin on DMI, as reported in many studies[2], or an interaction with diet quality.

Waugh and co-workers[10] reported that the milksolids response to Rumensin increased as diet quality decreased. Cows supplemented with Rumensin produced +70 g MS/cow/d compared with -70 g MS/cow/d when metabolisable energy (ME) of the diet was 11.6 MJ/kg DM compared with 12.1 MJ/kg. These results suggest that the best milk production response to Rumensin may be during a dry summer, when pasture quality declines, while the lowest response is likely during spring, when pasture quality is highest. However, further research is required to determine the cow response under different diets.

Reproduction and health

Most research studies indicate no effect of Rumensin on fertility. Rumensin is an effective bloat prevention agent and reduces the concentration of ketone bodies in blood, but its effect on the incidence of ketosis is inconsistent.

Reproduction: because of Rumensin's effect on rumen fermentation and consequential effects on blood glucose and ß-hydroxy butyrate, it has been proposed that Rumensin may improve reproduction. A comprehensive review of world literature indicated no effect of Rumensin on either first service conception rate or days to pregnancy[4].

These results are consistent with a major Australian experiment involving more than 1000 cows across 12 farms[9], which showed a lack of effect of Rumensin on fertility.

Bloat: bloat is a serious disorder of cattle and sheep involving a severe distension of the animal's rumen[11]. It generally involves cows grazing lush, leafy and often legume-dominant pastures during spring and autumn, and is the result of a gas-filled foam at the top of the rumen that the cow is unable to release (eructate)[11].

The actual cause of foamy bloat is not completely known, but products of the breakdown of proteins in legumes and grasses have been implicated[11]. Research undertaken in grazing cows in New Zealand and Australia indicates that monensin can reduce the incidence of visible bloat and bloat deaths by approximately 80 percent[7,11]. However, no one method of prevention will be 100 percent successful against a large bloat challenge, and farmer vigilance and experience is probably the most important factor in bloat prevention.

Ketosis: Rumensin has been proposed as a strategy for reducing the risk of ketosis. Ketosis occurs when there is an imbalance between energy demand and energy supply, and cows are not able to fully utilise mobilised body fat[1]. In these situations, intermediates in the breakdown of fat accumulate and cause ketosis. The disorder occurs when cows undergo a sudden reduction in intake (type I ketosis) and is particularly prevalent in fatter cows after calving, particularly when energy demands are high in early lactation (type II ketosis). It can also occur if cows are fed poor quality silages (silage ketosis).

One of the factors contributing to ketosis is a lack of propionate to stimulate the full breakdown of mobilised fat[12]. Therefore, one of the prevention strategies for ketosis is to provide dietary ingredients that shift ruminal fermentation towards more propionate production and increase the liver's production of glucose. In a comprehensive review[3], authors concluded that monensin use in lactating dairy cattle reduced blood ketone body concentrations by 13 percent and circulating concentrations of fatty acids by 7 percent. Monensin also increased plasma glucose concentrations by 3 percent.

These results imply that Rumensin use in early lactation may reduce the risk of ketosis. However, what would be regarded as high blood ketone levels in housed cows fed mixed rations, (i.e. levels of ß-hydroxy butyrate greater than 1.2 mmol/L) is not indicative of ketosis or reduced performance in grazing dairy cows, according to recent New Zealand research.

Blood ß-hydroxy butyrate concentrations tend to be higher in grazing cows[13] because of the greater production of butyrate in the rumen, and these are not associated with reduced performance. Therefore, it is questionable as to whether Rumensin would reduce the risk of ketosis in a pasture based system. Furthermore, feeding cows starch-based feeds in early lactation, which reduced blood ß-hydroxy butyrate, had no effect on reproduction or six-week in-calf rate in on-farm studies in New Zealand[13].

Conclusions

Sodium monensin, the active ingredient in Rumensin, alters the populations of micro-organisms in the rumen to increase the production of propionate and reduce the production of acetate and butyrate. In theory, this should increase blood glucose and, blood insulin,and reduce ß-hydroxy butyrate. Based on these changes, Rumensin should, therefore, increase milk protein, reduce milk fat, reduce the risk of ketosis in early lactation and improve fertility.

However, in reality, the response to Rumensin in grazing cows is inconsistent, with

- both positive and negative effects on milk production (on average 15-25 g milk protein/cow/day)
- variable responses in blood metabolites and, therefore, in Rumensin's effect on the risk of ketosis, and
- on average, no effect on reproduction.

Rumensin is an effective bloat control strategy, although, it must be recognised, that no one strategy will offer 100 percent protection against bloat.

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Proving freedom from TB: the pathway to eradication

Bovine tuberculosis likely arrived in New Zealand in the mid-19th century when cattle and deer were first introduced. The brush-tailed possum arrived shortly afterwards, but it was not until almost a century later that the link was identified between infected possum populations and the failure to control tuberculosis (TB) in certain regions.

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Graham Nugent, **Landcare Research**

This disease's establishment in the possum population led to TB becoming a serious threat to the New Zealand economy. With possums being widespread and abundant (population currently estimated at approximately 30 million) and the main wildlife vector of TB, the control and eradication of this disease might be considered audacious, but it is possible.

The national TB Plan aims to reach freedom from bovine TB in New Zealand's livestock by 2026, achieving freedom from disease in possums by 2040 and eradication of TB from New Zealand livestock and wildlife by 2055.

The plan aims to produce 'Proof of Freedom', evidence of the probability that bovine TB has been eliminated from an area or a population of wildlife, such as possums or ferrets. It is not a novel concept, being based on the principles of epidemiological surveying to monitor diseases in populations. For TB, the underpinning data and theory of the Proof of Freedom

Key findings

- Possums are the main wildlife vector (transmitter) of bovine tuberculosis (TB) in New Zealand.
- Proof of Freedom is evidence that an animal population or area is free of disease based on the probability that the disease has been eliminated.
- The TB Proof of Freedom Model uses surveillance of wildlife and livestock, as well as pest control history, to ascertain if TB has been eradicated.
- This model has shifted thinking and action from possum control to eradication of TB from possums. This is the only means by which eradication of TB in the national dairy herd can be achieved.

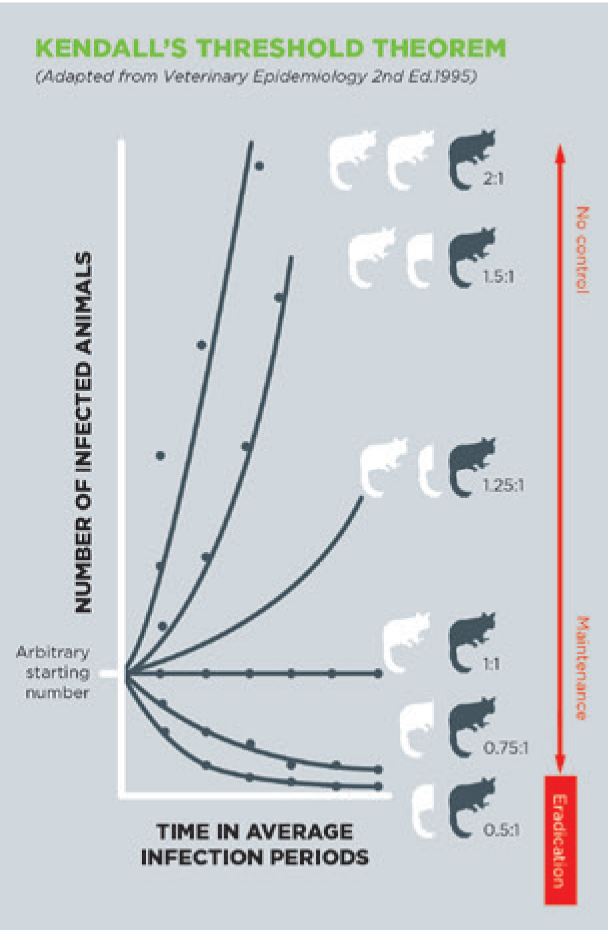
model includes Bayesian probability and classical sampling methodology, TB epidemiology, population dynamics, home-range utilisation and movement patterns of possums and sentinel species, and the trap-ability or detectability of possums (see footnote 1 for definitions).

Proof of Freedom

TBfree's declaration of Proof of Freedom is based on a calculated probability that there is less than a 5 percent chance the possums in the area surveyed are still infected, and that the disease cannot persist at that level.

The general conceptual theory is identical for mammalian pests all over the world. International theory[1] as used to describe the concept of rabies eradication from foxes in Europe applies equally to TB in possums in New Zealand. It suggests that, if we reduce a population of possums below a particular threshold density, which for New Zealand is about one possum per 2ha, infected animals die before they can transmit disease to susceptible animals (Figure1).

Figure 1: Kendall's Threshold Theorem predicts that spread of a disease will rapidly increase if an infected animal infects more than one other before it dies. Conversely, a disease will rapidly decrease if an infected animal infects less than one other in its lifetime. The ratio of white to black possums represents the probability of transmission, e.g. at a ratio of 2:1, an infected possum (black) is likely to transmit TB to two other possums.



If the reduction in the infected possum population covers a large enough land area to limit immigration of a susceptible population, the disease will die out.

The theory sits at the foundation of the National Pest Management Plan (NPMP) to eradicate TB, alongside the knowledge that possums are the main vectors (transmitters) of TB between wildlife and farmed livestock.

Since the peak of TB herd infection in 1994, when 1700 cattle and deer herds were known to be affected, the TBfree programme has reduced the number of infected herds to fewer than 50 (Figure 2).

Assessment tool

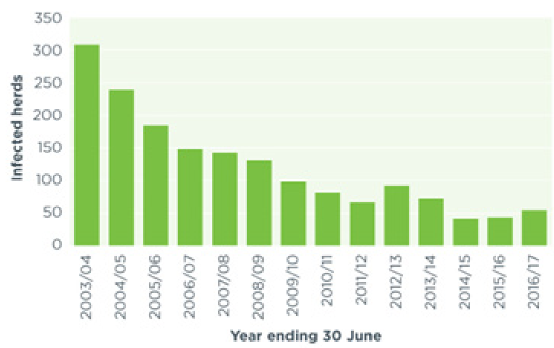
The model is a tool to assess the probability that bovine TB has been eliminated from possums in a local area and was first used in 2012. Manaaki Whenua – Landcare Research scientists developed the tool in collaboration with the veterinary epidemiologists who designed the TBfree programme, now managed by OSPRI.

The general approach is that, once the model predicts probability-of-freedom (pFree) at an agreed trigger level (generally greater than 80 percent probability that TB has been eradicated), surveillance is initiated to provide evidence that TB is indeed absent from a possum population (Figure 3).

Surveillance data can be derived not only directly from possums, but also from surveys of 'sentinel' species such as deer, pigs and ferrets. The surveillance effort continues until a pre-defined pFree (so-called 'stopping' probability) is achieved.

At that point, possum TB freedom is declared. There are a series of 'stopping' probabilities, based on the risk and consequence of a TB freedom declaration being incorrect.

Figure 2: The number of New Zealand cattle and deer herds infected with bovine tuberculosis.



1. Baysian probability: Is the expected probability based on the current state of knowledge, rather than being based on frequency of an outcome.
Epidemiology: The study and analysis of disease incidence and distribution to determine disease risk factors.
Population dynamics: Population size and age, birth and death rates, and net migration in and out of a region
Sentinel species: Other species that may carry TB, such as wild deer, pigs and ferrets.

Simply, the greater the risk and consequence of a false declaration of freedom, the higher the 'stopping' probability is set, therefore requiring a greater surveillance effort to achieve true TB freedom.

When the set level of confidence (probability of eradication) is achieved, an area can be reclassified from being a Vector Risk Area (VRA; areas where TB is found in wildlife) to being a Vector Free Area (VFA). At this point it can be declared that possum populations in an area have been below the level at which the disease can be maintained for a long enough period to confidently define the area as TB-free.

Vector-free areas

Since the beginning of the TBfree programme, more than 1.8 million hectares of Vector Risk Area have been declared vector-free. However, at 1 July 2017, a further 7.9m hectares remains to be declared TB-free.

It took almost a decade to develop and adopt the Proof of Freedom framework as a major new paradigm in TB management. This is partly because it takes time to 'sell' new concepts to users, but mainly because the need became a priority when local TB eradication became a formal management goal. There is now a strong focus on refinement and extension of the framework and associated software.

An example is the development of new theory by Landcare Research to allow inclusion of data from livestock TB testing and slaughterhouse inspections, which will give confidence that TB is absent from possums in particular farmed areas. Currently, that information is used only as supporting information and does not affect the calculated probabilities of TB freedom.

Other research is exploring when to start TB surveillance, with the potential to greatly shorten the cost and duration of possum control required for TB freedom.

By providing an objective measure for comparing progress between areas, the Proof of Freedom tool has enabled a quantum shift in thinking from possum control to the elimination of TB from possums. This is the key objective of TB management and is the only means by which we can successfully eradicate TB from our farmed livestock.

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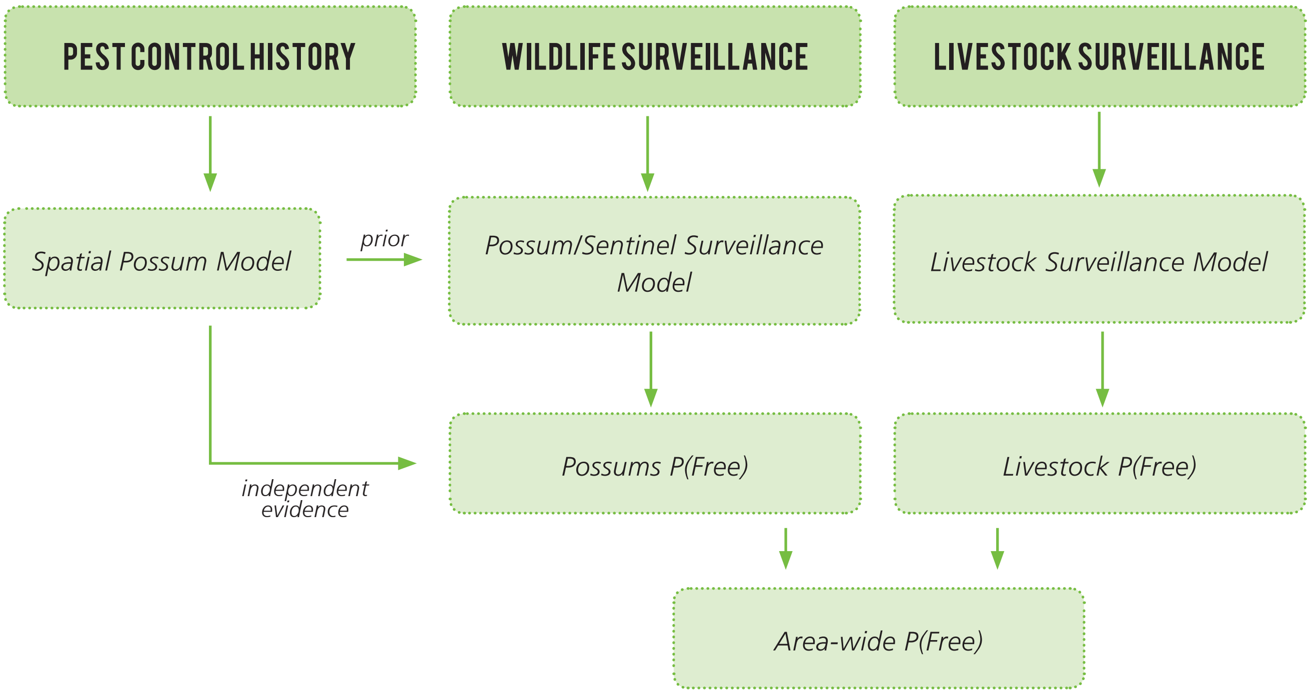
Figure 3: The Proof of Freedom Model uses evidence from pest control history and wildlife/livestock surveillance to determine the probability that TB has been eradicated from an area.



Science snapshot

Chicory and plantain can improve production when ryegrass quality is poor[1]

Field trials have shown that chicory and plantain (herbs), grow more feed of better quality than perennial ryegrass during dry summers and autumns. But what about the effect of these herbs on milk production and urinary nitrogen (N)?

In a late-summer/autumn DairyNZ study, cows were fed these herbs at either 20 or 40 percent of their total diet, with the remainder being a ryegrass-based pasture of moderate (~10.5 MJ ME/kg DM) or poor (~9.6 MJ ME/kg DM) quality. The production performance of these cows was compared with others fed pasture only.

Milk solids and milk volume were similar between the cows fed the herbs with moderate quality ryegrass and those fed ryegrass only. However, cows fed herbs ate about 1kg less DM/day than those fed ryegrass only to achieve that similar milk production.

When ryegrass quality was poor, the cows fed herbs ate more and produced 17 percent more milksolids. The better milk response to herbs was due to the greater intake and quaity of the herbs compared with the ryegrass (average 12 vs. 9.6 MJ ME/kg DM, respectively).

Including herbs at up to 40 percent of the diet had little effect on milk composition. Therefore, in regions prone to poor ryegrass growth and quality in dry summers and autumns, feeding chicory or plantain on-farm is a viable option for maintaining milk production.

Feeding these herbs may also provide environmental benefits. Urinary N concentration was reduced by up to 38 percent lower when herbs were fed. This is because these herbs have a lower N concentration compared with ryegrass, meaning cows fed herbs consume less N.

Also, the lower DM content (conversely, greater water content) of herbs compared with ryegrass may mean more and more dilute urine is excreted. Because the amount of urinary N from cows is related to the amount of N leached from soil, research is underway to understand how much herb is required in the diet to reduce N leaching, and how to best incorporate these herbs in the farm system.

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