Calf wastage - how big an issue is it?

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What is calf wastage
Breeding female cattle in north Australia contribute best to business success by producing a heavy weaner each year at the first weaning round. This maximises increase in value by the cow unit over the year, generally from mid-year to mid-year. The ability to cycle in both maiden heifers and lactating cows is the primary limitation to achieving this. Wastage of a calf at any stage between conception and weaning also substantially limits fertility and value increase at a herd level. Embryo loss may result in later calves as cows re-conceive; the calves produced are smaller at weaning and have to be weaned later into the dry season. Late calf wastage usually results in breeders missing a calf for the year. Late calving often also results in failure to cycle, thus cows rear a calf in the subsequent year.

Prevalence and causes
Embryonic mortality is loss between fertilisation and Day 45 of pregnancy. Foetal mortality occurs from the 45\textsuperscript{th} day of pregnancy to the commencement of birth/parturition. Neo-natal mortality occurs when calves die or exhibit illness within 2 days of birth resulting in death within one week of birth. Post-natal mortality occurs between the neo-natal period and weaning. It is difficult to define prevalence or incidence of losses at various stages, as well as causes, and reports of losses are often simply between confirmed pregnancy and weaning.

Burns \textit{et al.} (2007) reviewed reports from the mid-1960’s to 2005 of prevalence of pregnancy to weaning losses ranging from 2% to 61% for extensively-managed north Australian herds. Recent anecdotal evidence is that losses from first-lactation females are typically 20% with no cause identified. For example, in a recent NW Queensland study, unexplained losses in this period averaged 17% in a well-managed herd.

Pre-natal loss
Embryonic mortality rates of 25% are considered normal in extensively-managed beef cattle. At least 80% of embryonic mortality occurs between Day 8 and Day 18 after fertilisation Burns \textit{et al.} (2007), which usually results in seamless transition to the subsequent oestrus cycle. Combined with fertilisation failure rates of 85-90%, a pregnancy rate per cycle of 65-70% is expected in healthy cattle. Foetal loss averages no more than 3% in healthy cattle. However, the Burns \textit{et al.} (2007) review reported foetal losses in north Australia, ranging from 1% to 17% between the mid-1960’s and 2005.

In north Australian herds, Burns \textit{et al.} (2007) reported that primary causes of pre-natal loss include:

- \textit{Campylobacter foetus} subspecies \textit{veneralis} (vibrio) and \textit{Tritrichomonas foetus}. The primary evidence of these diseases is delayed conception patterns, resulting in smaller weaners. Prevalence of vibrio is suspected to be high across north Australia, but probably low for trichomoniasis.

- Bovine pestivirus. This disease appears to be endemic in the majority of large north Australian herds. Its impact in causing embryo and foetal loss, and mortalities in animals born as persistently-infected (PI) may be highest in herds where animal management is better, (ie, create small-herd situations) and naïve age groups are often created. It is within these naïve groups that significant pre-natal loss occurs when exposed to PI cattle.

- \textit{Neospora caninum}. Has been suggested as a significant cause of losses, but despite it being present in most herds with a prevalence level of 20% not unusual, and it clearly being a cause of loss in specific intensively-managed herds, there is no evidence to date that it causes significant loss in extensive north Australian herds.
• Stress may be the most significant cause of non-infectious prenatal loss. Causes of stress include handling, heat, and mal-nutrition. Stress-related losses may be prevalent in most herds, but incidence is only likely to be significant where specific conditions are created.
• Impaired female reproductive tract environment and function related to endocrine (hormonal) and immunological dysfunction may also cause losses that could be related to diet, the environment, and genetic predisposition. The prevalence and incidence of these losses have not been quantified.

Neonatal loss
Burns et al. (2007) reviewed reports from the mid-1960’s to 2005 of neo-natal losses in north Australia ranging from 2% to 12%. Significant causes of losses included:
• Stress due to nutrition or heat, eg, heatstroke/hyperthermic shock in non-adapted genotypes
• Predators, especially wild dogs. Losses can exceed 20% and are proportional to the dog population.
• Lack of milk to the calf because of either poor milk production, teat and udder abnormalities, or accidental mismothering. These problems may be caused by malnutrition, genetic predisposition (eg, bottle teats or very large udders), environmental conditions, handling/mustering, immaturity of the dam, and twins where usually only one survives.
• Calf abnormality or weakness associated with abandonment by the dam. Affected calves are often of low birthweight.
• Leptospirosis spp are known to cause late abortions and weak calves. A minority of species are considered a problem. Though the organisms are ubiquitous, the incidence of significant loss confirmed as due to leptospirosis in extensive areas is low. Vaccination also prevents zoonoses.
• Dystocia occurs in all breeds, including Brahmins, and primarily in 2-year-olds. The primary problem is foeto-pelvic disproportion (Norman 2006). Control measures include avoiding matings that cause big calves, preventing heifer obesity at calving and most importantly, keeping heifers growing at all times, especially during the first half of pregnancy. Under-nutrition at any stage can retard pelvic growth. Under-nutrition in early pregnancy may increase size and efficiency of the placenta, which disproportionately increases foetal growth when adequate nutrition is restored. In a recent north Queensland study, dystocia did not occur when preventative management was implemented; however, 5-10% of heifers experienced dystocia when positive action was not taken.
• Misadventure, which is sporadic.
• Unknown causes. Unfortunately this is consistently the primary cause given for loss, even in detailed studies.

Post-natal loss
Burns et al. (2007) reviewed reports from the mid-1960’s to 2005 of post-natal losses in north Australia ranging from 0% to 53%. Main causes of losses included:
• Bovine pestivirus as discussed earlier.
• Botulism. There are many anecdotal reports of suckling calf botulism deaths, usually where cows were unvaccinated.
• Arboviruses. The primary viruses are Akabane and Bovine ephemeral fever. Both are ubiquitous and potentially have greatest effect following a series of dry years when heifers do not gain exposure, thus immunity. The effect of BEF on calf wastage in north Australia remains unquantified.
• Malnutrition, including deficiency of diet components such as Vitamin A, which was identified as the primary cause of calf loss exceeding 40% in one NW Queensland herd following a series of dry years.
• Predators, misadventure and environmental stresses as for neo-natal losses.

Benchmarks
From the above, and other reported data for north Australia, benchmark levels for reproductive wastage elements are proposed as: 25% of embryos, 3% of foetuses, 5% of neonates, and 1% of the remaining calves.
The review of Burns et al. (2007), reinforced by recent observations, indicates that many herds are not achieving the benchmark levels indicated above. Though specific incidence of losses has not been quantified for north Australian beef herds, all indicators are that they are not low. It highlights substantial opportunities in north Australian herds to reduce reproductive wastage, with the most significant opportunities being to:

- increase the rate of established pregnancy per cycle in breeding female cattle, with specific emphasis on early embryonic mortality, and
- reduce neo-natal mortality.

**Financial impact**

Foetal and calf wastage affects business efficiency by reducing the number and size of calves at weaning as a function of the number of breeding females retained in the previous year, i.e., it reduces or eliminates the increase in value achieved by a breeding animal over a year.

Foetal, neo-natal, and post-natal losses all generally result in failure to rear a calf to weaning within the year. Cows may re-conceive after embryo or early foetal loss, and still raise a calf, though it will be a small weaner and the delayed calving results in the cow having little chance of rearing a calf in the next year. Figure 1 provides an example of how weanings may be affected in this way.

Figure 1. Predicted calves/100 heifers after 3 months mating to _Campylobacter_-infected bulls (grey columns; ~40% pregnant per cycle and weaning 79% at 203 kg) in a north Queensland herd compared to potential calves in the absence of disease (black columns; ~70% pregnant per cycle and weaning 84% at 214 kg).

Table 1 shows estimates of the financial effect of reproductive disease and calf losses in a representative NW Queensland cattle herd using BREEDCOW modelling. Model input was generated by detailed analysis of available data on reproductive function in breeding herds. Vibrio was estimated to reduce weaning rates in heifers by 5% and their progeny weights by 11kg. Mid-pregnancy to weaning losses were taken to increase from 10% to 18% in heifers where high calf loss is occurring. The effect in cows was taken as 20% of that in heifers. The effects were taken as additive when they occurred together. Steers were sold at weaning. Surplus heifers are sold at 2.5 years of age.
Table 1. BREEDCOW estimates of the financial effect of vibrio and/or high calf loss in a representative NW Queensland 3,000 adult equivalent beef herd.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>No vibrio &amp; normal calf loss</th>
<th>Vibrio present</th>
<th>High calf loss</th>
<th>Vibrio &amp; high calf loss</th>
</tr>
</thead>
<tbody>
<tr>
<td>2-year-old heifers mated</td>
<td>646</td>
<td>640</td>
<td>632</td>
<td>625</td>
</tr>
<tr>
<td>Total females mated &amp; kept</td>
<td>1570</td>
<td>1592</td>
<td>1603</td>
<td>1627</td>
</tr>
<tr>
<td>Heifer weaning rate</td>
<td>84%</td>
<td>79%</td>
<td>76%</td>
<td>71%</td>
</tr>
<tr>
<td>Heifer pregnancy-weaning loss</td>
<td>10%</td>
<td>10%</td>
<td>18%</td>
<td>18%</td>
</tr>
<tr>
<td>Cow weaning rate</td>
<td>84%</td>
<td>83%</td>
<td>82%</td>
<td>81%</td>
</tr>
<tr>
<td>Cow pregnancy-weaning loss</td>
<td>10%</td>
<td>10%</td>
<td>12%</td>
<td>12%</td>
</tr>
<tr>
<td>Total calves weaned</td>
<td>1318</td>
<td>1306</td>
<td>1290</td>
<td>1276</td>
</tr>
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<td>1318</td>
<td>1306</td>
<td>1290</td>
<td>1276</td>
</tr>
<tr>
<td>Weaner size (kg)</td>
<td>214</td>
<td>203</td>
<td>214</td>
<td>203</td>
</tr>
<tr>
<td>Reproductive efficiency(^a)</td>
<td>180</td>
<td>167</td>
<td>172</td>
<td>159</td>
</tr>
<tr>
<td>Capital value of herd</td>
<td>$1,522,655</td>
<td>$1,521,379</td>
<td>$1,523,600</td>
<td>$1,522,367</td>
</tr>
<tr>
<td>Net cattle sales</td>
<td>$621,713</td>
<td>$610,637</td>
<td>$613,263</td>
<td>$601,935</td>
</tr>
<tr>
<td>Direct costs</td>
<td>$53,000</td>
<td>$52,796</td>
<td>$52,606</td>
<td>$52,372</td>
</tr>
<tr>
<td>Gross margin for herd</td>
<td>$568,713</td>
<td>$557,841</td>
<td>$560,657</td>
<td>$549,563</td>
</tr>
<tr>
<td>GM per adult equivalent</td>
<td>$190</td>
<td>$186</td>
<td>$187</td>
<td>$183</td>
</tr>
</tbody>
</table>

\(^a\) kg weaned/cow retained

This analysis indicates that for every 1,000 cows mated and retained, business profit is reduced by:
- >$600 for each percentage unit in pregnancy-weaning loss incurred.
- >$200 for each percentage unit in embryo loss incurred.

In both scenarios presented in Table 1, the gross margin was reduced by $3-$4 per Adult Equivalent.

This estimate does not account for other substantial secondary benefits that may also accrue from reducing calf wastage, eg the opportunity to implement more effective genetic improvement programs.

Table 2 shows suggested impacts of vibriosis and trichomoniasis in when it is assumed that one third of the north Australian herds is affected, and of pregnancy to weaning loss when possibly 20% of cattle are in herds that experience high losses.

**Table 2. Possible impact of reproductive disease and pregnancy to weaning loss in the north Australian cattle herd.**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Derivation</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>North Australian herd</td>
<td>AE</td>
<td>12M adult equivalents</td>
</tr>
<tr>
<td>Cows mated and kept</td>
<td>AE(*0.5)</td>
<td>6M</td>
</tr>
<tr>
<td><strong>Impact of vibrio &amp; trichomoniasis</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percent of cattle in affected herds</td>
<td>Assumption</td>
<td>33%</td>
</tr>
<tr>
<td>Loss within a herd</td>
<td>Embryo loss +30%(*200/1000)</td>
<td>$6/cow mated &amp; kept</td>
</tr>
<tr>
<td>Cost to north Australian herd</td>
<td>$6(*0.5/3)</td>
<td>$1/cow mated and kept</td>
</tr>
<tr>
<td><strong>Impact of high pregnancy-weaning losses</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percent of cattle in affected herds</td>
<td>Assumption</td>
<td>20%</td>
</tr>
<tr>
<td>Loss within a herd</td>
<td>Foetal and calf loss +8%(*600/1000)</td>
<td>$5/cow mated &amp; kept</td>
</tr>
<tr>
<td>Cost to north Australian herd</td>
<td>$5(<em>0.5</em>20%)</td>
<td>$0.50/cow mated and kept</td>
</tr>
</tbody>
</table>

<p>| | | |</p>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>$3M</td>
</tr>
</tbody>
</table>
Estimates in Table 2 indicate that there is significant opportunity to improve business performance by identifying and implementing solutions to the causes of embryo, foetal and calf wastage. Modelling developed by the CRC for Cattle and Beef Quality indicates that the benefit to the community of improving beef business performance is three times that accruing to producers (Farquharson et al. 2003).

**Current recommendations to minimise losses**

There are currently many recommendations which may significantly reduce embryo, foetal and calf wastage. These are summarised as follows:

1. **Cow selection**
   - Select female cattle for future breeding at all ages if they rear calves to weaning, and do not have attributes that may contribute to calf loss, eg, bottle teats, poor maternal temperament

2. **Mating management**
   - Use bulls identified as fertile by a breeding soundness evaluation prior to initial mating at least.
   - Manage time of mating to achieve calving when nutrition is adequate, and when cows and calves are able to tolerate prevailing climatic stresses (temperature extremes, inclement weather).

3. **Metabolic status management**
   - Nutrition
     - Ensure adequate feed and water quality, quantity and access.
     - Prevent hypovitaminosis A. Specific practical strategies are yet to be developed and tested, but may include targeted supplementation over the latter half of the usual growing season in years when availability of green feed is limited.
   - Minimise handling stress, especially in the latter half of pregnancy.

4. **Disease management**
   - Use ultrasound examination of ovaries in conjunction with pregnancy diagnosis and animal description to differentiate malnutrition and infectious disease as the primary potential causes of delayed pregnancy or low pregnancy rates, especially in heifers.
   - Diagnose prevailing reproductive disease. Basic sampling for infectious disease if this is considered a probability includes:
     - Vaginal mucus for a *Campylobacter foetus* subsp. *venerealis* ELISA antibody test.
     - Preputial secretions for a real-time-PCR test of both *Campylobacter foetus* subsp. *venerealis* (McMillen et al. 2006) and *Tritrichomonas foetus* (Lew et al. 2006) from up to 20 bulls.
     - Serum from up to 20 heifers for pestivirus antibody.
   - Use vaccination or allied strategies where significant loss due to a specific disease may occur
     - Pestivirus. Use strategic sampling of management groups to assess previous exposure or potential presence of persistently-infected (PI) carriers in a herd. Vaccinate naïve groups of females prior to mating if there is any chance of exposure to a PI during mating.
     - Campylobacteriosis. Vaccinate heifers prior to first mating. Vaccinate bulls, starting early in life to establish immunity before homosexual transmission occurs.
     - Leptospirosis. Implement the recommended vaccination program where this disease is known to cause reproductive wastage, or there is a high probability that it may. Commence vaccination in calves at the earliest practical time.
     - Clostridial diseases. At marking, 5-in1 (especially to prevent tetanus) and botulism vaccines should be given to suckling calves in areas where these diseases are endemic.

5. **Dystocia management**
   - If possible, only mate yearlings that have reached a target mating weight (Fordyce 2006).
   - Select sires and females with traits indicative of calving ease; eg: when available, use EBVs for calving ease and short gestation length; mate older heifers; avoid cross-breeding of yearlings.
• Ideally, maintain growth and forward body condition in pregnant yearlings, especially during the first half of pregnancy; however, avoid obesity at calving.
• Supervise calving and provide early assistance if required, especially of 2-year-olds.

6. Predator management
• Control predators where there is a risk that these may cause significant calf loss.

The future through R&D
There are a number of recent, current, and planned projects in north Australia that will provide outcomes to limit embryo, foetal and calf loss. They include the following:
• Sperm chromatin (DNA) instability and reproductive wastage. Imperfect condensation of DNA into the head of sperm has been shown to cause significant embryo mortality in other species and there is some suggestion that a similar problem occurs in cattle. A recent study in Queensland showed there is a relatively low prevalence in bulls of levels of sperm chromatin instability that may be of concern.
• Improved diagnosis of reproductive disease in cattle. Highly-sensitive and highly-specific PCR tests were developed to detect the infectious agent in vibriosis and trichmoniasis. This will greatly enhance future investigation of these diseases. In the short term they are useful for herd diagnoses, to prevent potential impact of these diseases in single-sire mated herds, and ensuring the infectious agents do not contaminate frozen semen (Lew et al 2006).
• Impact of pestivirus and Neospora on beef herds. This is a current study where the disease impacts are being monitored in small, medium and large herds primarily in Queensland and NSW. A project report will be prepared in 2007.
• CRC for Beef Genetic Technologies. Female selection strategies will be more targeted when the repeatability and genetics of calf wastage is determined as part of current herd studies. The inheritance of sperm attributes that may affect embryo survival may extend from other parallel studies.
• Impact of BEF on north beef herds. A current study is attempting to measure the effect of BEF vaccination on fertility of heifers, including calf wastage, in north Australian herds.
• Diagnosis and prevention of hypovitaminosis A. This is an on-going study within the DPI&F in Queensland following recent diagnosis of substantial calf losses caused by this condition. There is a linked investigation of a newly-described condition which presents as neo-natal haemolysis.
• Levels and causes of foetal and calf loss in north Australian herds. A four-year prospective epidemiological study of region-, property-, mob-, and animal-level factors affecting selected measures of fertility is in the planning stages. Using selected representative herds, achievable levels of reproductive performance and the primary influencing factors will be defined and quantified to form the basis of decision support tools to profitably improve fertility of north Australian breeding herds. Methods to monitor reproductive performance will also be determined.

Basic studies have been proposed that may also lead to practical solutions to high embryo, foetal and calf loss. These include study of the mode of action of nutritional, environmental and genetic factors that influence ova and embryo viability and acceptance by the uterus, and development of remedial strategies for negative factors where possible.

References


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