Reproductive productivity of farmed red deer: a review

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Abstract

Reproductive wastage is a significant economic burden on the New Zealand deer farming industry. This paper reviews existing knowledge and gaps in our knowledge on reproductive losses. Identification of the true sources of wastage is clouded by confusing terminology, occasional misuse of statistics and insufficient monitoring at key stages of the reproductive cycle. The principal sources of wastage known to influence national productivity include high incidences of puberty/conception failure in R2 hinds and post-natal calf mortality. However, there is growing concern over the possible contribution of foetal death (abortion/resorption) to overall reproductive wastage, as it is often difficult to reconcile differences between scan pregnancy rates and calf weaning rates with observed post-natal mortality incidences. Foetal death has been largely dismissed previously due to insufficient data but efforts are currently underway to identify the true incidences of such losses, particularly amongst R2 hinds. This principally involves repeated ultrasonographic scanning of hinds over the entire course of pregnancy. Recent evidence indicates >10% foetal loss amongst R2 hinds on some farms, and investigations are underway to identify possible causal links with biotic factors. This review also identifies the potential role of disease in overall reproductive wastage.

Key words*: red deer, wapiti, reproduction, productivity, puberty, foetal wastage*

Introduction

Reproductive ‘wastage’ (i.e. lost reproductive potential) is a significant economic burden on the New Zealand deer farming industry and improvement of reproductive outputs features prominently in the industry’s productivity strategy.

The extent of wastage among breeding herds is often clouded by inappropriate terminology and misuse of statistics. This leads to confusion about the true sources and causes of wastage. For example, while many producers refer to ‘calving rate’ as their ultimate measure of hind output, they are often actually referring to ‘weaning rate’. The former refers to calves born per capita of hinds present (at mating or calving) while the latter refers to surviving calves weaned per capita of hinds present. There is often the assumption that ‘dry’ hinds failed to conceive when they may have actually lost calves at or after birth. This is further complicated when management strategies include the culling of non-pregnant hinds at or shortly after ultrasound scanning, thus skewing the stated ‘calving rate’ of the remaining herd relative to the original base herd at mating.

Thus we have a scenario where there is a paucity of interpretable data and confusion about true sources of reproductive wastage across our farmed deer herds. Ignoring at this stage any measure of conception incidence due to our inability to monitor embryonic wastage in deer, we propose the following standardised terms and definitions to provide clarity in this review.

(1) **Early pregnancy rate**: Percentage of all joined hinds scanned pregnant by rectal ultrasonography within the first trimester of pregnancy (i.e. May-July).

(2) **Late pregnancy rate:** Percentage of all joined hinds scanned pregnant by flank ultrasonography within the third trimester of pregnancy (i.e. September-October).

(3) **Foetal wastage**: Difference between 'Early pregnancy rate' and 'Late pregnancy rate' that provides an (under-) estimate of foetal mortality (abortion/resorption).

(4**) Calving rate**: Percentage of all hinds joined delivering calves.

(5) **Calf mortality rate**: Percentage of all calves born that die within 3 months of birth (i.e. March; pre-rut weaning).

Comment [CB1]: Otherwise it reads 'born dying' which is a tad odd

(6) **Weaning rate**: Percentage of all joined hinds presenting live calves pre-rut (March).

Comment [CB2]: The timing difference between (5) and (6) isn't desperately clear, as both reference March and pre-rut. Can you distinguish (5) better?

Clearly, the 'hands-off' nature of deer farming precludes the systematic collection of data to accommodate all these parameters. However, common statistics measured across many commercial herds include early-scan pregnancy rate and weaning rate (again recognising that this is often calculated after the cull removal of scanned non-pregnant hinds from the calving herd).

Limitations in our knowledge of reproductive wastage include the extent of true ovulation failure, conception failure, pre-implantation embryo loss, foetal loss and true incidences of peri- and post-natal calf mortality. However, these are clearly the primary components of reproductive wastage (this review will not cover lactational outputs that relate to calf growth but accepting that lactational insufficiency could also be viewed as wastage). Retrospective calculation of their relative importance to the overall reproductive productivity of each herd is dependent on accurate and unbiased assessment of the aforementioned parameters, and in many cases will require additional monitoring inputs in order to establish true incidences (e.g. assessment of foetal mortality may require both early and late pregnancy scanning to be conducted for each hind).

There are some generalisations about reproductive productivity of hinds that are largely accepted within the industry:

(1) The incidence of twin ovulations amongst red deer hinds is general very low (<0.5%) and probably outside the scope of genetic selection for dramatic increases in the 'twinning rate' (percentage twins born) in the foreseeable future.

(2) Ovulation failure of adult (>3 years old) hinds that are of modest-good body condition score (BCS >2.5) is generally low (<2-3%). This attests to the high annual fertility of the species, despite low fecundity.

(3) Puberty (i.e. ovulation) failure, amongst rising-two-year-old (R2) hinds on many New Zealand farms is disappointingly high (5-30+%). The inference of ovulation failure as the primary cause of wastage is based on early pregnancy scanning, acknowledging that the possible contribution of embryo wastage is unknown.

(4) While overall foetal wastage (abortion/resorption) is assumed to be low (<1%) based on some early estimates (0.6-1.0%; Audige et al. 1999), there is growing concern that some herds may be currently exhibiting incidences >5%. However, direct evidence of abortion is difficult to obtain and the assumption of foetal loss is often based on data showing a substantial difference between scanned conception rates and weaning rate (rather than calving rate). This clearly fails to factor post-natal calf mortality.

(5) Post-natal calf mortality is generally recognised as the most significant form of reproductive wastage, but its true extent is generally under-estimated on commercial farms due to the 'hands-off' approach to hinds over the calving period. Industry estimates of calf mortality range from 5-15% of calves born, recognising huge between-farm variation in actual and perceived losses.

Reproductive productivity of adult hinds

Most adult red deer hinds successfully conceive and produce a live calf each year, although the true lifetime reproductive output of individuals across a range of herds and environments has yet to be fully assessed.

1. Ovulation failure: Ovulation failure *per se* during the autumn breeding season is probably an uncommon phenomenon for hinds of reasonable BCS, irrespective of whether they are lactating or not. Indeed, most wild hinds initiate ovulation 3-4 months into their 8-9 month lactation, indicating that 'lactational anoestrus' (i.e. direct disruption of ovulatory activity by 'lactation' hormones) is not a normal feature of this species. However, lactation can undoubtedly exert a detrimental effect on the annual re-instigation of ovulation if the negative energy balance of milk production depletes hind body energy reserves to the point that BCS falls below a certain threshold just prior to the mating season. Evidence from studies investigating incidences and seasonal timing of pregnancies of lactating and non-lactating hinds indicated that modest negative energy balances of lactation that reduced adult hind BCS to 3.0-2.5 were associated with delays (7-10 days) in the timing of conceptions but did not greatly influence ultimate early pregnancy rate (Pollard et al. 2002). However, anecdotal evidence arising from severe summer drought conditions over the last decade have indicated that more severe reductions in hind BCS (i.e. <2.0), exacerbated by lactation demands, have resulted in high incidences of pregnancy failure (> 30% of adult hinds) largely attributed to anovulation.

There is some evidence that chronic stress associated with frequent yarding of groups of adult and yearling hinds around the mating period can perturb the ovulatory

process, leading to delays of 2-3 weeks in onset of oestrus and conception dates (Asher et al. 2011) but no direct evidence that it can or will ablate ovulation altogether.

2. Embryonic mortality: A question mark hangs over the role of pre-implantation embryo mortality (i.e. embryo loss within ~30 days of conception) in reproductive wastage in red deer. It is very difficult to measure embryo wastage as the first indications of reproductive output generally occur with early pregnancy scanning 2-3 months after the start of the rut. At this stage, embryo wastage may have occurred but with little consequence to the hind other than to induce a return to oestrus/ovulation. Early studies have indicated that about 85% of hinds appear to establish a viable pregnancy to their first oestrus/ovulation of the breeding season, and it is assumed that a similar proportion of returning hinds with do so to their second oestrus. Indeed, the observed synchrony of subsequent calving supports this view. However, it remains unknown as to why 15% of hinds do not establish a pregnancy to first oestrus and it is possible that embryo wastage is partly responsible. Studies by Berg et al. (1994) have indicated that embryo wastage in red deer is surprisingly low compared to other domestic ruminants, a conclusion perhaps supported by the generally high level of success with embryo transfer programmes in this species.

3. Foetal mortality: There is some current contention over the perceived incidence of foetal wastage amongst adult red deer hinds. Owing to a general lack of direct evidence of loss (i.e. observed abortions) the role of foetal loss has been largely downplayed in the past. Now questions have arisen as to whether some herds have exhibited significant wastage due to mid- to late-term abortion/foetal resorption. Clearly it is difficult to find aborted foetuses in the field due to their small size, state of decomposition, scavenging and even ingestion by the hind. Furthermore, it is likely that at least some foetal mortalities fail to be expelled and are resorbed internally. Generally, the perceived link between poor reproductive performance of a herd and foetal loss is by inference based on ultrasound scanning data and calving data. Unfortunately, there is an assumption by some observers that a lack of physical evidence of post-natal mortality of calves indicates a problem elsewhere. It should be noted that calf mortalities are themselves difficult to detect in most calving environments, particularly if no effort is made to detect them. However, if there is unequivocal evidence that there is a discrepancy between scanned pregnancy rates and the calving rate, foetal loss is implicated.

It can be argued that such unequivocal evidence based on actual calving data is ‘closing the gate after the horse has bolted’ in terms of identifying the causal problem. Ideally, direct evidence of abortion and recovery of tissues from aborted foetuses and their dams, would go a long way to identifying risk factors associated with foetal death. Direct ultrasonographic evidence could also be a powerful tool, and we are presently encouraging farmers who suspect a problem in their herd to consider repeated scanning at various stages of the pregnancy cycle. This would allow identification of hinds undergoing foetal loss, although it may not indicate the actual timing of such loss with wide scanning intervals.

Abortifacient agents are not well known for red deer. It is very unlikely that normal levels of stress associated with farming practices will contribute significantly to foetal death. Indeed, the robustness of the red deer pregnancy in the face of quite harsh

environmental challenges is quite remarkable (as is perhaps evinced by pregnancy maintenance following capture of wild hinds and placement on farms in the early days of the industry). It would seem more likely that actual incidences of foetal loss result directly from the action of biotic agents, such as infectious disease agents, ingestion of toxic plants or possibly even specific medicinal treatments. Needless to say, it is important for managers of affected herds to seek identification of causal agents in order to mitigate future losses.

Obvious candidates include known abortifacients amongst other ruminant species, including infectious agents such as *Leptospira*, *Toxoplasma*, and *Campylobacter*, and toxins arising from ingestion of *Pinus* and *Cupressus macrocarpa*, to name a few. However, clinical symptoms vary across species and other candidates not important in traditional ruminants should also be considered as possible abortifacients in red deer.

4. Calf mortality: Peri-natal and post-natal (pre-weaning) calf losses probably still account for significant herd wastage across red deer farms. Early data from daily monitoring of hinds over the calving period indicated losses of 8-15% of calves born (Asher & Adam 1985). While these figures may have been influenced by lower levels of hind habituation to pastoral environments evident during the earlier years of deer farming and also by artefacts (e.g. calf abandonment) caused by disturbance of parturient hinds due to the monitoring process, they are essentially similar to other observational studies since (Audige et al. 1999; Beatson et al. 2000). All of these studies have confirmed, however, (1) the difficulty in observing the true extent of calf mortality and (2) the relative importance of the birthing environment for optimising calf survival. The later point has been well recognised by the industry over the last few decades, and most farmers strive to provide an appropriate calving environment in which parturient hinds can seek isolation and low-level cover for calf concealment. But this also means that opportunities to directly observe the birthing process and to monitor calf losses are severely limited. Thus, we have little current data on actual calf losses presently occurring on deer farms.

Reproductive productivity of R2 hinds

Most red deer hinds within New Zealand's relatively benign environment attain puberty (first ovulation) at ~16 months of age as rising-two-year-olds (R2's) and successfully rear their first calf between 24 and 27 months of age (Audige et al.1999; Beatson et al. 2000; Asher & Pearse 2002). In more hostile environments, it is not uncommon for puberty to be delayed by a year or more (Clutton-Brock et al. 1982). The precise timing of the onset of puberty is constrained by two principal factors in this species; body mass and photoperiod. Hinds must attain at least 65-70% of their ultimate mature body mass (often referred to as the "70% rule") by the time they enter their second phase of decreasing daylight length (i.e late summer-autumn in their R2 year) in order to enter into a reproductive state (Lincoln & Short 1980). Failure to attain sufficient body mass will delay puberty by at least one year, as is generally the case on the Isle of Rhum, Scotland (Clutton-Brock et al. 1982).

However, it is recognised that reproductive productivity of R2 hinds on many New Zealand deer farms falls well below biological expectation (Audige et al. 1999; Beatson et al. 2000; Asher & Pearse 2002) and this is seen as a significant source of

overall reproductive wastage within the national herd (Asher and Pearse 2002). This has been the focus of recent research, particularly in relation to puberty failure (Asher et al. 2005, 2011). It is acknowledged, however, that other sources of wastage (e.g. foetal loss, neonate mortality) may well be important for R2 hinds and warrant on-going research.

1. Puberty failure in R2 hinds: A large body of data show that early-scan pregnancy rates of R2 hinds (i.e.before 60-80 days of pregnancy) are highly variable across farms and even populations within farms. There has been an implicit assumption that the non-pregnant hinds have failed to enter puberty in their second autumn. While this assumption is probably reasonable, it is acknowledged that there will also be components of conception failure (e.g. failure of stags to mate oestrous hinds) and embryonic mortality. However, what is notable across all populations studied to date is the strong and profound association between R2 hind body mass and early pregnancy incidence (Asher et al 2005, 2011), with the probability of pregnancy increasing with increasing body mass. This implicates strongly the role of the well-known association between body mass and the incidence of first ovulation (i.e. puberty) that is seen across a wide range of mammalian species.

The issue that arises from applying the "70% rule" is that it is specific to genotype because of the huge differences in mature body mass between the various red deer subspecies that are widely introgressed into our national herd. These range from the small stature Scottish red deer (*Cervus elaphus scoticus*) that underpins much of our feral sourced livestock, the larger Eastern European red deer (*C.e.hippelaphus* and *pannonensis*) imported during the 80's and 90's, and the much larger North American wapiti (*C.e. nelson, roosevelti* and *manitobensis*) derived from Fiordland or more recently imported from Canada. Despite a 2.5x differential in body mass between largest and smallest subspecies, crossbreeding is not generally problematical and introgression has been pervasive, particularly over the last 15-20 years. An obvious consequence of widespread crossbreeding is the development of various unstabilised composite genotypes and variable genotypic composition of individuals within and between herds. This is reflected in highly variable mature body weights of breeding hinds within populations. Consequently, puberty body mass thresholds are no longer a fixed target based on any particular genotype but reflect individual hind genotypic composition (Figure 1). For example, the live-weight targets recommended for yearling hinds during the early 1980's (Kelly & Moore 1977), based largely on the smaller *scoticus* subspecies, are not particularly relevant today given that the average genetic body mass of hinds has probably increased by 20-30 %. The problem with setting targets for any given population is the high level genetic heterozygosity often encountered within that population; this being exemplified by high levels of wapiti introgression within many New Zealand farmed deer populations (Asher et al. 2005). Growth and live-weight targets set for the 'average' hind genotype will clearly penalise those hinds with a higher proportion of wapiti genes that have a genetically larger mature body mass. While 'wapiti scoring' systems have been developed to allow farmers to estimate the relative proportion of wapiti genes of individual R1 hinds (Ward et al. 2006), it is not a precise measure of genetic parentage and does not factor in the more recent introgression of Eastern genotypes (which do not exhibit such extremes in readily observable phenotype from other 'red deer'). The general message to deer farmers, therefore, is to raise target live-weight expectations to account for genetic variation within the herd.

Comment [CB3]: Not sure what N American wapiti derived from Fiordland means: are they from N. America or Fiordland? Perhaps you mean the N. American-type species that are bred from the wild population principally found in Fiordland?

Even for a given genotype the "70% rule" is a generalisation and a simplification. We now know from studies involving known genotypes that the curvilinear relationship between body mass and pregnancy (puberty) attainment can vary between herds and even between populations within the same herd (Figure 2). In effect the threshold body mass requirement for entry into puberty is not necessarily a fixed target for a given genotype, and is clearly influenced by other factors. There are two notable observations from the data from two herds presented in Figure 2; (1) the 'stud' populations within each herd, which had received preferential management inputs throughout, had lower live-weight thresholds than the respective 'commercial' populations; and (2) the unusually high live-weight thresholds exhibited by the commercial hinds on Farm A may in some way be linked to the fact that that particular cohort had been subjected to severe drought conditions as calves, having been weaned about 10 kg lighter than earlier years' cohorts (despite the fact that considerable subsequent effort went into their nutritional environment to compensate for such low weaning weights). This introduces the intriguing concept that genotype-specific puberty live-weight thresholds may be influenced by the prior nutritional environment of the R1 hind. In effect, the growth rate pathway may be an important determinant of the required body mass for entry into puberty at 16 months of age. If this hypothesis is valid, it clearly puts emphasis on optimising the entire nutritional milieu from birth to puberty, rather than focussing on 'catch-up' late in the game.

Another interesting observation reported recently (Asher et al. 2011) was the influence of exogenous melatonin treatment of R2 hinds on puberty live-weight thresholds. For one of the study herds, melatonin treatment of hinds at 11-12 months of age not only advanced conception dates by 2-3 weeks, as expected, but also dramatically increased the proportion of hinds pregnant at scanning at 18-20 months of age. In effect, the prior melatonin treatment decreased the puberty live-weight threshold, allowing small hinds to jump the hurdle required for initiation of puberty. While this effect was not universal across the study populations, it does indicate a profound relationship between photoperiod and body mass. While this clearly is not a practical tool for on-farm application, as the biological consequences of 'forcing' puberty in small hinds are likely to be counter-productive, it does open up an interesting model on how photoperiod influences hind growth (and perhaps body composition) and onset of breeding.

2. Joining practices for R2 hinds

Early considerations on low pregnancy rates of R2 hinds included concerns that a high proportion of R2 hinds were excluded from mating due to disruptive management regimes and inappropriate herd social structure (e.g. dominant older hinds exhibiting aggressive responses towards younger hinds; older stags actively avoiding oestrous yearlings in favour of older hinds). Out of these considerations arose modified mating management systems specifically for R2 hinds; these principally being the early joining of R2 hinds with high ratios (1:10) of R2 stags and the exclusion of older hinds from these mating groups. While there is anecdotal evidence that such practices have generally improved pregnancy rates of R2 hinds, strong empirical data to support this are lacking. Furthermore, high incidences of high non-pregnancy rates amongst R2 hinds still exist despite such management practices. While we contend that careful attention to mating management is an important

consideration, the evidence suggests that puberty failure rather than mating failure is the main contributing factor in incidences of pregnancy failure in R2 hinds.

3. Foetal wastage amongst R2 hinds

As with adult hinds, foetal wastage amongst R2 hinds has been largely overlooked until recently. However, there is growing concern with some herds that the differential between the scan-pregnancy rate and the subsequent weaning rate is very large (>15%) and cannot be reconciled with observed incidences of neonatal mortality. Recent clinical investigations involving repeat pregnancy scanning of R2 hinds on two large Southland properties have clearly identified significant foetal losses (10% and 16%, respectively, between May and September scanning) during the gestation period (P.R.Wilson and G.R.Sinclair; unpublished data presented elsewhere in these proceedings). This is the first concrete evidence of significant foetal wastage in R2 hinds and these studies are being extended to other large herds throughout the country, including those involved in the earlier puberty studies (Asher et al 2011). To date, the available evidence suggests that such wastage is largely confined to the R2 cohorts, but again acknowledging a paucity of data on foetal wastage in general.

Clearly, identifying the causal factor(s) is of primary consideration following demonstration of foetal loss. That losses on the two farms under investigation were only in young hinds is consistent with an infectious causative agent to which animals develop an immunity or resistance. Although results are preliminary at this stage, there does appear to be an strong association with *Toxoplasma* infection (P.R.Wilson and G.R.Sinclair ; unpublished data). Further investigations are underway to provide more evidence of causation.

4. Post-natal mortality

There are few data pertaining to calf losses for first calving hinds. It is generally assumed that such losses are similar to those of older hinds but acknowledging that naive dams are probably more prone to dystocia and mismothering. Such losses may be exacerbated by human disturbance; hence the paucity of data.

Stag Factors

The ability of sire stags to service and fertilize oestrous hinds is often overlooked in relation to reproductive performance of the herd, except in catastrophic situations of complete or near complete failure of the breeding herd. However, most farmers understand the risks associated with single sire mating systems, and mitigate such risk by attending to stag:hind ratios and strategic sire stag replacement during the mating period.

In response to considerable publicity on the benefits of early stag joining (Beatson et al. 2000), the date of joining has generally shifted forward by about 2-3 weeks (i.e. from mid-late March to early March-mid February) over the last 10 years to ensure that farmers capture the benefits of earlier calving dates. This has been reinforced by the demonstration that introgression of Eastern red deer genotypes may be leading to earlier seasonal breeding activity (Scott et al. 2006) that was not being captured by the

later joining practices. Furthermore, recent research has further demonstrated a modest heritability for early conception trait in hinds, leading to the development and establishment of a breeding value (BV) for this trait in DEERSelect (Archer et al. 2008). Such early joining coupled with conception date estimation based on ultrasonographic foetal aging, has allowed the identification of early conceiving hind phenotypes for assigning BV values for sire stags.

Animal health-related causes of reproductive wastage in deer

The focus of improvement in reproductive performance across the deer industry has appropriately been on improving conception rates through nutrition and management, and improving calving and post-calving management and environment to increase calf survival to weaning. The potential role of disease in reproductive failure has been largely passively overlooked, because it is likely that reproductive losses of this nature are likely to be sporadic and not common to all farms, are difficult to observe given the behavioural nature of deer and reasonably "hands-off" farming systems. However, there is past evidence that disease conditions have resulted in reproductive wastage in deer herds in New Zealand, and as other causes of poor reproductive performance as discussed above are eliminated or managed, the relative importance of animal health-related causes of reproductive loss will likely increase.

While the difficulty in partitioning reproductive losses has been discussed above, there is an added difficulty in investigating potential disease causes. This is because it is difficult under routine management practices to identify where losses are occurring through the reproductive cycle, and even more difficult to identify affected animals, including dams, foetuses or progeny, to get tissues and samples for disease investigations. However, there are some examples where this has happened and there is growing evidence of disease as a cause of reproductive loss in some and perhaps many as yet un-investigated circumstances.

(1) Disease causes of failure to conceive/early embryonic loss.

Any condition that causes infertility, loss of libido, anatomical abnormality, or inability to mate will result in reproductive loss.

Causes of male infertility or failure to mate.

- Cryptorchidism. This has been observed in stags. It is easily detected by a reproductive soundness examination, but these are rarely performed.
- Anatomical abnormalities such as penile deviation, or the aftermath of non-specific infections are observed occasionally but are not commonly documented
- Injury is common during the rut, and physical incapacity may result
- *Brucella ovis* has been diagnosed in deer and results in poor semen quality or aspermia.
- Cervid herpesvirus-1 has been shown to produce severe pustular and ulcerative lesions of the penis and prepuce which are likely to result in pain and loss of apparent libido. The first identification of CvHV-1 in deer in New Zealand was in a stag semen sample which was abnormal, so semen quality may also be affected. These observations need to be verified.

- Non-specific causes of epididymitis or testicular infection should always be considered as a potential cause of infertility.
- Any observation of stag infertility should be investigated for a variety of other pathogens such as Campylobacter and Trichomonas.

Causes of female infertility and early embryonic loss.
No disease causes of hind infertility of early embryonic loss have been identified or confirmed. This is probably because of the lack of investigation of hinds that are not pregnant at scanning, and difficulty in detecting whether the failed pregnancy is due to failure to conceive, failure to implant, or early embryonic loss. Given the persistently lower pregnancy rate in R-2-yr old hinds, this is worthy of investigation in this age group.

- Non-specific causes such as endometritis, perhaps resulting from previous dystocia and/or retained placenta, will result in infertility. It has been observed that hinds that fail to conceive or fail to rear a calf are more likely not to conceive or rear a calf the subsequent season. While this could be associated with mothering ability and or other individual behavioural or metabolic effects, uterine abnormalities may contribute.
- Selenium deficiency in sheep is associated with early embryonic loss. This could also be the case in deer.
- CvHV-1 is known to infect and affect the reproductive tract of hinds, but its effect on fertility is unknown. In cattle, the similar virus, BvHV-1 causes reproductive wastage.
- It is possible that campylobacter may cause early embryonic loss.

(2) Disease causes of abortion

There are more than 30 known causes of abortion in ruminants in New Zealand (See Wilson and Sinclair, elsewhere in these proceedings). To date there have been no proven causes in deer, since it is uncommon for abortions to be detected, and if suspected, it has been uncommon for those occurrences to be investigated or reported.
Any disease condition that results in sub-optimum health in hinds risks lower reproductive performance. For example, copper deficiency.
There is clinical or published evidence for the following:

- Toxoplasma (see above) has been suspected in two herds with losses of pregnancy of 44/440 (10%) and 60/380 (16%) between scans in late May and early-mid September. A high seroprevalence was observed in both herds, and 10/10 and 6/20 uteri from aborting hinds in those herds, respectively, were PCR positive for Toxoplasma. Further evidence of causation is currently being pursued (Wilson and Sinclair, unpublished).
- Leptospirosis has been suspected as a cause of abortion in deer in New Zealand but proof of causation has not been established since the ultimate diagnostic test is identification of leptospires in aborted foetal tissues. Association with seropositivity is insufficient evidence of causation because of the high prevalence of infection in New Zealand deer herds. In the USA, artificial challenge of whitetailed deer with *Leptospira pomona* has been shown to cause abortion. Thus, leptospirosis as a cause of foetal loss remains to be confirmed.
- Selenium deficiency was associated with the abortion of a small number of calves on a deer farm in New Zealand.

- Iodine deficiency has been shown in Switzerland to result in late abortion in fallow deer. It is likely that iodine deficiency contributes to loss of pregnancy in some cases in New Zealand since there have been several diagnoses of iodine deficiency in deer here.

(3) Disease causes of perinatal loss

Again, it is difficult to partition disease-related perinatal losses from those resulting from mothering ability, hind lactation, management and environmental factors.

- Leptospirosis has been shown to cause lower reproductive performance (weaning percentage) overall. Recent research results showed that in 4 herds studied, carriage of foetus was not affected, yet weaning rate was on average five percentage points lower. On two farms studied, where the seroprevalence was high (87% and100%) the difference in weaning rate between vaccinated and non-vaccinated deer (both 2-yr-old and adult) was 10% and 11%, respectively.
- It is possible that iodine deficiency is a cause of perinatal mortality due to weakness of the newborn calf.
- Sporadic navel infection has been reported

(4) Disease causes of postnatal losses

It is widely believed that failure to rear a calf is the major cause of reproductive wastage, other than failure to conceive. While this may result from perinatal mortalities, there are some causes of postnatal mortality that have been diagnosed in New Zealand.

- Iodine deficiency. Goitre has been shown in deer up to 2-3 months of age, causing death, or requiring euthanasia because of poor growth and health
- Copper deficiency resulting in osteochondrosis is possibly an under-diagnosed cause of wastage, since this condition is reported only in pre-weaned deer causing severe osteroarthritis as a result of collapse of subchondral bone, which is an indirect effect of metabolic deficiency of copper-containing enzyme activity maintaining the organic matrix of bone.
- White muscle disease associated with selenium deficiency occurs in young ruminants of other species. It could be a cause of postnatal losses in deer.
- Cryptospiridiosis has been reported on many farms in New Zealand as a cause of postnatal mortality.
- Enteric pathogens such as *E. coli,* Rotavirus and Coronavirus have all been diagnosed as causes of postnatal losses in deer in New Zealand.

Many of the diseases potentially affecting reproductive performance are preventable by management, appropriate monitoring for trace elements and animal health interventions aimed at maintaining overall health of the animal and the herd.

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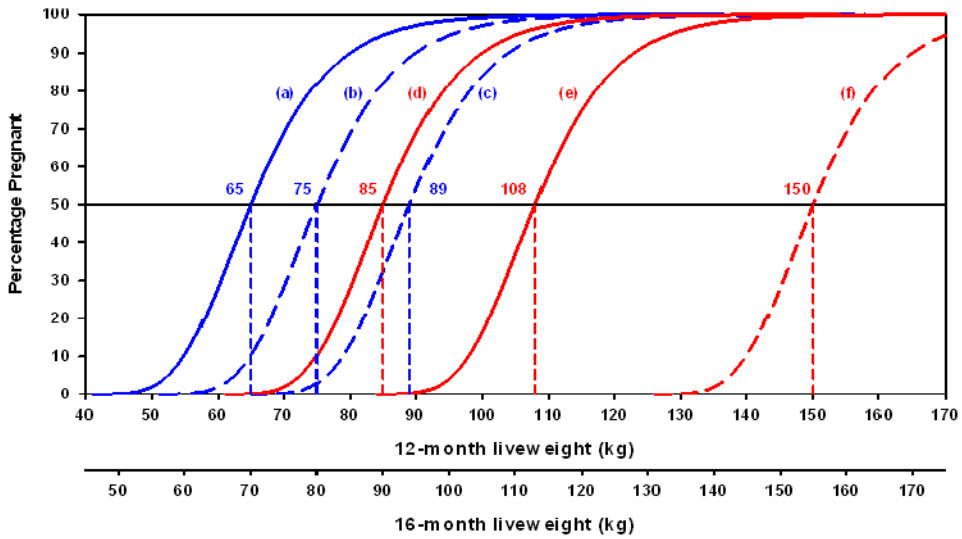

Figure 1: R2 hind live-weight vs. percentage pregnancy curves for a range of genotypes modelled from actual scanning and DNA data (a) 100% Western (*scoticus*), (b) 50:50% Western x Eastern, (c) 100% Eastern (*hippelaphus*, *pannonensis*), (d) 30% wapiti, (e) 50% wapiti and (f) 100% wapiti (e.g. *nelsoni*).

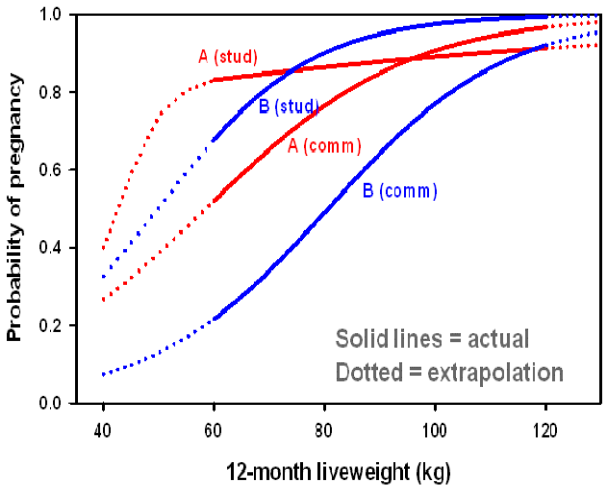

Figure 2: R2 hind 12-month live-weight vs. probability of pregnancy modelled from data from two farms of stabilised composite hinds (60% Eastern genes). Each farm runs separate stud and commercial (comm.) herds.