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A review of factors that impact on the capacity of beef cattle females to conceive, maintain a pregnancy and wean a calf—Implications for reproductive efficiency in northern Australia

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A review of factors that may impact on the capacity of beef cattle females, grazing semi-extensive to extensive pastures in northern Australia, to conceive, maintain a pregnancy and wean a calf was conducted. Pregnancy and weaning rates have generally been used to measure the reproductive performance of herds. However, this review recognises that reproductive efficiency and the general measures associated with it more effectively describe the economic performance of beef cattle enterprises. More specifically, reproductive efficiency is influenced by (1) pregnancy rate which is influenced by (i) age at puberty; (ii) duration of post-partum anoestrus; (iii) fertilisation failure and (iv) embryo survival; while (2) weight by number of calves per breeding female retained for mating is influenced by (i) cow survival; (ii) foetal survival; and (iii) calf survival; and (3) overall lifetime calf weight weaned per mating. These measures of reproductive efficiency are discussed in depth. Further, a range of infectious and non-infectious factors, namely, environmental, physiological, breed and genetic factors and their impact on these stages of the reproductive cycle are investigated and implications for the northern Australian beef industry are discussed. Finally, conclusions and recommendations to minimise reproductive inefficiencies based on current knowledge are presented.
1. Introduction

The reproductive efficiency of a herd is an important component of beef cattle productivity in northern Australia. Main factors influencing reproductive efficiency in cattle include age at puberty and first conception, duration of post-partum anoestrus and total lifetime productivity. Lifetime production, or total weight of calves weaned during a cow's lifetime, is the most important output of the female must be reduced. These include reducing age at first calving and the post-partum anoestrus period and minimising fertilisation failure and embryo, foetal (pre-natal) and peri- and post-natal calf mortalities from conception to weaning. While marked year to year variation in weaning rate does occur in this environment, Hasker (2000) has suggested a production objective of 80 calves annually weaned per 100 cows mated. Therefore, this review investigates these unproductive periods that impact on the capacity of breeding females, grazing semi-extensive to extensive pastures in this region, to conceive, maintain a pregnancy and wean a calf at approximately 6 months of age. The prevalence and causes of the failure of a stage of the reproductive process are discussed where possible. Main causes of losses discussed include infectious and non-infectious causes. Where limited information exists for northern Australia in the areas identified above, information from international, extensive beef and intensive beef and dairy cattle studies are drawn upon to discuss the implications for reproductive efficiency in northern Australia.

2. Northern Australian beef cattle production system environment

Beef cattle production is the main form of land utilisation in the northern Australia region discussed in this review (Turner, 1975). It is a vast region located within the sub-tropics and tropics; has a range of different production systems and environmental stressors; encompasses the state of Queensland, the Northern Territory and the Kimberley and Pilbara regions of the state of Western Australia; and covers approximately 4 million km² (Anon, 2008). Mean temperatures corrected to sea level makes this region comparable, in this respect, to other tropical regions of the world (Turner, 1975). Extreme maxima, mainly due to the great east-west extent of the continent in the vicinity of the Tropic of Capricorn, reach 50 °C over the inland in summer, while winters are warm in the north and cooler in the south, with overnight frosts common in inland areas south of the Tropic of Capricorn (Anon, 2008). Australia is relatively dry with 50% of the country having a median rainfall of less than 300 mm per year and 80% less than 600 mm (Anon, 2006). A monsoonal 'wet' season generally occurs in northern Australia from approximately November through April, when almost all the rainfall occurs, and a 'dry' season occurs from May through October (Anon, 2006).

Because of the vast nature of this region, the northern Australian beef cattle industry is managed across a diverse collection of rangeland vegetation and pasture communities (Tothill and Gillies, 1992). The major characteristics of these diverse northern Australian production systems include low stocking rates (up to 1 beast to 150 ha)

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**Table 1a**
Mean ages and weights at puberty for different genotypes in northern Australia (Belmont Research Station, central Queensland).

| Author          | Genotype                          | Number of animals | Age (days) | Weight (kg) |
|-----------------|-----------------------------------|-------------------|------------|-------------|
| **Study 1**     |                                   |                   |            |             |
| Post and Reich  | Africander cross (AX)             | 52                | 545        | 269         |
|                 | Brahman cross (BX)                | 41                | 525        | 261         |
|                 | Hereford–Shorthorn, selected (HSS)| 23                | 532        | 240         |
|                 | Hereford–Shorthorn, Random bred (HSR)| 12           | 563        | 236         |
|                 | Grade Africander (GA)             | 19                | 511        | 258         |
|                 | Grade Brahman (GB)                | 12                | 481        | 246         |
|                 | F2 AABAB                          | 11                | 494        | 264         |
|                 | F2 ABBA                           | 8                 | 508        | 279         |
|                 | F2 BAAB                           | 8                 | 576        | 283         |
|                 | F2 BABA                           | 11                | 536        | 269         |

* Within columns means followed by the same superscript are not significantly different (P = 0.05).

**Table 1b**
Mean ages and weights at puberty for different genotypes in northern Australia (Brigalow Research Station, central Queensland).

| Author          | Genotype   | Number of animals | 1st oestrus | 2nd oestrus |
|-----------------|------------|-------------------|-------------|-------------|
|                 |            |                   | Age (days)  | Weight (kg) |
| **Study 2**     |            |                   |             |             |
| Burns et al.    | Hereford   | 137               | 565         | 277         |
|                 | Simmental  | 179               | 550         | 322         |
|                 | Belmont Red| 102               | 527         | 293         |

Within columns and classes values followed by the same letter are not significantly different (P = 0.05). Means presented as mean ± standard error of mean.

**Table 1c**
Unadjusted trait means ± SED for Brahman and Tropical Composite puberty traits across for locations in northern Australia (Brian Pastures (south-east Queensland), Toorak (north-west Queensland), Belmont (central Queensland) and Swans Lagoon (north Queensland) Research Stations).

| Author          | Trait      | Genotype | Number of animals | AGECL (days) |
|-----------------|------------|----------|-------------------|--------------|
| **Study 3**     | AGECL      | BRAH     | 1007              | 750 ± 142.1  |
|                 | WTCL       | BRAH     | 993               | 334 ± 44.8   |
|                 | AGECL      | TCOMP    | 1108              | 650 ± 119.5  |
|                 | WTCL       | TCOMP    | 1094              | 329 ± 45.9   |

AGECL = Age at first corpus luteum (CL) 9 days—number of days from birth to the first CL or corpus albicans (CA) on either the left or right ovary, observed by real-time ultrasound scan. WTCL = Weight at first CL (kg)—Heifer liveweight on the day (or within 7 days) of the first-observed CL or CA. Brahman = BRAH and Tropical Composite = TCOMP.

(Tothill and Gillies, 1992); limited introduction of pasture species to improve production from native pastures (Coates et al., 1997); weight gain occurring during the hot, wet summer months (November–February) and weight loss during the dry winter-spring (March–October) period, which is managed using strategic supplementation (Winks, 1984; McCosker and Winks, 1994). Management groups of 500–1000 animals are common, with very little single sire mating; a predominance of continuous mating with peak calving in October–December; and irregular handling and husbandry of cattle—typically twice annually in April–July and August–September (Bortolussi et al., 2005).

The cattle tick (*Boophilus microplus*), which is endemic in large areas of this region; gastro-intestinal helminths (*Haemonchus placei*, *Cooperia* spp., *Trichostrongylus axei* and *Oesphagostomum radiatum*); high ambient temperatures and solar radiation; Bovine Infectious Keratoconjunctivitis (BIK) and other diseases; reliability of rainfall; soil fertility; woody vegetation; and the fluctuation in both quality and quantity of available forage are the major constraints.

**Table 1d**
Model-predicted means for Brahman and Tropical Composite heifer puberty traits by location in northern Australia.

| Author          | Trait      | Location     | Study 3 |
|-----------------|------------|--------------|---------|
|                 | AGECL      | Brian Pastures| 643<sup>b</sup> |
|                 |            | Belmont      | 652<sup>b</sup> |
| Johnston et al. | AGECL      | Toorak BRAH  | 724<sup>a</sup> |
|                 | WTCL       | Toorak BRAH  | 706<sup>a</sup> |
|                 |            | Swans lagoon| 805<sup>b</sup> |
|                 |            |      BRAH    | 357<sup>a</sup> |
|                 |            |      TCOMP   | 339<sup>b</sup> |
|                 |            |      TCOMP   | 323<sup>a</sup> |

The location effect at Toorak was considered separately for Brahman (Toorak BRAH) and Tropical Composite (Toorak TCOMP) because of confounding of genotype with the property of origin. Within the Belmont location (column), trait means followed by different superscripts represent significant differences between the two genotypes (P = 0.05). Within rows, means followed by different letters indicate significant location differences within a genotype (P = 0.05).
to animal production in this region (Turner, 1975; Burns et al., 1988, 1997). Because of heritage, up to the mid-to-late 1950s the Australian beef cattle industry was based on British genotypes of cattle imported from temperate regions (Daly, 1981). With the realisation of the superior production performance of Bos indicus and B. indicus × Bos taurus genotypes in this harsh northern Australian environment (Turner, 1975), the proportion of B. indicus and B. indicus cross cattle in northern Australia has risen from 5% in 1970 to approximately 85% at present, principally through the use of Brahman cattle and their derivatives, i.e., Droughtmaster, Santa Gertrudis, Braford, Brangus, Charbray, Simbrak and Brahmos (Farquharson and Banks, 2002; Bortolussi et al., 2005).

Therefore, the varying levels of environmental stressors, parasite burdens and beef cattle genotypes grazing the diverse production systems in northern Australia all ‘impact on the capacity of beef cattle females to conceive, maintain a pregnancy and wean a calf’ in this harsh production region.

3. Reproductive inefficiencies during the attainment of puberty and of first ovulation post-partum

3.1. Attainment of puberty

Puberty is the process whereby animals become capable of reproducing themselves and in female cattle it involves a transition from a state of ovarian inactivity to one in which there is a first oestrus that is followed by a normal luteal phase (Moran et al., 1989). Normal events that occur during the attainment of puberty in heifers can include corpus luteum (CL) inadequacies, absence of antecedent progesterone ‘priming’, lack of oestrus and failure of ovulation or silent ovulations (Byerley et al., 1987), all of which increase the incidence of embryonic mortality and/or fertilisation failure.

It is widely recognised that the age at which herd replacement heifers reach puberty and therefore produce their first calf influences the reproductive efficiency of a beef herd (Burns et al., 1992b; Cundiff et al., 1992). Therefore, heifers that calve first at 2 years old produce more calves during their lifetime than heifers that calve first at ≥3 years of age (Fordyce et al., 1994). However, while Fordyce et al. (1994) also demonstrated that heifers calving at 2 years of age have a higher lifetime output, a higher level of nutritional management and subsequent higher cost of inputs were required to minimise reductions in mature size in this class of animal (Burns et al., 1997).

Tables 1a, 1b, 1c and 1d clearly demonstrate that heifers of all genotypes from a number of northern Australian studies had not reached puberty by the start of mating as yearlings, but by 24 months of age the majority of both non-tropically adapted and tropically adapted B. taurus, B. indicus and B. indicus cross heifers had reached puberty (Post and Reich, 1980; Burns et al., 1992b; Johnston et al., 2009), except Brahman at Swans Lagoon in the dry tropics of northern Queensland (Johnston et al., 2009). However, given the review of Moran et al. (1989), which reported that first ovulation is not synonymous with puberty in most heifers, care should be taken in the interpretation of the age at first CL results from the study of Johnston et al. (2009) as an indication of attainment of puberty in the genotypes described.

Three of the studies discussed above have reported a number of ways that puberty can be measured and include observation of heat (Burns et al., 1992b), monitoring of progesterone levels (Post and Reich, 1980) and scanning using real-time ultrasound and detection of a CL (Johnston et al., 2009). From the results presented, it would appear that weight at age of puberty has increased over the years from a mean value of 260 kg in Post and Reich (1980), to 321 kg in Burns et al. (1992b) to 334 kg in Johnston et al. (2009). Given that these results are confounded by location and seasonal effects, these differences possibly reflect the methods of measurement rather than a change in age or weight at which puberty occurs.

Moran et al. (1989) also reported that the onset of puberty is primarily a function of liveweight, that is, a physiological rather than a chronological age (Moran et al., 1989). Further, maturation involves a complex interaction between endocrine factors that results in the development of the reproductive tract and the rate at which this occurs depends mainly on genetic and environmental factors such as plane of nutrition throughout life that determines an animal’s size and liveweight (Moran et al., 1989). Subsequently, for the majority of northern Australia production systems, genetic and nutritional constraints prevent the attainment of puberty in the majority of non-tropically adapted and tropically adapted B. taurus, B. indicus and B. indicus cross heifers as yearlings and supplementation strategies to attain this goal are not economically practical. Therefore, a more realistic strategy for heifer management in northern Australia to optimise female lifetime productivity is to ensure that all heifers have attained puberty by 2 years of age and are ready to conceive at the time when bulls are first introduced.

This brief review of puberty highlights that puberty and first ovulation are not necessarily synonymous and that under the range of northern Australian production systems it is not important to know precisely know when puberty occurs. What is important in this environment is that heifers are sexually mature at the start of mating.

3.2. Attainment of first ovulation post-partum

The post-partum anoestrous interval is the period between parturition and the return to the normal cycling state of the ovaries and uterus and first ovulation (Arthur et al., 1996). Prolonged post-partum anoestrus has been identified as the major problem area of cow fertility in tropical northern Australian herds and can have a significant impact on herd profitability (Entwistle, 1983; Teleni et al., 1988). Intervals to first ovulation and first oestrus tend to be longer in B. indicus cattle particularly in first-calf cows (Teleni et al., 1988). On average in northern Australia, lactating B. indicus and B. indicus infused females will first ovulate 7 months post-partum and average weaning
rates of 50–60% occur due to lactation anoestrous (Entwistle, 1983). A characteristic pregnancy pattern in young, tropically adapted breeding females in this northern Australian region is one in which females may only calve in alternate years, that is, heifers that conceive at 2 years old fail to conceive at the subsequent mating, but conceive again during the next mating season at 4 years old (Entwistle, 1983). This observation is not specific to B. indicus and B. indicus infused females in northern Australia as long calving intervals have also been reported in tropical production systems of Colombia where the calving interval for suckled zebu cows has ranged from 15.6 to 17.6 months (Olvera-Angel and Martínez, 1990). Further, several factors reported to influence the length of this interval include pre- and post-partum nutrition/energy balance (Rutter and Randel, 1984; Ruiz-Cortés et al., 1997); frequency of suckling (Randel, 1981; Ruiz-Cortés et al., 1997), age (Dimmick et al., 1991) and body condition (Randel, 1990). These factors have also been observed to influence the length of the post-partum anoestrous interval in tropical beef cattle in northern Australia.

4. Investigation of the main periods and prevalence of reproductive inefficiencies post-ovulation

4.1. Fertilisation failure

Fertilisation failure is a measure of the number of ova that fail to be fertilised for various reasons and direct estimates can only be obtained following recovery and morphological examination of ova or embryos soon after breeding (Sreenan and Diskin, 1986).

Post (1980) measured plasma progesterone levels twice weekly in a well managed central Queensland research herd and observed high losses due to fertilisation failure ranging from 12% to 19%. Post (1980) concluded that these losses could have included some very early mortalities of fertilised ova in Hereford-Shorthorn cattle (selected (18%) and unselected (19%) lines) compared to Brahman (14%) and Africander (12%) cross cattle. In artificial insemination (AI) and natural mating (NM) studies in both B. taurus dairy and B. taurus and B. indicus beef cattle, fertilisation rates were reported to range from 71% to 100% (Diskin and Sreenan, 1980) (nulliparous B. taurus cross beef heifers—AI); Roche et al., 1981 (nulliparous B. taurus cross beef heifers—AI); Smith et al., 1982 (nulliparous B. indicus cross beef heifers—AI); Saacke et al., 2000 (lactating B. taurus dairy cows—NM and AI)). In a review of fertilisation rates in different classes of B. taurus beef and dairy cattle subjected to NM and AI, Santos et al. (2004) reported average fertilisation rates of 75% in post-partum suckled beef cows (range 60.0–100%—Breuel et al., 1993 (multiparous cows—NM and AI)); 98.6% in non-lactating beef cross cows (range 94.0–100%—Maurer and Chenault, 1983 (parous and multiparous cows—NM); Ahmad et al., 1995 (multiparous cows—NM)) and 88.0% in growing beef cross heifers (range 75.0–100%—Maurer and Chenault, 1983 (nulliparous—NM); Dunne et al., 2000 (nulliparous—AI)), respectively. Santos et al. (2004) concluded that lactation status may exert a negative effect on fertilisation rate in beef cattle.

Further, Diskin and Sreenan (1980) reported that in genitally normal heifers/cows, fertilisation failure accounts for approximately 10% of overall reproductive failure levels and should be considered an acceptable level of loss (Diskin and Sreenan, 1980). However, both Post (1980) and Sreenan and Diskin (1986) both suggested that the values recorded from their studies, conducted under reasonably well managed environmental conditions, may be an underestimate compared to those that may be observed in animals grazing pastures under harsher environmental conditions. Therefore, the studies of Post (1980), Sreenan and Diskin (1986) and Santos et al. (2004) suggest that fertilisation failure rates >10% could occur in beef cattle herds.

4.2. Embryo mortality

Early embryo mortality (EEM) and late embryo mortality (LEM) refer to mortalities occurring between fertilisation and approximately day 24 of gestation and between days 25 and 45 of gestation, respectively (Committee on Bovine Reproductive Nomenclature, 1972). Days 14–19 represent the period of maternal recognition of pregnancy or the anti-luteolytic effect.

Embryo mortality rates have been reported in a number of sub-tropical and tropical research studies. These included mortalities of 31% (4/14), 25% (3/12) and 38% (5/13) in small numbers of Brahman cross, Africander cross and British cross cows, respectively, in central Queensland (both EEM and LEM; Donaldson, 1971); 16.9% (14/83) in zebu cross cows in central Queensland (inferred LEM; Turner, 1982); and 25% (11/44) and 31.8% (14/44) in 50% and 75% Brahman cross heifers, respectively, in north Queensland’s dry tropics (inferred LEM; Holroyd et al., 1993). In two international studies, embryo mortalities in B. taurus beef heifers in temperate environments accounted for more than 30% (Diskin and Sreenan, 1980) and 25–28% (Roche et al., 1981) of reproductive inefficiency in heifers. EEM accounted for about 75–80% of all embryo and foetal mortalities, with the greatest mortalities occurring between days 8 and 18 after fertilisation (Diskin and Sreenan, 1980; Roche et al., 1981). Of the remaining mortalities, most estimates suggest that about 10–15% mortality occurs at or near the time of implantation (days 25–45) and 5–8% mortality between placental attachment and birth (Sreenan and Diskin, 1983). Therefore, the reported levels of embryo mortality in tropical beef cattle genotypes in northern Australia are comparable with those levels reported for B. taurus beef heifers in temperate environments and represent a significant source of reproductive inefficiency.

4.3. Pre-natal (foetal) mortality

Foetal mortality occurs in the period from the 45th day of gestation to the commencement of parturition (Committee on Bovine Reproductive Nomenclature, 1972). Foetal mortalities reported in northern Australia include data collected in the (i) mid-1980s to mid-1990s—1–13%; and (ii) mid-1990s to end of 2005—2–8% (Table 2).
Table 2
Pregnancy rate (%), calving rate (%), branding/weaning rate (%), losses to branding (%), prenatal (%), perinatal (%) and postnatal (%) mortalities, and total mortalities from confirmed pregnancy to weaning.

| Author                        | Genotype Class          | PR-wet | PR-dry | PR-herd | CR % | B/W % | Pre % | Peri % | Post % | Total CP-W |
|-------------------------------|-------------------------|--------|--------|---------|------|-------|-------|--------|--------|------------|
| Queensland—northern speargrass|                         |        |        |         |      |       |       |        |        |            |
| Holroyd et al. (1983)         | Brahman cross           | FCH    | 58     | 55      | 50   | 8     | 1–5   | 3–5    |        |            |
| Mature                        |                         | 61–96  |        | 57–93   | 57–92| 4–11  |        |        |        |            |
| Entwistle and Goddard (1984)  | *Bos indicus*           | FCH    | 19     |         |      |       |       |        |        |            |
| Mature                        |                         | 20     | 87     | 20–86   |      |        | 4–10  |        |        |            |
| Holroyd (1987)                | 1/2 Brahman             |        |        |         |      |       |       |        |        |            |
| 1/4 Brahman                   |                         |        |        |         |      |       |       |        |        |            |
| 1/2 Sahiwal                   |                         |        |        |         |      |       |       |        |        |            |
| 1/4 Sahiwal                   |                         |        |        |         |      |       |       |        |        |            |
| Holroyd et al. (1988a)        | *Bos indicus* cross     | FCH    | 28     | 39      | 37   | 8     | 22    | 11     |        |            |
| Mature                        |                         | 41–83  | 92–100 |        |      |       | 72    | 55     | 17     | 17         |
| Holroyd et al. (1988b)        | Brahma cross            |        |        |         |      |       |       |        |        |            |
| Hetzel et al. (1989)          | Droughtmaster           |        |        |         |      |       |       |        |        |            |
| Cordyce et al. (1990)         | Brahma cross            |        |        |         |      |       |       |        |        |            |
| Queensland—southern speargrass|                         |        |        |         |      |       |       |        |        |            |
| Coates et al. (1987)          | Hereford Cows           |        |        |         |      |       |       |        |        |            |
| Burns (1999)                  | Belmont Red cross       |        |        |         |      |       |       |        |        |            |
| Brangus Red cross             |                         |        |        |         |      |       |       |        |        |            |
| Queensland—Mitchell grass downs|                        |        |        |         |      |       |       |        |        |            |
| Holroyd et al. (1988c)        | Droughtmaster           |        |        |         |      |       |       |        |        |            |
| Hill et al. (2009)            | Brahman and Composite   |        |        |         |      |       |       |        |        |            |
| Queensland—Brigalow Carroll (1984)|                    |        |        |         |      |       |       |        |        |            |
| Coates et al. (1987)          | Belmont Red Cows        |        |        |         |      |       |       |        |        |            |
| Burns and Mannette (1990)     | Hereford Cows           |        |        |         |      |       |       |        |        |            |
| Burns et al. (1992a)          | Belmont Red Heifer      |        |        |         |      |       |       |        |        |            |
|                               | Hereford, Simmental     |        |        |         |      |       |       |        |        |            |
| Northern Territory (NT)—Darwin/Gulf Mc.Cosker and Eggington (1986) | Brahma cross |          |        |         |      |       |       |        |        |            |
| Mccosker and Eggington (1986) | Brahma cross             |        |        |         |      |       |       |        |        |            |

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| Study                          | Breed            | Sex    | Min       | Max       | Mean   | SD       | Range     |
|-------------------------------|------------------|--------|-----------|-----------|--------|----------|-----------|
| McCosker and Eggington (1986) | Brahman cross    | Mature | 16–68     | 100       | 52     | 49       | 23–79     |
| McCosker and Eggington (1986) | Brahman cross    | Mature | 40–50     | 91–98     | 93     | 91       | 65–82     |
| McCosker and Eggington (1986) | Brahman cross    | Mature | 8–25      | 8–50      | 100    | 100      | 29–64     |
| McCosker et al. (1991)        | Brahman cross    | Mature | 17–58     | 77–96     | 42     | 42       | 37–61     |
| O'Rourke et al. (1995)         | Droughtmaster    | All    | 21–25     | 25        | 15     | 15       | 10–30     |
| MacDonald et al. (1997)       | Brahman cross    | All    | 3.4–10.6  | 3.4–10.6  | 9.3    | 9.3      | 6.0–10.0  |
| Sullivan and O'Rourke (1997)   | Droughtmaster    | All    | 21–12     | 21        | 9.3    | 9.3      | 6.0–10.0  |
| Northern Territory—Victoria   | Droughtmaster    | All    | 21–12     | 21        | 9.3    | 9.3      | 6.0–10.0  |
| River District (VRD)           | Droughtmaster    | All    | 21–12     | 21        | 9.3    | 9.3      | 6.0–10.0  |
| Northern Territory—Barkly      | Brahman          | Mixed  | 84–76     | 84–76     | 8      | 8        | 2–10      |
| Tableland                      | Brahman          | Heifer | 84–76     | 84–76     | 8      | 8        | 2–10      |
| Northern Territory—Gulf of     | Brahman          | Mixed  | 71–76     | 71–76     | 54     | 54       | 13–31     |
| Carpenteria                    | Brahman          | Mixed  | 71–76     | 71–76     | 54     | 54       | 13–31     |
| Western Australia—Northern     | Mixed            | Cows   | 26–66     | 26–66     | 57     | 57       | 3–13      |
| Region                         | Mixed            | Cows   | 26–66     | 26–66     | 57     | 57       | 3–13      |
| Northern Territory—Monsoon     | Mixed            | Cows   | 49        | 49        | 25     | 25       | 0–25      |
| zone                           | Mixed            | Cows   | 49        | 49        | 25     | 25       | 0–25      |
| Kirby (1984)                   | Mixed            | Cows   | 49        | 49        | 25     | 25       | 0–25      |

PR-wet, pregnancy rate in lactating females; PR-dry, pregnancy rate in lactating females; PR-herd, whole herd pregnancy rate; CR, calving rate; LB, loss from confirmed pregnancy to branding; Pre, pre-natal mortalities; Peri, peri-natal mortalities; Post, post-natal mortalities; Total CP-W, total mortalities from confirmed pregnancy to weaning.
4.4. Peri-natal mortality

The peri-natal period is the period just prior to birth, during birth, and the first 28 days of life (Committee on Bovine Reproductive Nomenclature, 1972). However, there appears to be some differences in the literature with respect to the definition of this period. Holroyd (1987) referred to the peri-natal period as that period from the commencement of parturition until 48 h after the birth of the calf. This latter definition is used for the purposes of this review.

Reproductive studies in northern Australia reported peri-natal mortalities ranging from 2% to 12% (Table 2). Two percent and 10.2% peri-natal mortalities, respectively, were reported for a mixed aged Brahman herd (Brown, 1998) and 200 first-lactation 3-year-old Brahman heifers (Brown et al., 2003) grazing Mitchell grass pastures in extensive commercial production systems on the Barkly Tableland of the Northern Territory during the summer months where environmental temperatures were extreme (>40°C). In another study, peri-natal mortalities of 6% and 12%, respectively, were reported for the 2004 calving period for 4 and 5-year-old Brahman and Tropical Composite cows grazing drought affected Mitchell grass pastures in the tropical, semi-arid region of north-west Queensland where environmental temperatures in this production system during the summer months are extreme (>40°C) (Holroyd et al., 2005). In the following year, a combined mortality of 41% (167/407) was recorded for the same group of Brahman and Tropical Composite cows from parturition up to 15 weeks post-calving with 75% occurring within 24 h of birth (Holroyd et al., 2005). These losses were much higher than expected in this class of cattle grazing Mitchell grass pastures in this production system and a detailed clinical, pathological and biochemical investigation identified that gestational Vitamin A deficiency was an important contributor to these high peri-natal calf mortalities (Hill et al., 2009).

4.5. Post-natal mortality

The post-natal period extends from 48 h after the birth of the calf until weaning which typically averages around 5–7 months of age (Holroyd, 1987). Reports of post-natal mortalities from well designed studies have ranged from 0.3% to 15% (Table 2).

4.6. Acceptable levels of loss for specific reproductive categories from confirmed pregnancy to branding/weaning

In an attempt to determine what acceptable levels of mortality should be from confirmed pregnancy to branding/weaning in northern Australian herds, a brief comparison of South American reproductive studies was conducted. Pre-natal mortalities reported from a number of South American reproductive studies (mid-1980s–late 1990s), involving B. indicus, B. indicus × B. taurus cross and adapted B. taurus breeding herds, ranged from 5.3% to 17%: (1) Venezuela (Plasse et al., 1998 (7.9%); Linares and Rodriguez, 1983 (12.5%); Hoogesteijn et al., 1983 (17%); and Vera and Seré, 1985 (16%)); (2) Brazil (Vera and Seré, 1985 (8%)); (3) Colombia (Vera and Seré, 1985 (14%)) and (4) Bolivia (Plasse et al., 1993 (5.3%)). In addition, Plasse et al. (1998) reported a mean pre-weaning (birth to weaning) mortality of 4.9% (range 2.4 to 11.5%) from the same Venezuelan reproductive performance study above. The differences in the levels of mortalities reported for herds in northern Australia compared to those reported above for South American herds could be a reflection of the: extentiveness of the cattle operations; different levels of management and disease control; and different levels of accuracy in recording results in these two regions.

In well-managed herds in tropical northern Australia, with low exposure to reproductive diseases, Holroyd (1987) reported that an ‘accepted level’ of foetal and calf mortality in Brahman cross females (≥ 50% B. indicus) from confirmed pregnancy to weaning should be in the order of 12% and represents 5% pre-natal, 4% peri-natal and 3% post-natal mortality. Given the range of factors that may be responsible for these mortalities in northern Australia, any attempt to reduce mortalities below these levels may not be economically sound. While a number of the studies referred to in this review have reported levels of mortality of the same magnitude as the ‘acceptable levels’ just discussed, other levels of reported mortalities are well above these levels and subsequently have a significant impact on herd profitability. Therefore, these ‘acceptable levels of mortalities’ should be used as a benchmark in northern Australia and higher levels of mortalities warrant further investigation.

However, in many cases, the nature of extensive beef production systems in northern Australia has made it difficult to differentiate between pre-, peri- and post-natal mortalities. Reported mortality levels from confirmed pregnancy to weaning include 0–42% from the mid-1980s to mid-1990s and 3.4–25% in the mid-1990s to end-2004 (Table 2). While some of these reported mortalities are high, the reduction in losses over the past 25 years in this region is possibly due to the implementation of improved management strategies such as property development and cattle control; an increase in tropically adapted B. indicus content of herds; vaccination programs and better weaner, heifer, cow and bull management.

5. Infectious causes of reproductive inefficiencies

5.1. Fertilisation failure

While both Bovine Viral Diarrhoea/Pestivirus (BVDV) (Kirkland and Mackintosh, 2006) and Bovine Ephemeral Fever (BEF) (Kirkland, 1993), which are both endemic to northern Australia, have been reported as important infectious causes of fertilisation failure in beef cattle in this region (Table 3), the research required to determine if they are the major causes of fertilisation failure in this region has not been conducted. The extensive nature of the northern Australian beef cattle industry makes it difficult to determine the prevalence of fertilisation failure from these two diseases. Bovine herpes virus-1 (Infectious bovine rhinotracheitis virus caused by BHV-1.2b), which is a low-virulence strain (abortogenic form of this disease not confirmed in Australia), has also been reported to be
Table 3
Main infectious disease causes of fertilisation failure and embryonic, foetal and new born calf mortalities in northern Australia.

| Category   | Epidemiology                                                                 | Clinical signs                                                                 | Authors                        |
|------------|------------------------------------------------------------------------------|--------------------------------------------------------------------------------|--------------------------------|
| (1) Bacteria<br>
Campylobacter foetus<br>subspecies veneralis | Venereally transmitted by infected bulls during coitus<br><sup>ab</sup>          | Major cause of embryonic and foetal mortality and infertility<br> Late embryonic mortality<br> Foetal mortality—6th to 8th month of pregnancy<br> Prolonged interoestrus intervals<br> Sporadic abortion<br> Lack of clinical signs in bull | Ladds et al. (1973)<sup>a</sup>McCool et al. (1988)<sup>b</sup> |
| Leptospira hardjo, Leptospira pomona, Leptospira tarrassovi | Environmental contact with urine from infected cattle<br><sup>cd</sup> Main maintenance hosts—domestic and feral pigs for pomona and tarrassovi; bandicoots and introduced rodents for other serovars; sheep can act as reservoir for campylobacter<br><sup>d</sup> Overall seroprevalence of 35% (hardjo) and 17% (pomona)<br> Central Queensland (1980–1982)—1.9–33.2% (hardjo) and 0.8–8.1% (pomona)<br><sup>e</sup> | Acute, subacute or chronic<br> Sporadic or multiple abortions—5th to 9th month of pregnancy<br> Premature births, still births; birth of weak calves<br> Atypical mastitis/aglactica in herd | Elder et al. (1985)<sup>c</sup>Hallett et al. (1996)<sup>d</sup>Black et al. (2001)<sup>e</sup>McGowan (2003)<sup>f</sup> |
| (2) Protozoan<br>
Neospora caninum | Major cause of abortion in beef cattle herds worldwide and suspected in Australian herds for many years<br><sup>ghi</sup> Little known about epidemiology<br><sup>ijk</sup> Proposed transplacental (vertical transmission in infected herds from dam to daughter) and post-natal (horizontal transmission in uninfected cows/herds by a carnivorous dog/fox/dingo) definitive host<br> Transmission<br> Central Queensland beef cattle tested (1997)—seroprevalence of 14.9% suggests parasite endemic<br> Survey of 10 northern and one south-east Queensland dairy herds (2002) reported a mean seroprevalence of 25%<br> Majority of infected foetuses not aborted and while some infected calves are diseased at birth and die in the neonatal period with lesions similar to those of aborted calves, majority of congenitally infected calves are born healthy and persistently infected | Sporadic abortions<br> Mummification common<br> Abortions mainly diagnosed in mid to late gestation (3rd to 9th month of pregnancy)—observation may reflect difficulty of confirming losses due to Neosporosis in early gestation<br> Majority of infected foetuses not aborted and while some infected calves are diseased at birth and die in the neonatal period with lesions similar to those of aborted calves, majority of congenitally infected calves are born healthy and persistently infected | Dubey (1999)<sup>f</sup>Gunn et al. (2002)<sup>j</sup>Windsor et al. (2002)<sup>k</sup>Stoessel et al. (2003)<sup>j</sup>Clark et al. (1986)<sup>k</sup>Yule et al. (1989)<sup>j</sup>Riley et al. (1995)<sup>jm</sup> |
| Tritrichomonas foetus | Venereally transmitted by infected bulls during coitus<br><sup>klm</sup> Most prevalent in the prepuce of bulls >5 years of age<br> Endemic in extensive cattle areas of northern Australia<br><sup>k</sup> | Embryonic mortality<br> Sporadic abortions (1st to 5th month of pregnancy)<br> Pyometra<br> Prolonged interoestrus intervals<br> May be more common in older cows<br> Lack of clinical signs in bulls | Clark et al. (1986)<sup>k</sup>Yule et al. (1989)<sup>j</sup>Riley et al. (1995)<sup>jm</sup> |
| (3) Viruses<br>
Akabane virus | Biting midge (Culicoides brevitarsis) | Sporadic abortions (3rd to 9th month of pregnancy) precede birth of congenitally abnormal calves—major cause of congenital abnormalities—porencephaly, arthrogryposis (can cause dystocia), hydrancephaly (can result in blind ‘dunmmy’ calves)<br> Sporadic occurrence of stillbirths, premature births | Kirkland and Barry (1984)<sup>n</sup>Kirkland and Barry (1986)<sup>n</sup> |
| Bovine herpes virus-1<br>(Infectious bovine rhinotracheitis virus—abortogenic form of this disease has not been confirmed in Australia). | Majority venerally transmitted/some respiratory<br> Genital carrier state important<br><sup>op</sup> | Overseas<br> Frequent abortions (4th to 9th month of pregnancy preceded by outbreak of respiratory or conjunctival form of disease | Bitsch (1973)<sup>p</sup>Studdert (1989)<sup>q</sup> |
| Category                                      | Epidemiology                                                                 | Clinical signs                                                                                                                                                                                                 | Authors                  |
|-----------------------------------------------|------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|--------------------------|
| Bulls unable to serve and fertilisation failure seen in Australia (BHV1.2b) | 96% of bulls and 53% of cows seropositive<sup>a</sup>                        | Sporadic or multiple abortions                                                                                                                                                                                                                                  | Smith et al. (1993)<sup>f</sup> |
| Bovine pestivirus                              | Direct contact from persistently infected cattle is main method of transmission<sup>d,w</sup>  | Sporadic occurrence of stillbirths, birth of weak non-viable neonates May cause embryonic mortality Australia Mild rhinitis and conjunctivitis Vulvovaginitis, mild to severe chronic endometritis and in some cases salpingitis Shortened oestrus cycles (probably premature luteolysis associated with viral induced necrosis of corpus luteum) No adverse effects on female reproductive performance Reduced bull libido - clinical phase of infection | McGowan and Kirkland (1995)<sup>b</sup> Taylor and Rodwell (2001)<sup>f</sup> |
| Bovine Viral Diarrhoea Virus (BVDV)            | Endemic in Australian beef cattle herds<sup>u</sup> Seroprevalence of 37–44% in beef cattle<sup>u</sup> | Fertilisation failure Embryonic mortality Usually sporadic, occasional multiple abortions (1st to 4th month of pregnancy) Occurrence of foetal mummification, stillbirths, birth of weak, frequently stunted neonates, congenital malformation (skin, eyes, brain, in particular cerebellum) Deaths and chronic ill-thrift in weaners | Taylor et al. (2006)<sup>u</sup> Kirkland and MacKintosh (2006)<sup>p</sup> |
| Bovine Ephemeral Fever (3-day sickness)        | Mosquito (Culex annulirostris)<sup>y</sup> Summer and early autumn<sup>wa</sup> Occurs enzootically/endemically in much of Australia<sup>wa</sup> Morbidity rate usually 35% but 100% in highly susceptible populations<sup>y</sup> | Abortion—generally during the last half of gestation but have been described between the 2nd to 7th month of pregnancy Temporary sterility in bulls<sup>wa</sup> | Chenoweth and Burgess (1972)<sup>wa</sup> Uren (1989)<sup>a</sup> Kirkland (1993)<sup>f</sup> |
Campylobacteriosis in northern Australia have been reported as a cause of embryo mortality. BVDF causes fertilisation failure through ovariotomy and subsequent failure of ovulation or delayed ovulation and abnormal semen or deterioration of sperm quality (McCowan and Kirkland, 1995; Kirkland and MacKintosh, 2006). BVDF infections are considered to be the most important viral pathogens of cattle in Australia as they also cause embryo mortality, abortion, foetal mummification, congenital malformations, stillbirths and mortalities at any age from birth (Kirkland and MacKintosh, 2006). These losses follow infection of susceptible females by direct contact with persistently infected cattle between the time of mating through to 180 days of gestation (McCowan and Kirkland, 1995; Kirkland and MacKintosh, 2006). Level of cattle control/management may be a major factor associated with the incidence of BVDF in northern Australian herds. Increased segregation of younger breeding females can lead to an increase in proportions of herds susceptible to BVDF. A combination of serological survey, management and vaccination can minimise losses due to BVDF.

BEF can result in viraemia, fever and generalised inflammation (Chenoweth and Burgess, 1972; Uren, 1989; Kirkland, 1993) which can also cause temporary sterility in bulls (Chenoweth and Burgess, 1972). BEF occurs as seasonal outbreaks associated with rain periods in the region (Kirkland, 1993). In areas of northern Australia with more reliable wet seasons, outbreaks occur in most years and most cattle are immune before becoming adults (Kirkland, 1993). However, in regions where outbreaks of BEF are intermittent, large numbers of adult cattle can be affected (Kirkland, 1993). Vaccination of cows and bulls can be used to reduce the impact of the disease.

Genital infection with BHV-1.2b occurs in both sexes and is a more frequent manifestation of this herpesvirus infection in cattle grazing pasture (Studdert, 1989). Infection may result in the development of vesicles, pustules and erosions or ulcers in the mucosa of the vulva and vagina (infectious pustular vulvovaginitis (IPV)) within a few days of mating or on the penis and prepuce (infectious pustular balanoposthitis (IPB)) (Bitsch, 1973; Studdert, 1989). The pustules can progress to ulcers with a mucopurulent discharge and may prevent a bull from serving. Further, a proportion of infected bulls will also excrete virus in their semen (Bitsch, 1973; Studdert, 1989). In turn, infected semen can infect susceptible females (Bitsch, 1973; Studdert, 1989). Losses are incurred due to epidemics of infertility due to IPV in heifers/cows and IPB in bulls.

5.3. Pre-natal mortality

Infectious causes of foetal mortalities in northern Australian herds include bacteria (C. foetus subspecies veneralis, Leptospira hardjo and L. pomona); protozoa (Neospora caninum, T. foetus) and viruses (Akabane virus, BVDF and BEF) (Table 3). The relative impact of these diseases on the reproductive performance of northern Australian herds is not clear.

BVDF, BEF, campylobacteriosis and trichomoniasis have been previously discussed. The prevalence of leptospirosis in Australian animals, domestic or otherwise, has been reported (Carroll and Campbell, 1987; Pitt and Houston, 1999; Black et al., 2001). While leptospirosis is often considered as the most likely cause of increased mortalities between pregnancy diagnosis and weaning, its significance as a cause of reproductive failure in Australian beef cattle herds has been somewhat circumstantial and as a result has come under increased scrutiny (Smith et al., 1997). While the evidence linking L. pomona to foetal mortalities is much more substantial, the prevalence of infection with this serovar is significantly less than that recorded for L. hardjo (Hallett et al., 1996). As L. hardjo does not normally infect the foetus, other infectious agents such as N. caninum could be a more significant cause of bovine foetal mortality (Smith et al., 1997). While L. tarrassovi has been demonstrated to infect cattle in northern Australia, the significance of this infection has not been determined (Hallett et al., 1996).

N. caninum is recognised as a major cause of bovine abortion in both dairy and beef cattle herds worldwide. Despite the considerable global attention this parasite has attracted, little is known about the epidemiology of N. caninum in Australian beef cattle herds (Windsor et al., 2002). N. caninum abortions are mainly diagnosed in mid to late gestation, however, this observation may reflect the difficulty of confirming losses due to neosporosis in early gestation. The majority of infected foetuses are not aborted and while neosporosis causes some neonatal mortalities with lesions similar to those of aborted calves, the majority of congenitally infected calves are born healthy and are persistently infected (Windsor et al., 2002).

The highest prevalence of calf abnormalities due to Akabane virus tends to occur after cows are infected.
with the virus between 3 and 6 months of pregnancy. The virus infection is recognised as the major congenital cause of arthrogryposis and hydrancephaly and can result in abortions and stillbirths (Kirkland and Barry, 1984). During an epidemic, these abortions and stillbirths are often associated with the birth of calves unable to stand (encephalopathy); calves with varying degrees of arthrogryposis (one or more limbs affected and often associated with dystocia); and blind 'dummy' calves (hydrancephaly) (Kirkland and Barry, 1986). Though strategic use of inactivated vaccines in susceptible animals before the time of potential exposure to vector activity had been recommended to control the impact of Akabane virus, the extensive nature of the northern Australian beef industry limits their use. Further, a vaccine was developed in Australia and made commercially available to the industry but due to lack of uptake it was withdrawn.

5.4. Peri- and post-natal mortalities

The main infectious causes of peri- and post-natal mortalities in northern Australian herds are bacteria (L. hardjo and L. pomona), protozoa (N. caninum), and viruses (Akabane virus, BVDV) (previously discussed; Table 3). Once again, the relative impact of these diseases on the reproductive performance of northern Australian herds is not known.

Calf diarhoea does not appear to be a major cause of peri- or post-natal mortalities in northern Australian beef cattle herds although one case has been reported by Mitchell et al. (1981). Mortalities (14/21) were observed in both Hereford and B. indicus cross calves during summer on the Barkly Tablelands (Mitchell et al., 1981). There appeared to be a strong association between calves with diarrhoea and those that were heat stressed. This observation is supported by a North American study (Clement et al., 1995) where hot humid summer weather was identified as an important risk factor associated with an increased incidence of calf mortality due to diarrhoea. Eimeria spp. which causes coccidiosis in calves about 21 days after birth or a stress event can be an important cause of diarrhoea in calves (Parker et al., 1986). This condition occurs commonly in calves post-weaning under northern Australian conditions (Parker et al., 1986). Acute undifferentiated diarrhoea, caused by an interaction between enteropathogenic bacteria, viruses and protozoa, the colostral immunity of the animal and the effects of the environment, has been reported to cause mortalities in intensively reared newborn calves under 30 days of age in North America (McDonough et al., 1994). Collectively, enterotoxigenic Escherichia coli, verocytotoxigenic E. coli, necrototoxic E. coli, coronaviruses, and Cryptosporidium spp. have been reported to be responsible for 75–95% of infections in neonatal calves worldwide (Kodituwakku and Harbour, 1990). However, these infectious agents are not readily differentiated on clinical findings alone which include dehydration, emaciation and a fluid filled intestinal tract with no other obvious gross lesions (McDonough et al., 1994). The incidence of clinical disease due to these pathogens under extensive northern Australian conditions is unknown as most calves are born late in the calendar year generally during the start of the monsoonal wet season when cattle are not handled.

6. Climatic and nutritional causes of reproductive inefficiencies

6.1. Extended periods of low-rainfall

Extended periods of low-rainfall with associated nutritional deficiencies frequently occur across and within years in northern Australia. One consequence of prolonged periods of drought in Australia can be hypovitaminosis A (NSW Dept. of Primary Industries, 2003; Holroyd et al., 2005). In a Canadian study, Rode et al. (1995) reported that hypovitaminosis A could be responsible for decreased paired testes weight, sperm quality (morphology), sperm production and epididymal sperm reserves. These conditions can potentially cause fertilisation failure (Rode et al., 1995) and embryo mortality (Ross et al., 2000).

Acute negative energy balance in intensively managed North American cattle herds has been reported as a cause of embryo mortality (Vanroose et al., 2000: Bilodeau-Goesels and Kastelic, 2003). Periods of acute negative energy balance occur on an annual basis in tropical regions of Australia and as a consequence could be associated with embryo mortalities in beef cattle herds in these regions.

Drought conditions in north-east Queensland and the Northern Territory, respectively, have caused both pregnant heifer and cow mortalities resulting in foetal and calf mortalities from confirmed pregnancy to branding/weaning. Fordyce et al. (1990) reported that, of the 31% of foetal and calf mortalities from confirmed pregnancy to weaning, 22% were a result of cow mortality as a consequence of drought. Similarly, McCosker et al. (1991) also reported foetal and calf mortalities from confirmed pregnancy to weaning of 7.5% and 11.6%, respectively, in Brahman cross cows and heifers, due to drought.

In a recent northern Australian study (Hill et al., 2009), of the 41% of mortalities reported from parturition up to 15 weeks post-calving in Brahman and Tropical Composite genotypes in the tropical semi-arid region of north-west Queensland, 87% were reported as being probably due to gestational Vitamin A deficiency as a consequence of extended periods of drought.

6.2. Nutritional factors

A number of nutritional factors have been implicated in causing embryo mortality and may be relevant to northern Australian production systems.

Prolonged dry periods/drought or after periods of starvation can cause cattle to consume plants containing toxic agents that can cause embryo mortality. In Australia, McKenzie (2002) reported that plants containing high nitrate levels can be responsible for embryo mortalities.

Gartner et al. (1980) reported widespread areas of copper deficiency in south-eastern Queensland, northern coastal and central Queensland while Wesley-Smith and
Ford (1982) reported copper deficiencies in the north Queensland wet tropics, Cape York, the West Kimberleys and the Top End of the Northern Territory. The main signs of copper deficiency observed included a stiff-legged, humped-back appearance in suckling calves (3–6 months) while ill-thrift was the dominant feature in weaner and yearling cattle with adults not obviously affected (Gartner et al., 1980). In contrast, overseas studies reported embryo and foetal mortalities in sheep (Mills and Delgarno, 1972) and cattle (Graham et al., 1995) as a consequence of severe copper deficiency.

This review has repeatedly reported that prolonged dry periods and drought regularly occur in northern Australia. These situations require the feeding of hay and other supplements to minimise cattle losses. Hay can contain toxins such as mycotoxins (Osweiler, 1990) which includes zearalenone, an aflatoxin reported to be responsible for early abortions in cattle fed hay (Kallela and Ettala, 1984; Zavy, 1994).

Further, whole cottonseed and cottonseed meal is regularly fed as a cattle supplement in northern Australia. Gossypol contained in cottonseed has been reported to impair the fertility of some natural service dairy bulls in North America by decreasing sperm production, sperm motility and the proportion of normal spermatozoa (Risco, 2000). While a number of additional studies (Gu et al., 1990, 1991; Lin et al., 1994; Zavy, 1994) have also implicated gossypol and one of its major metabolites (gossypolone) in causing embryo mortalities, one Australian study (Cusack and Perry, 1995) found no effect. Therefore, given the amounts of cottonseed meal and whole cottonseed that are used as a cattle feed supplement in northern Australia and the lack of any reports of gossypol causing significant reproductive inefficiencies in this region, it would appear that cottonseed meal and whole cottonseed can continue to be fed as supplements.

6.3. High environmental temperature and humidity conditions

High environmental temperature and humidity conditions commonly occur over large areas of northern Australia. Heat stress conditions can compromise steriodogenesis (Zeron et al., 2001), reduce oocyte quality and the viability of oocytes (Hansen, 2002), cause a decline in oocyte competence (Al-Katanani et al., 2002), and a decrease in fertilisation rate (Post, 1980) and embryo survival in non-tropically adapted and tropically adapted B. taurus, B. indicus and B. indicus cross heifers and cows (Post, 1980; Turner, 1982; Holroyd et al., 1993; Smith and Stevenson, 1995). Brown et al. (2003) reported that heatstroke/hyperthermic shock in calves during periods of extreme environmental temperatures was another factor that could cause peri-natal mortalities (0.5% mortality of calves) in northern Australia. In earlier studies, hyperthermia was reported to cause post-natal mortalities in unadapted genotypes (Daly, 1971; Entwistle, 1974). However, this was not considered a problem in B. indicus genotypes (Holroyd, 1987) unless calves were predisposed to other conditions such as Vitamin A deficiency (Holroyd et al., 2005).

6.4. Other environmental factors

Other environmental factors reported to cause post-natal mortalities include dingo predation (Daly, 1971; Entwistle, 1974); and mustering, mismothering and mis-adventure (Brown et al., 2003).

7. Male factor causes of reproductive inefficiencies

Fertilisation failure can be due to the lack of normal viable sperm reaching the ova. Physical defects or low libido in bulls can be a cause of fertilisation failure in B. indicus and B. indicus cross heifers/cows in both single- and multiple-sire herds (Entwistle and Fordyce, 2003). Percent morphologically normal sperm, an easily measured and moderately to highly repeatable trait in bulls once they have reached sexual maturity, has been reported to be one of the main factors affecting calf output of individual bulls in northern Australia under multiple sire mating conditions (Fitzpatrick et al., 2002; Holroyd et al., 2002; Fordyce, 2005). About 35% of bulls tested in this environment have been reported to have less than 70% normal sperm and 15% produced less than 50% normal sperm (Fitzpatrick et al., 2002; Holroyd et al., 2002). The level of additive genetic variance for sperm morphology is unknown.

Early research reported that the prevalence of embryo mortality and subsequent higher return rates in breeding females was almost doubled when semen from low-fertility sires was used compared to semen from high fertility sires (Kidder et al., 1954; Bearden et al., 1956), with no or equivalent effect of sire breed or sire within breed (Bishop, 1964; Wijeratne, 1973; Markusfeld-Nir, 1997). Further research has reported that bulls can vary in their influence on the time of embryo/foetal mortality ranging from up to day 24 of pregnancy (Humblot and Denis, 1986), 5–9 weeks (Starbuck et al., 2004), day 38–day 90 of gestation (López-Gatius et al., 2002) and from fertilisation to term (Eid et al., 1994).

DNA chromatin aberrations in sperm that occur during spermiogenesis appear to be the best candidates for uncompensable deficiency (D’Occhio et al., 2007). These aberrations have been implicated in cases of reproductive inefficiency through early embryo mortality (D’Occhio et al., 2007).

Variations in epididymal sperm proteins and other marker compounds such as P25b—a glycosylphosphatidylinositol (sperm-zona pellucida interactions) (Frenette and Sullivan, 2001; Sullivan, 2004), aldose reductase (modulate utilisation of carbohydrates as osmoregulators or modulators of energy sources used by spermatozoa) (Frenette et al., 2003), macrophage mobility inhibiting factor (flagellum motility) (Frenette et al., 2003) and platelet activation factor (sperm motility) (Brackett et al., 2004) have been implicated in bull subfertility. Further, the presence of a family of heparin binding proteins (HBP) on sperm has been reported to be useful in predicting a measure of a bull’s potential fertility (Bellin et al., 1994, 1998). Heparin-binding proteins (HBP) are produced by the bull’s accessory glands, secreted into the seminal fluid
(Nass et al., 1990) and upon ejaculation bind to sperm (Miller et al., 1990). The addition of HBP to epididymal sperm induces heparin-stimulated acrosome reactions (Miller et al., 1990). Bellin et al. (1994) reported that groups of bulls with the greatest affinity for HBP-B5 in sperm membranes, but not in seminal fluid, had greater fertility than did groups with other HBP-B5 profiles. However, a study of tropically adapted bulls in northern Australia questioned the efficacy of this association as bulls without detectable HBPs on sperm membranes sired as many calves as those with HBPs (Fitzpatrick et al., 2002). As well, only 7% of bulls were identified without HBP in their sperm membranes (Fitzpatrick et al., 2002).

8. Female factor causes of reproductive inefficiencies

8.1. Follicular waves and development

Conception rates, a consequence of both fertilisation rates and embryo survival, are reported to be significantly higher in heifers/cows with three follicular waves compared to two follicular waves: 96% versus 70% in Bos taurus beef cattle (Ahmad et al., 1997—pubertal heifers and multiparous lactating cows); 100% versus 82% and 95% versus 58% in B. taurus beef and dairy cows (Inskeep, 2002); and 81% versus 63% in B. taurus dairy cattle (Townson et al., 2002)—lactating, multiparous cows). Brahman (B. indicus) cattle have been observed to have up to four large follicles (or waves) occurring in sequence during an oestrous cycle (Rhodes et al., 1995—post-pubertal, nulliparous heifers) compared to generally two in B. taurus dairy cattle (Ginther et al., 1989c—nulliparous heifers) and sometimes three in B. taurus dairy × beef cattle (Savio et al., 1988—post-pubertal, nulliparous heifers) and B. taurus dairy cattle (Sirois and Fortune, 1988—nulliparous heifers). It has been reported that the proportion of animals with two or three waves per cycle varies between herds (Sirois and Fortune, 1988 (nulliparous heifers); Ginther et al., 1989a (nulliparous B. taurus dairy heifers)) and is repeatable for individual Brahman heifers (Rhodes et al., 1995) and other B. taurus heifers and cows (Ginther et al., 1989b—post-pubertal, nulliparous dairy heifers; Ahmad et al., 1997—post-pubertal beef heifers and multiparous lactating beef cows), and varies with nutrition (Murphy et al., 1991) and body condition (Burke et al., 1995). The growth of the ovulatory follicle can be longer around the time of maternal recognition of pregnancy (Zavy, 1994) than the time of maternal recognition of pregnancy in cattle (Varner et al., 1983) by affecting sperm transport and maintenance of pregnancy (Edlington, 2000). In a study of Santa Gertrudis females from four southern Queensland properties, sire effects for cervical size were found to be significant and a heritability of 0.60 ± 0.27 estimated, which would make selection for this trait feasible (Finch et al., 2003).

In a northern Australian study, Hetzel et al. (1989) reported that old B. indicus infested cows (>13 years) conceived at lower rates than young cows. Hetzel et al. (1989) concluded that this was probably a consequence of the higher number of reproductive tract abnormalities that occur with increasing age, as reported by Christensen (1980). This conclusion is supported by reports from dairy and beef cattle studies that indicate that increased age and parity of the uterine environment can result in oviduct obstructions, abnormal ova, ovarian adhesions, endometriosis and high plasma progesterone concentrations just prior to oestrus affecting ova development and all can potentially have an adverse affect on fertilisation rate (Ayalon, 1969; Biggers, 1969; Roche et al., 1981). These results suggest that both selection for cervical size and shape in conjunction with the culling of cows with abnormal reproductive tracts along with culling of non-performing and older breeding females should alleviate these problems and reduce reproductive inefficiencies.

8.2. Size and shape of the cervix

Size and shape of the cervix has been reported to affect pregnancy rate in B. indicus and B. indicus derived cattle (Gonzalez et al., 1983; Edlington, 2000) and B. taurus cattle (Varner et al., 1983) by affecting sperm transport and maintenance of pregnancy (Edlington, 2000). In a study of Santa Gertrudis females from four southern Queensland properties, sire effects for cervical size were found to be significant and a heritability of 0.60 ± 0.27 estimated, which would make selection for this trait feasible (Finch et al., 2003).

Reports from intensive dairy and beef cattle studies have implicated a number of physiological impairments causing embryo and foetal mortalities: (1) impaired functional CL during the first 200 days of gestation (Niswender et al., 2000); (2) embryo—maternal asynchrony at or near the time of maternal recognition of pregnancy (Zavy, 1994) as a result of oestradiol-to-progesterone ratio imbalances (Pope, 1988) caused by restraint stress (Wiebold et al., 1986), variability in progesterone synthesis between animals (Ashworth et al., 1984), thermal stress (Biggers et al., 1987), age (Blockey et al., 1975) and undernutrition (Cumming et al., 1971; Parr et al., 1982); (3) impaired local uterine immune system resulting in bacterial infection of the uterus with inflammation directly or indirectly affecting sperm and embryo viability (Zavy, 1994); (4) failure of the anti-luteolytic interferon-τ (IFN-τ) secretory mechanism by the conceptus or the inadequate reaction of the endometrium to IFN-τ around the time of maternal recognition of pregnancy at about days 14–19 (Mathialagan and Roberts, 1994; Wolf et al., 2003); and (5) impaired interactions between the conceptus and its dam which requires a number of cytokines, involved in immune implantation processes, and growth factors, involved in growth and differentiation of the conceptus (Martal et al., 1997). While the relevance of these effects to northern Australian beef production is unknown, they have been included
in this discussion for future reference when considering possible causes of reproductive inefficiencies in this region.

8.4. Dystocia

Generally, dystocia has not been considered to be a major cause of foetal mortalities in northern Australian beef cattle herds but there are some exceptions. The reported low incidence of dystocia in tropical environments (Entwistle, 1983; Holroyd, 1987) reflects: the genotypes used as B. indicus (Cartwright et al., 1964) and tropically adapted B. taurus (Burns et al., 1992a; Herring et al., 1996) genotypes have the maternal ability to produce smaller calf sizes; first calving generally occurring at three rather than 2 years of age; generally lower levels of nutrition occurring during late pregnancy (reduced calf size); and possibly that a degree of natural selection is operating as under extensive grazing conditions with no calving supervision the final result of dystocia is usually death of both dam and calf (Entwistle, 1983; Holroyd, 1987). However, when dystocia problems have been observed in northern Australian environments they have been generally associated with foeto-pelvic disproportion and related to the use of: yearling mating in British breed heifers in southern areas of northern Australia (Norman, 2003); large European breeds such as the Simmental in central Queensland (Burns et al., 1992a); and Charbray bulls mated to primiparous Brahman heifers to calve at 3 years in a Northern Territory study in which approximately 4% of calves were lost as result of dystocia (Brown et al., 2003).

9. Breed-related causes of reproductive efficiency

Brown et al. (2003) reported peri-natal mortalities of 1% in Brahman females as a result of the birth of small and weak calves that could not rise and suckle. Abandoned calves, maternal mortalities, congenital abnormalities and weak calves at birth contributed to an overall higher rate of peri-natal and post-natal mortalities in Sahiwal than in Brahman genotypes (Holroyd, 1987). One of the most important factors causing mortalities was bottle teats with the prevalence higher in Sahiwal compared to Brahman genotypes and increased with advancing age (Frisch, 1982; Entwistle and Goddard, 1984; Holroyd, 1987). Holroyd (1987) suggested that the more nervous temperament of the Sahiwal compared to the Brahman (Fordyce, 1985) could have been part of the reason for the higher mortalities observed during the peri-natal period. In a subtropical environment, Burns et al. (1992a) reported higher post-natal calf mortality in Hereford calves compared with Belmont Red calves. Reductions in post-natal mortality as the B. indicus (Frisch, 1973) or tropically adapted B. taurus (Frisch, 1973; Burns et al., 1992a) content of the calves increased suggests that post-natal calf survival in tropical environments may partially reflect increasing environmental adaptation.

10. Genetic factors associated with reproductive efficiencies

Potential genetic influences on fertilisation failure and embryo and foetal mortalities also exist that could be relevant to northern Australia. Research suggests that impaired IFN-γ gene expression in the trophoectoderm layer from the time of blastocyst hatching (days 8–9) until implantation may retard successful maternal recognition of the embryo around day 15 of pregnancy in the cow (Mathialagan and Roberts, 1994). In addition, genetic differences in the fertilisation rates of sires and a genetic effect of the dam and her sire in the dam’s capacity to maintain pregnancy have been observed (Bar-Anan et al., 1980). Further, chromosomal abnormalities may occur either due to a hereditary factor, or because of spontaneous changes during gametogenesis, at fertilisation or during embryo development and not related to the chromosomal make up of the sire or dam (King, 1990).

Both between and within breed differences for both tropically adapted and non-adapted genotypes have been reported for the association between a lower fertilisation rate with the high ambient temperatures and high humidity experienced in the summer months in tropical environments (Post, 1980; Smith et al., 1982). Holroyd et al. (1993) also reported that more embryo mortalities occurred in 50–75% B. indicus content heifers that were lighter and had significantly higher rectal temperatures. This apparent genetic variation should allow the selection of sires in northern Australia to produce daughters with superior heat tolerance resulting in subsequent low fertilisation failure rates and embryo mortalities (Post, 1980; Smith et al., 1982; Turner, 1982; Holroyd et al., 1993).

The intense selection pressure in B. taurus cattle for growth and fertility in temperate environments has not occurred in B. indicus genotypes in harsher tropical environments where selection emphasis has been generally placed on survival. The resultant higher genetic variation that exists for fertility is reflected in the higher, but moderate, heritability estimates reported for B. indicus cattle (Deese and Koger, 1967; Seebeck, 1973; Cruz et al., 1978; Thorpe et al., 1981; Turner, 1982) than for B. taurus cattle (Davenport et al., 1965; Dearborn et al., 1973). The hypothesis that female fertility can be indirectly improved by selection on male fertility traits (Land, 1973) is supported by favourable genetic correlations in sheep (Walkley and Smith, 1980) and cattle (Mackinnon et al., 1989). Changes in female reproductive characteristics by selection in the prepubertal ram for high LH response to GnRH challenge were demonstrated in sheep (Evans et al., 1989) and may exist in cattle as there is genetic variation (h² > 0.5) between young bulls in their ability to secrete testosterone in response to GnRH stimulation (Mackinnon et al., 1991). These genetically influenced hormonal control mechanisms coupled with reports that fertility is repeatable (Rudd et al., 1976; Goddard, 1980; Seifert et al., 1980; Entwistle, 1983, 1984; Winter et al., 1985; Mackinnon, 1988; Mackinnon et al., 1987; O’Neill et al., 2000) suggest that an opportunity exists to genetically improve calf output in tropical genotypes in northern Australia, partly by reducing reproductive inefficiencies.
11. Unknown causes of reproductive inefficiencies

Up to 1983, most causes of foetal and calf mortalities in northern Australia were unknown which was a reflection of the extensive nature of the beef cattle industry in this region and the lack of any intensive investigations into these mortalities (Entwistle, 1983). Since 1983, the cause of 35.9% of all peri-natal calf mortalities in B. indicus derived cattle in a north Queensland study were unknown (Holroyd, 1987). Pre-natal and post-natal calf mortalities due to unknown causes were reported for Hereford and Belmont Red genotypes in a two-phase study in central southern Queensland (Coates and Mannetje, 1990; Table 2). A 3.6% peri-natal calf mortality that was classed as due to unknown causes was observed in Brahman calves in the Northern Territory (Brown et al., 2003). Additional northern Australian studies have also reported pre-natal and calf mortalities due to unknown causes (Fordyce et al., 1990; Brown et al., 1994; Schlink et al., 1994; Brown, 1998). There would appear to be little progress in determining causes of pre-natal and calf mortalities from confirmed pregnancy to weaning since 1983. This situation reflects the difficulties of investigating causes of mortalities in extensive herds because of the inability to observe cattle on a daily basis and the remoteness from laboratories.

12. Conclusions, implications and recommendations for the northern Australian beef industry

In this review, foetal and calf mortalities from confirmed pregnancy to weaning and the causes of these mortalities were reported over an approximate period of 25 years. This review highlights the difficulty in accurately estimating foetal and calf mortalities, their causes and their economic impact in northern Australia. As a consequence, until more accurate estimates of the magnitude and causes of these losses are obtained, it is also difficult to make recommendations on research and management strategies to reduce these mortalities.

This review also identified variation in terminology in both scientific and beef industry literature. Therefore, there is an urgent need to standardise bovine reproductive/production language to enable objective comparisons and discussion.

From the data reported in this review, the reproductive benchmark level for weaning rate in tropically adapted B. taurus, B. indicus and B. indicus cross breeding females in northern Australia is 75–80%. Therefore, from the partitioned areas of reproductive losses reported in this review, tropically adapted breeding females recording losses from fertilisation failure (>10%), EEM (30%) or LEM (10%) may subsequently conceive late in a 3–6 months mating season. However, when losses in these breeding females occur later in the mating season due to pre-natal mortalities, only limited conceptions may occur during the same 3–6 months mating season.

This review establishes that many herds are not achieving the benchmark levels identified above. It highlights substantial opportunities in northern Australia to increase the rate of established pregnancy per cycle in tropically adapted B. taurus, B. indicus and B. indicus cross breeding female cattle, with specific emphasis on early embryo mortality and mortalities during the peri- and early post-natal periods.

Viable opportunities to increase conception rates per cycle through reducing both fertilisation failure and embryo mortality may develop from methods to more readily diagnose genetic, disease, nutritional, stress and other causes of fertilisation failure and embryo mortality. Genetic improvement opportunities may include both selection and gene expression management for improved competence and viability of ova and embryos through reducing chronic and acute dietary restrictions and heat stress effects, increasing follicular waves within oestrus cycles (3–4 versus 2), reducing the post-insemination inflammatory reaction to spermatozoa and embryos within the reproductive tract of the cow and control of IFN-γ secretion during the period of maternal recognition of pregnancy and hormonal influences on oestrus, fertilisation and embryo survival. Fertilisation success and embryo survival may increase through a better understanding and control of factors that impact on sperm function such as vitamin A sufficiency, genetic variation (for compensable and non-compensable sperm morphology traits), seminal plasma proteins, sperm chromatin structure and chromosomal abnormalities.

This review also identifies that the incidence of the large range of causes of peri-natal mortality rates is poorly understood. Cost-effective management of foetal and calf mortality between confirmed pregnancy and weaning may be achieved by better diagnostic procedures and understanding of the epidemiology of the causative infectious diseases, by targeted nutritional management and also potentially by genetic selection.

A range of methods, based on current knowledge, exists that can be implemented by beef producers in northern Australia to reduce reproductive inefficiencies in tropically adapted B. taurus, B. indicus cross and B. indicus heifers and cows:

1. General management practices:
   (i) Select female cattle for future breeding at all ages if they rear calves to weaning and do not have phenotypic attributes that may contribute to calf loss, e.g., bottle teats, poor maternal ability and temperament.
   (ii) Manage mating and weaning to achieve lactation when nutrition is adequate and when cows and calves are able to tolerate prevailing climatic stresses (temperature extremes and inclement weather).
   (iii) Manage the metabolic status of animals by: ensuring adequate feed and water quality, quantity and access are available and preventing specific nutrient deficiencies and minimising handling stress, especially in the latter half of pregnancy.
   (iv) Use vaccines to control reproductive diseases, especially campylobacteriosis and pestivirus.
cination against Leptospirosis may also reduce losses in some situations. Further, vaccination of cows pre-calving and calves during suckling against clostridial diseases will prevent mortalities due to tetanus and botulism.

(v) Control predators where there is a risk that these may cause significant calf mortality.

(2) Specific management programs:

(i) Bulls

i. Use bulls that have passed a Bull Breeding Soundness Evaluation (BBSE) prior to their initial mating.

ii. Join bulls at 2 years old and cull them at 7 years old to minimise reproductive losses due to venereal diseases and subfertility and infertility and their association with older dominant bulls.

(ii) Heifers

i. Prevent dystocia in females calving at 2 years of age by initially preventing such matings and also, where possible, preventing matings of low liveweight maiden heifers. Select sires with traits indicative of calving ease when the information is available for use over maiden 2-year-old heifers. Segregate pregnant yearlings, if this occurs, for management to sustain them in moderate to good body condition throughout pregnancy and supervise calving and provide assistance.

ii. Nutritionally manage heifers from weaning to joining at 2 years old to ensure that all heifers have reached puberty and are cycling normally when first exposed to bulls to ensure early conceptions and subsequent early calving in the following year to maximise the probability of early reconceptions and the production of heavier calves at weaning.

iii. Control mate heifers for approximately 3 months or less depending on animal numbers and season, at 2 years old, and non-pregnant heifers subsequently culled.

iv. Manage heifers as a 3-year-old lactating group which may involve access to better quality pastures or supplementation and early weaning practices to minimise liveweight loss and mortalities and maximise reconceptions.

(iii) Mature breeders

i. Grazed at acceptable stocking pressures and receive strategic supplementation as a normal practice depending on the production system or during times of drought.

ii. Early weaning strategies employed as a normal management practice or during times when nutrition levels are compromised by drought.

iii. Cows that do not wean calves in consecutive years are culled.

iv. Depending on numbers of breeding females available (dependent on weaning rates), all non-lactating cows at the time of weaning are grouped together and pregnancy diagnosed to determine if pregnant and stage of pregnancy and cows that are non-pregnant or will calve late in the season are culled to remove inefficient non-pregnant or late pregnant breeding females to tighten up the herd calving patterns for ease of herd management during failed wet seasons and droughts.

v. Older cows are culled after weaning at 10 years of age to avoid survival and reproductive tract problems.

Finally, this comprehensive review has successfully identified a range of ‘factors that impact on the capacity of beef cattle females to conceive, maintain a pregnancy and wean a calf’ in northern Australia and the deficiencies in current knowledge with respect to these factors. The authors have also reported on the implications of the impact of these factors on conception to weaning and offered recommendations to reduce the impact of these factors. However, in recent times, a major concern of funding agencies has been the slow adoption rates of research results by the Australian beef cattle industry in general. Given this trend, coupled with the current global financial situation, increasing costs of production and labour shortages, the prospects for increasing the uptake of new information/strategies and technologies by the northern beef industry are limited. Therefore, there is an urgent need to consider new strategies that might improve the rate of adoption and implementation of research results and recommendations. While it is still critical to maintain the integrity of ‘government managed’ research facilities to conduct more basic research activities, it is also important to consider ‘new research models’. These ‘new research models’ should focus on more applied research/investigatory studies being conducted on ‘industry enterprises’ (‘satellite industry research enterprises’) across a range of different northern Australian production system environments in collaboration with strategically located ‘government managed’ research facilities. These government–industry collaborative/partnership research activities would facilitate a better understanding of factors limiting reproductive performance in these various production systems, the implementation of a standard industry language and measurement techniques to assist in describing and quantifying the impact of these factors and the subsequent adoption and implementation of strategies to improve overall herd productivity and profitability. Over time, the results from the more basic research activities conducted on ‘government managed’ research facilities could be evaluated/validated in these ‘satellite industry research enterprises’.

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References

Ahmad, N., Townsend, E.C., Dailey, R.A., Inskeep, E.K., 1997. Relationships of hormonal patterns and fertility to occurrence of two to three waves of ovarian follicles, before and after breeding, in beef cows and heifers. Anim. Reprod. Sci. 49, 13–28.

Ahmad, N., Schrick, F.N., Butcher, R.L., Inskeep, E.K., 1995. Effect of persistent follicles on early embryonic losses in beef cows. Biol. Reprod. 52, 1179–1185.

Al-Katanani, Y.M., Paula-Lopes, F.F., Hansen, P.J., 2002. Effect of season and exposure to heat stress on oocyte competence in Holstein cows. J. Dairy Sci. 85, 390–396.

Anon, 2008. Australian Bureau of Statistics, 2008. 1301.0—Year Book Australia. ISBN 0-7020-1785-X.

Ashworth, C.J., Sales, D.I., Wilmut, I., 1984. Patterns of progesterone secretion and embryonic survival during repeated pregnancies in Dam line ewes. In: Proc. 10th Int. Cong. Anim. Reprod. AI, vol. 2, p. 74.

Ayton, N., 1989. Final Report of Research and Comparative Studies of Repeat Breeders and Normal Cows and Heifers. Kimron Veterinary Institute, Beit Dagan (Israel), pp. 1–92.

Bar-Anan, R., Osterkorn, K., Kräusslich, H., 1980. Genetic effects on return intervals in cows. Livestk. Prod. Sci. 7, 225–233.

Beardow, R.J., Hansel, W., Bratton, R.W., 1956. Fertilization and embryonic mortality in rats of strains with histories of either low or high fertility in artificial breeding. J. Dairy Sci. 39, 312–318.

Bellin, M.E., Hawkins, H.E., Ax, R.L., 1994. Fertility of range beef bulls grouped according to presence or absence of heparin-binding proteins in sperm membranes and seminal fluid. J. Anim. Sci. 72, 2441–2448.

Bellin, M.E., Oyarzo, J.N., Hawkins, H.E., Zhang, H., Smith, R.G., Forrest, D.W., Sprott, L.R., Ax, R.L., 1998. Fertility-associated antigen on bull sperm indicates fertility potential. J. Anim. Sci. 76, 2032–2039.

Biggers, J.D., 1969. Problems concerning the uterine cause of abortion. J. Reprod. Fertil. Suppl. 8, 27–43.

Bigger, B.G., Geisert, R.D., Wettemann, R.P., Buchanan, D.S., 1987. Effect of temperature and exposure to heat stress on early embryonic development in the beef cow. J. Anim. Sci. 65, 645–650.

Carroll, A.G., 1984. Studies on reproductive performance of beef cattle in central Queensland. MSc thesis. James Cook University of North Queensland, Townsville.

Carroll, A.G., Campbell, R.K., 1987. Reproductive and leptospirology studies on beef cattle in Central Queensland. Aust. Vet. J. 64 (1), 1–5.

Carr, W.C., Dagan, J., Ellis Jr., G.F., Kruse, W.E., Crouch, E.K., 1964. Hybrid vigor in Brahman-Hereford crosses. Texas Agric. Exp. Stat. Tech. Monogr., Beit Dagan (Israel), pp. 1–92.

Chenoweth, P.J., Burgess, G.W., 1972. Mid-piece abnormalities in bovine semen following embryonic death. Aust. Vet. J. 48, 37–38.

Christensen, H.R., 1980. Biological causes of infertility. Proc. Symp. Fertility Tropic. Cattle. CSIRO, Rockhampton, pp. 8–13.

Clark, R.L., Dufuy, J.H., Parsonson, I.M., 1986. The frequency of infertility in cattle infected with Trittenichomonas foetus, var. brisbane. Aust. Vet. J. 63 (1), 31–32.

Clement, J.C., King, M.E., Salmon, M.D., Wittum, T.E., Casper, H.H., Odde, K.G., 1995. Use of epidemiologic principles to identify risk factors associated with the development of diarrhea in calves in five beef herds. J. Am. Vet. Med. Assoc. 207, 1334–1338.

Coates, D.B., Miller, C.P., Hendriksen, R.E., Jones, R.J., 1997. Stability and productivity of Stylonythtes pastures in Australia. II. Animal production from Stylonythtes pastures, Trop. Grass. 31, 494–502.

Coates, D.B., Marnette, L.T., 1990. Productivity of cows and calves on native and improved pasture in sub-coastal, subtropical Queensland. Trop. Grass. 4, 46–54.

Coates, D.B., Marnette, L.T., Seifert, G.W., 1987. Reproductive performance and calf growth to weaning of Hereford and Belmont Red cattle in subtropical, subcoastal Queensland. Aust. J. Exp. Agric. 27, 1–10.

Committee on Bovine Reproductive Nomenclature, 1972. Recommendations for standardizing bovine reproductive terms. Cornell Vet. 62, 217–237.

Cruz, V., Koger, M., Warnick, C., 1978. Heritability estimates of reproductive efficiency in Brahman beef cattle. In: Proc. 4th World Conf. Anim. Prod. Buenos Aires, p. 605.

Cumming, I.A., Mole, B.J., Obst, J., Blockey, M.A. deB, Winfield, C.G., 1995. Use of epidemiologic principles to identify risk factors asso- ciated with the development of diarrhea in calves in five beef herds. J. Am. Vet. Med. Assoc. 207, 1334–1338.

Cusack, P.M.V., Perry, V., 1995. The effect of feeding whole cottonseed on reproductive efficiency of PAF with fertility. Reprod. Fertil. Dev. 16 (2), 265.

Dearborn, D.D., Koch, R.M., Cundiff, L.V., Gregory, K.E., Dickerson, G.E., 1995. The effect of feeding whole cottonseed on reproduction and embryonic survival during repeated pregnancies in Dam line ewes. In: Proc. 10th Int. Cong. Anim. Reprod. AI, vol. 2, p. 74.

Denniston, R.O., 1973. An analysis of reproduction traits in beef cattle. J. Anim. Sci. 39, 36, 1032–1040.

Denniston, R.O., 1973. An analysis of reproduction traits in beef cattle. J. Anim. Sci. 36, 1032–1040.

Denniston, R.O., 1973. An analysis of reproduction traits in beef cattle. J. Anim. Sci. 36, 1032–1040.

Denniston, R.O., 1973. An analysis of reproduction traits in beef cattle. J. Anim. Sci. 36, 1032–1040.

Denniston, R.O., 1973. An analysis of reproduction traits in beef cattle. J. Anim. Sci. 36, 1032–1040.

Denniston, R.O., 1973. An analysis of reproduction traits in beef cattle. J. Anim. Sci. 36, 1032–1040.

Denniston, R.O., 1973. An analysis of reproduction traits in beef cattle. J. Anim. Sci. 36, 1032–1040.

Denniston, R.O., 1973. An analysis of reproduction traits in beef cattle. J. Anim. Sci. 36, 1032–1040.

Denniston, R.O., 1973. An analysis of reproduction traits in beef cattle. J. Anim. Sci. 36, 1032–1040.
Holroyd, R.G., Smith, P.C., Thompson, P.J.M., Toolem, M.A., 1988c. Reproductive performance of 50% Bos indicus cattle grazing the Mitchell grasslands of north Queensland. Proc. Aust. Range. Soc. 5, 49–53.

Holroyd, R.G., O'Rourke, P.K., Clarke, M.R., Loxton, I.D., 1981. Influence of pasture type and supplement on fertility and liveweight of cows, and pregnancy growth in the dry tropics of northern Queensland. Aust. J. Exp. Agric. Anim. Husb. 23, 4–13.

Hoogestijn, R., Rodríguez, R., Verde, O., Plasse, D., 1983. Porcentaje de preñez en cebú comercial en el Estado Apure. III Cong. Venezolano Zoot. F32.

Humbolt, P., Denis, J.B., 1986. Sire effects on cow fertility and late embryonic mortality in the Montbéliard breed. Livestock Prod. Sci. 14, 139–148.

Inskip, E.K., 2002. Factors that affect embryonic survival in the cow: application of technology to improve calf crop. In: Fields, M.J., Sand, R.S., Velich, J.Y. (Eds.), Factors Affecting Calf Crop: Biotechnology of Reproduction. CRC Press, Boca Raton, FL, pp. 255–279.

Johnston, D.J., Barwick, S.A., Corbet, N.J., Fordyce, G., Holroyd, R.G., Williams, P.J., Burrow, H.M., 2009. Genetics of heifer puberty in two beef genotypes in northern Australia and associations with heifer and steer production traits. Anim. Prod. Sci. 49, 399–412.

Kallela, K., Etta, E., 1984. The oestrogenic Fusarium toxin (zearalenone) in hay as a cause of early abortions in the cow. Nord. Veterinarmed. 36, 305–309.

Kidder, H.E., Black, W.G., Wiltbank, J.N., Ulberg, L.C., Casida, L.E., 1954. Fertilisation rates and embryonic death rates in cows bred to bulls of different levels of fertility. J. Dairy Sci. 37, 691–697.

King, W.A., 1990. Chromosomal aberrations in bovine and pregnancy failure in domestic animals. McFeely, R.A. (Ed.), Advances in Veterinary Science and Comparative Medicine, vol. 34. Academic Press Inc., Harcourt Brace Jovanovich, Publishers, pp. 229–250.

Kirby, G.W.M., 1984. Genotype evaluation of bovid production in the monsoon zone of the Northern Territory. In: Copland, J.W. (Ed.), Proceedings of Large Ruminants for the Tropics. Australian Centre for International Agricultural Research (ACIAR) Proceedings Series No. 5. ACIAR, Canberra, pp. 138–144.

Kirkland, P.D., 1993. Bovine ephemeral fever and related arboviruses. In: Proc. 1st Int. Symp. ACIAR, vol. 44, Beijing, pp. 33–37.

Kirkland, P.D., Corby, R.D., 1986. The economic impact of Akabane virus and the cost effectiveness of vaccination in New South Wales. In: St. George, T.D., Kay, B.H., Blok, J. (Eds.), Arbovirus Research in Australia, Proc. 4th Symp. Queensland Institute of Medical Research, Brisbane, pp. 29–252.

Kirkland, P.D., Corby, R.D., 1984. The epidemiology and control of Akabane diseases. In: Della-Porta, A.J. (Ed.), Veterinary Viral Diseases: Their Significance in Southeast Asia and the Western Pacific. Academic, Sydney.

Kirkland, P.D., MacKintosh, S.G., April 2006. Ruminant pestivirus infections. part 1. Diagnostic Overview Aust. New Zealand Standard Diagn. Procedures, 1-30.

Kodituwakku, S.N., Harbour, D.A., 1990. Persistent excretion of rotavirus by pregnant cows. Vet. Rec. 126, 547–549.

Ladds, P.W., Dennett, D.P., Glazebrook, J.S., 1973. A survey of the genitalia of bulls in northern Australia. Aust. Vet. J. 49, 335–340.

Land, R.B., 1973. The expression of female sex-limited characters in the bovine. Nature 241, 208.

Lin, Y.C., Coskun, S., Sanbuissaho, A., 1994. Effects of goospyol on in vitro bovine oocyte maturation and steroidogenesis in bovine granulosa cells. Theriogenology 41 (8), 1601–1611.

Linares, T., Rodriguez, R., 1983. Mortalidad prenatal en vacas cebú después de la temporada de servicios. III Cong. Venezolano Zoot. F33 (Abstract).

L'Haridon, R., Charpigny, G., Charlier, M., Chaouat, G., 1997. Recent developments and potentialities for reducing embryo mortality in ruminants: the role of IFN-γ and other cytokines in early pregnancy. Reprod. Fertil. Dev. 9, 355–380.

McCoyan, N., Roberts, K.M., 1994. A role for cytokines in early pregnancy. Indian J. Physiol. Pharmac. 38, 153–162.

Maurer, R.R., Chenaust, J.R., 1983. Fertilization failure and embryonic mortality in parous and nonparous beef cattle. J. Anim. Sci. 36, 1186–1189.

McCool, C.J., Townsend, M.P., Wolfe, S.G., Simpson, M.A., Olim, T.C., Johnson, G.A., Cline, B.J., 1988. Prevalence of bovine venereal disease in the Victoria River District of the Northern Territory: likely economic effects and practicable control measures. Aust. Vet. J. 65, 153–156.

McCossker, T.H., Winks, L., 1994. Phosphorus Nutrition of Beef Cattle in Northern Australia. Queensland Department of Primary Industries, Brisbane.

McCossker, T.H., O'Rourke, P.K., Eggington, A.R., 1991. Effects of providing supplements during the wet season on beef production in the Darwin district of the Northern Territory. Rangeland J. 13, 3–13.

McCosker, T.H., Eggington, A.R., 1986. Beef cattle production and herd dynamics in the monsoon tallgrass region of north Australia—case studies of several management and nutritional regimes. In: Technical Bulletin No. 93. Northern Territory Department of Primary Production, Darwin.

McDonough, S.P., Stull, C.L., O'Sullivan, B.L., 1994. Enteric pathogens in intensively reared veal calves. Am. J. Vet. Res. 55 (11), 1516–1520.

McGowan, M.R., 2003. The impact of Leptospirosis on the reproductive performance of beef heifers. In: Proc. Aust. Assoc. of Cattle Vet. Cairns Conference, 20–30 May, 2003, Cairns, pp. 94–99.

McGowan, M.R., Kirkland, P.D., 1995. Early reproductive loss due to bovine pestivirus infection. Br. Vet. J. 151, 263–270.

Mckenzie, R.A., 2002. Toxicology for Australian Veterinarians. Department of Primary Industries & Fisheries, Queensland.

Mihm, M., Bagusi, A., Boland, M., Roche, J.F., 1994. Association between the duration of dominance of the ovulatory follicle and pregnancy rate in beef heifers. J. Reprod. Fertil. 102, 123–130.

Miller, D.J., Winer, M.A., Ax, R.L., 1990. Heparin-binding proteins from seminal plasma bind to bovine spermatozoa and modulate capacitation by heparin. Biol. Reprod. 42 (5–6), 899–915.

Mills, C.F., Delgarno, A.C., 1972. Copper and zinc status of ewes and lambs receiving increased dietary concentrations of cadmium. Nature 239, 171–173.

Mitchell, P.J., Hooper, P.T., Colyer, D.N., 1981. Heat stress and diarrhoea in neonatal calves. Aust. Vet. J. 57, 397–398.

Moran C., Quirke, J.F., Roche, J.F., 1989. Puberty in heifers: a review. Anim. Reprod. Sci. 18, 167–182.

Murphy, M.G., Enright, W.J., Crowe, M.A., McConnell, K., Spencer, L.J., Boland, M.P., Roche, J.F., 1991. Effect of dietary intake on pattern of growth of different follicles during the oestrous cycle in beef heifers. J. Reprod. Fertil. 92, 333–338.

Nass, S.J., Miller, D.J., Winer, M.A., Ax, R.L., 1990. Male accessory sex glands produce heparin-binding proteins that bind to caudal epididymal spermatozoa and are testosterone dependent. Mol. Reprod. Dev. 25, 237–246.

Niswender, G.D., Juengel, J.L., Silva, P.J., Rollyso, M.K., McIntosh, E.W., 2000. Mechanisms controlling the function and life span of the corpus luteum. Physiol. Rev. 80, 1–29.

Norman, S., 2003. The management of dystocia in beef heifers. In: Proc. Aust. Assoc. of Cattle Vet. Cairns Conference, 26-30 May, 2003, Cairns, pp. 49–99.

O'Rourke, P.K., Fordyce, C., Holroyd, R.C., Sullivan, R.M., 1995. Mortality, wastage, and lifetime productivity of Bos indicus cows under extensive grazing in northern Australia 3.Comparison of culling strategies. Aust. J. Exp. Agric. 35, 307–316.

Owens, G.D., 1990. Mycotoxins and livestock: what role do fungal toxins play in illness and production losses? Vet. Med. 85, 89–94.
Wiebold, J.L., Stanfield, P.H., Becker, W.C., Hillers, J.K., 1986. The effect of restraint stress in early pregnancy in mice. J. Reprod. Fertil. 78, 185–192.

Wijeratne, W.V.S., 1973. A population study of apparent embryonic mortality in cattle with special references to genetic factors. Anim. Prod. 22, 275–278.

Windsor, P.A., Kirkland, P.D., Miller, C.M.D., Quinn, H.E., Ellis, J.T., 2002. The role of Neospora caninum in bovine abortion complex. In: Proc. Aust. Assoc. Cattle Vet. Adelaide Conference, 'Embracing Change through Technology', Adelaide Convention Centre, Adelaide, 6–10 May, pp. 37–39.

Winks, L., 1984. Cattle growth in the dry tropics of Australia. Aust. Meat Res. Comm. Rev. 45, 1–43.

Winter, W.H., McCosker, T.H., Prachett, D., Austin, J.D.A., 1985. Intensification of beef production. In: Muchow, R.C. (Ed.), Agro-research for the semi-arid tropics. University of Queensland Press, Brisbane, pp. 395–418.

Wolf, E., Arnold, G.J., Bauersachs, S., Beier, H.M., Blum, H., Einspanier, R., Fröhlich, T., Herrler, A., Hiendleder, S., Kölle, S., Pelle, K., Reichenbach, H.-D., Stojkovic, M., Wenigerkind, H., Sinowitz, F., 2003. Embryo-maternal communication in bovine—strategies for Deciphering a complex cross-talk. Reprod. Domest. Anim. 38, 276–289.

Yule, A., Skirrow, S., BonDurant, R.H., 1989. Bovine trichomoniasis. Parasit. Today 5, 373–377.

Zavy, M.T., 1994. Embryo mortality in cattle. In: Zavy, M.T., Geisert, R.D. (Eds.), Embryonic Mortality in Domestic Species. CRC Press, Boca Raton, Florida, USA, pp. 99–140.

Zeron, Y., Ocheretny, A., Kedar, O., Borochov, A., Sklan, D., Arav, A., 2001. Seasonal changes in bovine fertility: relation to developmental competence of oocytes, membrane properties and fatty acid composition of follicles. Reproduction 121, 447–545.