Jembrana Disease and the Bovine Lentiviruses

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Marked enlargement of the prescapular lymph node. Enlargement of the subcutaneous lymph nodes is a consistent clinical feature of Jembrana disease in Bali cattle.

Lymph node. Parafollicular reaction. Large lymphoid and reticular (dendritic) cells.
Section of lung from an animal with acute Jembrana disease, showing leucostasis (accumulation of mononuclear cells in the lumen of small and medium sized pulmonary blood vessels), a consistent pathological lesion in Bali cattle with Jembrana disease.
Section of lymph node, showing follicular atrophy and marked parafollicular hypertrophy, consistently present in Bali cattle during the acute clinical phase of Jembrana disease.

Section of kidney from an animal with acute Jembrana disease showing proliferative lymphoid infiltrate that is consistently detected in affected cattle.
Section of liver from an animal with acute Jembrana disease showing the proliferative lymphoid infiltrate that is consistently detected in affected cattle.

Enlargement of the spleen, often 4-6 times normal size, a consistent pathological change in cattle with Jembrana disease. A spleen from an unaffected animal has been included for comparison.
Welcome and opening address by the Director General of Livestock Services

Distinguished participants,
Ladies and Gentlemen.

First of all, let us give thanks that we could meet together in this place to attend the opening of the Workshop on Jembrana Disease in Indonesia.

On behalf of the Indonesian Government, allow me to welcome you all to Bali, Indonesia, and thank you for attending this workshop. It is my great pleasure to meet so many outstanding experts in veterinary sciences, especially those who work with retroviruses. I am sure that in the next four days each of you will share many experiences in this very important workshop.

The Government of Indonesia pays attention to the development of Bali Cattle, especially for the following reason:

Bali cattle, known as *Bos Sondaicus*, is a domesticated Benteng (*Bibos banteng*) considered as an Indonesian original breed of cattle. Nowadays, Bali cattle are distributed throughout Indonesia from Aceh to Irian Jaya, and they have proved to have high adaptability to various environments and can act as pioneer animals in new areas.

Bali cattle have good production and reproduction performances compared to other Indonesian cattle breeds, but they are most susceptible to certain diseases, i.e., Jembrana Diseases, Malignant Catarrhal Fever (MCF) and Bali Ziekte (BZ).

For these reasons, the GOI conducted two important projects. The first related to improvement of the genetic quality of Bali cattle, known as the Bali Cattle Breeding Project, which was started in 1978 and assisted by the New Zealand Government. The second related to the investigation of Jembrana disease by the Bali Cattle Disease Investigation Unit (BCDIU), which was funded by IFAD under the Smallholder Cattle Development Project (Loan No. 396 — ID).

This project actually finished last year, but in the interests of developing Jembrana vaccine quality and the method of Jembrana disease eradication, it will be continued this year through IFAD Project Phase III (1996/1997—1999/2000).

Since the First International Seminar on Jembrana Disease held in Bali, 22–24 September 1975, magnificent progress has been achieved. Thanks to the hard work of the BCDIU Team, and with the collaboration of scientists from all over the world, the most crucial question about the causal agent of Jembrana disease has been answered. It is a retrovirus.

In this workshop, all participants will be informed of the considerable progress which has been made by the BCDIU Team concerning their efforts to control Jembrana disease, work to establish the future research priorities, and efforts to promote the control of Jembrana disease in Indonesia. Your contributions to this workshop will be beneficial, not only to Indonesia, but also to the various countries where various species are affected by retroviruses.

On behalf of the Government of Indonesia, I would like to extend my gratitude to the Australian Centre for International Agricultural Research (ACIAR) for its kind sponsorship of this workshop. I would like also to thank especially Dr John Copland, Animal Science Coordinator of ACIAR and Dr Graham Wilcox, Associate Professor (Virology), School of Veterinary Studies, Murdoch University, for their efforts to make this workshop happen.

Although the time available for the preparation of this workshop was very short, I think the preparation has been very thorough thanks to the hard work of the Organising Committee. I appreciate that very much.

Finally, with your permission, I declare the Workshop on Jembrana Disease in Indonesia officially open. Thank you for your kind attention and I wish you luck and success in your deliberations.

*Director General of Livestock Services*
*Ir. Erwin Soetirto*
The Occurrence and History of Jembrana Disease in Indonesia

S. Soeharsono and I.G.N. Teken Temadja

Abstract

An outbreak of a new disease was reported in the Jembrana district of Bali province, Indonesia, in 1964. At first, it was reported to affect Bali cattle (Bos javanicus syn Bos sondaicus) and buffaloes (Bubalis bubalis), but only Bali cattle were reported in the later outbreaks in Bali and elsewhere in Indonesia. The disease was diagnosed firstly as a rinderpest-like disease and subsequently named Jembrana disease (JD).

Due to strict regulation of the exportation of Bali cattle from Bali island to other islands in Indonesia, the disease was confined to Bali island for more than 12 years. The first outbreaks of JD outside Bali island were reported in Lampung Tengah district, Sumatra island, in 1976. Illegal introduction of JD Bali cattle from Bali island was the suspected cause of the outbreak. From Lampung, JD spread northwards to West Sumatra in 1992, Bengkulu in 1994, and South Sumatra in 1995. The disease also occurred in the Banyuwangi district of Java island in 1978. More recently the disease was reported in the South Kalimantan and Central Kalimantan Tengah provinces of Kalimantan island.

Introduction

BALI island, located in the eastern part of Indonesia, consists of eight districts, i.e. (in alphabetical order) Badung, Bangli, Buleleng, Gianyar, Jembrana, Karangasem, Klungkung and Tabanan. Its main product is agricultural, and in general, the soil is very fertile.

Bali cattle are indigenous to Indonesia (Payne and Rollinson 1973) and were domesticated directly from wild banteng (Bos javanicus syn Bos sondaicus syn Bibos banteng) (Porter 1991). The economic importance of Bali cattle in Indonesia is presented elsewhere (these Proceedings). Individual farmers in Bali on average own only 3.3 head of Bali cattle (Soeharsono et al. 1983). In Bali and most other parts of Indonesia, Bali cattle are used as draught animals in the rice fields, young animals may be sold if the farmer needs cash for various purposes, and older animals are sold for beef production.

Before Jembrana disease (JD) occurred, the trade of Bali cattle from Bali to other Indonesian islands, or even for export to Hong Kong and Singapore for slaughter, was unrestricted, but the inter-island trade in Bali cattle from areas where JD is endemic is now banned. An exception is that Bali cattle from Bali may be trucked to Jakarta for slaughter.

This paper describes the occurrence and history of JD in Indonesia, based on available papers and reports, and the field observations and experiences of veterinarians.

History of the Outbreak

Origin

Information concerning the origin of the outbreak is scarce. The only written information before the outbreak concerned the presence of a foreign vessel in Buleleng harbour, about 100 km from the village where the disease was first reported. The ship was reported to contain a number of cattle, but there was no further information about the origin of the ship nor about deaths among the animals. No reports of a similar disease outbreak were received from any neighbouring countries.

Two other events preceded the outbreak of JD. The first was a mass vaccination against foot and
mouth disease using an inactivated vaccine produced in Surabaya in 1963. The vaccine had caused severe post-vaccinal reactions. As a number of Bali cattle collapsed and farmers were upset with the implementation of the vaccination program, it was temporarily stopped. A decision was made to import an inactivated monovalent type-O standard vaccine prepared in cell culture in England. The mass vaccination was continued using the imported vaccine. The question arises: did the inactivated vaccine contain a virus which was non-pathogenic in Bos taurus and Bos indicus, but pathogenic in Bos sondaicus? Jembrana disease appears to produce a marked clinical disease in Bali cattle only, and a subclinical disease in other species of cattle (Soeharsono et al. 1990a, b).

The second event was the eruption of Mount Agung volcano in 1964 when more than 1000 people died. Dust from the eruption covered the grass and leaves of plants used for cattle consumption, so farmers had to wash the grass and plants before feeding the cattle. This condition lasted for weeks and might have caused severe stress in Bali cattle, but it is unknown if there was an indirect relation between the JD outbreak and the eruption of Mount Agung.

An additional point worthy of note is that West Bali National Park is situated in the western part of the Jembrana district. Wildlife, including wild banteng and deer, are present in this protected park and indirect contact between deer and Bali cattle grazing nearby is likely to occur. There have not been any studies to determine the possible presence of the infectious agent associated with JD in wildlife.

**The first outbreak in Bali**

Sangkaragung, a village in the Jembrana district of Bali province, was the first place where an outbreak of a previously unrecognised disease was reported in December 1964 (Pranoto and Pudjiastono 1967). The disease was reported to affect Bali cattle (Bos javanicus syn. Bos sondaicus) and buffaloes (Bubalus bubalis) (Adiwinata 1968). The common disease affecting Bali cattle and buffalo in this region is haemorrhagic septicaemia (HS); in order to control the disease HS specific antisera and vaccine were used by the local veterinary service. The disease did not seem to cease after the application of HS antisera and vaccine. By August 1965, the disease had spread to all eight districts of the island and high mortality figures were reported (Pranoto and Pudjiastono 1967).

In April 1965, the first specimens for laboratory diagnosis were submitted to the Animal Disease Research Institute in Bogor (West Java); no potentially pathogenic bacteria or blood parasites were detected.

In June 1965, a team from the Institute for Animal Virus Disease (Lembaga Virologi Kehewanan, LVK), Surabaya (East Java), was sent to carry out a field investigation and to collect specimens for laboratory examination. After consideration of epidemiological, clinical and pathological findings, this group suspected the disease was rinderpest or a rinderpest-like disease (Adiwinata 1968).

In order to control the disease, the Directorate of Animal Health of the Directorate General of Livestock Services conducted a mass vaccination program using lapinised-avianised attenuated rinderpest vaccine imported from Japan. Coincidentally or not, for a couple of years after the mass rinderpest vaccination program, the disease seemed to disappear. A second approach was to ban the export of cattle from Bali to other islands within the Indonesian archipelago, with the exception that cattle were permitted to be exported from Bali to Jakarta for slaughter.

A second outbreak of JD occurred in 1972, in the Tabanan district (adjacent to the Jembrana district) of Bali island. The disease that occurred there was similar to that reported during the initial 1964 outbreak (Hardjosworo and Budiarso 1973). A third outbreak of JD occurred in 1981 in the Karangasem district in the Eastern part of Bali island (Putra et al. 1983). There were no reports of the occurrence of Jembrana disease between these three outbreaks but the disease is now endemic in Bali cattle on Bali island.

**Outbreaks of JD on other Indonesian islands**

In June 1976, a disease similar to JD was reported in Bali cattle in Lampung, South Sumatra (Soeharsono and Darmadi 1976; Ramachandran 1981). Severe clinical and pathological changes with high morbidity and mortality rates similar to those in the first outbreak in Bali were reported. Bali cattle, introduced by a Balinese transmigrant, were suspected of being the source of the outbreak.

In November 1978, JD was diagnosed in the district of Banyuwangi in East Java province, on the route that Bali cattle may be legally trucked from Bali to Jakarta. These cattle were a possible source of the outbreak. There is only a short distance between Bali and Banyuwangi, and the smuggling of live Bali cattle from Bali to Java island by small boats was a common practice at that time and difficult to prevent. Some JD cases were confused with malignant catarrhal fever, also endemic to this region (Tranggono 1988).

In April 1992, an outbreak of JD was reported in Sawahlunto–Sijunjung district of West Sumatra
province (Tembok 1992). The diagnosis of the disease was confirmed by histopathological examinations and serological tests (ELISA) for antibody to Jembrana disease virus at BCDIU, Denpasar. It is possible that JD carrier Bali cattle from Lampung were introduced into this area through the Solok cattle market in West Sumatra. Control of the movement of animals in Sumatra island is very difficult and therefore the spread of JD to other provinces is likely to occur. In 1995, JD was reported in Bengkulu province of Sumatra island.

In 1993, by histopathological examination of tissues from Bali cattle, JD was also diagnosed in South Kalimantan province (Kalianda 1993, pers. comm.). The morbidity and mortality rates were low, possibly because the population of Bali cattle was low and mixed with other breeds. Further confirmation of the disease in this area was made by histopathological examination and serological tests conducted at the BCDIU. The cause of this outbreak is unknown but importation of cattle, especially Ongole cattle, from East Java to South Kalimantan has long been practiced (Kalianda 1993, pers. comm.) and these cattle, when infected, develop viraemia. Bali cattle from an infected area in East Java (Banyuwangi) might also have been included in the shipment and caused the outbreak.

Conclusion

The source of the infectious agent responsible for the first cases of the disease in Bali cattle in the Jembrana district of Bali island has never been determined. The restriction of cattle movement has proved effective to control the spread of the disease. Introduction of live carrier animals from a JD-infected area to uninfected areas has contributed to the spread of JD.

References


Early Observations and Research on Jembrana Disease in Bali and Other Indonesian Islands

S. Ramachandran¹

Abstract

Jembrana disease, an enigmatic persistent virus disease of banteng (Bos sondaicus), is restricted to some Indonesian islands. Discovered in Jembrana, a district in West Bali, in 1964, it has several perplexing epidemiological and clinicopathological features.

The causal agent, a medium-sized (100-200 nm) virus, induces clinical disease only in banteng. Infections do occur naturally in the buffalo and banteng non-descript cattle crosses and can also be artificially induced in sheep and goats. Such infections result in virus persistence in blood and spleen for short periods.

Clinical disease in banteng is heralded by tell-tale features such as high fever, lymphopenia, thrombocytopenia, buccal erosions and haemorrhages in the skin (‘blood sweating’) and soft tissues, lymphadenopathy and diarrhoea. Clinical recovery is associated with a marked lymphocytosis. It is, however, transient as relapses are common.

Jembrana disease virus is thermolabile, pH sensitive (pH 3.0 and 11.0), ether and deoxycholate resistant. It has no immunising antigens and convalescent sera do not contain virus-neutralising antibodies. There is a significant hypogammaglobulinaemia which is in fact an IgG deficiency.

The disease is non-contagious. There is preliminary evidence suggesting that it spreads through Boophilus microplus ticks in which there is transovarian transmission of the agent.

In December 1964, the Desa Sankaragung in the Kabupaten Jembrana, Bali, witnessed a colossal bovine disaster. A virgin epizootic had erupted on an unprecedented scale and decimated almost the entire banteng (Bos sondaicus) population in a few days. More than three decades have passed since the recognition of this new murrain called Jembrana disease (JD), named after the locality. Although several salient features of this malady have been brought to light and doubts regarding its viral aetiology laid at rest, several questions still remain unanswered, particularly its mode of spread and the natural history of the causal agent.

Historical Aspects

The disease in Bali


In the virgin epizootic of 1964, an estimated 60% of the Bali cattle and buffalo populations was attacked. The rate of spread was cataclysmic. Total mortalities were 98.9% by December 1964. By August 1965, all the eight kabupatens (districts) were affected. Mean case fatalities in 1965, 1966 and 1967 were 71.6 ± 6.0, 31.3 ± 12.0 and 38.6 ± 10.0 respectively (Sonoda 1969).

The mistaken diagnosis that JD was an outbreak of a virulent form of rinderpest resulted in the mass anti-rinderpest vaccination by Sonoda. This was despite Ishitani’s (1968) finding that histopathological changes in JD were distinctly different from those documented for rinderpest. Also, in Prof. Tanjung Adiwinata’s transmission studies in Surabaya (Adiwinata, 1967), the causal agent of JD was found to be non-infective to Zebu cattle and pigs — species that are highly susceptible to rinderpest. The specificity of the JD agent to banteng was a

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novel finding and was Adiwinata’s singular contribution to research on JD epidemiology.

The next outbreak of JD was in 1971 in Kabupaten Tabanan, and was called ‘Tabanan Disease’. Although acute and peracute cases were observed on occasions, the disease was relatively mild with the total mortality in 1971–72 at 22.0%. Buffaloes were spared. A rickettsial aetiology was proposed by Budiarso and Hardjosworo (1976) on the basis of laboratory studies in Bogor, but unfortunately, these were not followed by experimental reproduction of the disease in banteng.

An FAO team comprising M. Lobry, H.P. Harding, E. Teuscher, H. Dennig and S. Ramachandran pursued investigations on the rickettsial hypothesis, but, by the end of 1975, reached the conclusion that the causal agent was a virus. This was based on several findings.

1) Prolonged treatment of infective tissue suspensions with bactericidal drugs did not alter infectivity to banteng, the only species in which the clinical disease could be readily reproduced.

2) Infectivity of suspensions was unaffected after filtration through a 200 nm membrane filter.

3) There were no significant differences in clinical parameters, mortality rates and severity of pathological changes in groups of banteng experimentally infected with filtered and unfiltered suspensions.

4) The suspensions were not infective to guinea pigs, rabbits or mice.

5) Sera of banteng recovering from natural or experimentally-induced JD did not develop anti-ehrlichial antibodies readily detectable in Weil–Felix and CF tests (CFT). Infective tissue suspensions also failed to react in the CFT with reference to sera containing antibodies to *Cowdria ruminantium*, *Coxiella burnetii* and *Ehrlichia phagocytophila*.

6) In spite of a careful research, rickettsiae were not detected in electron micrographs (EMs) of peripheral blood and tissue lymphocytes and monocytes.

Moreover, in the detailed histopathological studies of Teuscher et al. (1981) on 47 natural cases of JD, there was substantial evidence that tissue changes in JD were akin to those commonly observed in virus diseases. In particular, the microscopic picture was reminiscent of that in bovine malignant catarrh except that there was no encephalitis and that the vasculitis was not of the necrotising type. There was a significant endotheIiosis in several tissues and organs. A similar opinion was offered by Dr R.J. Brown of NAMRU-2, Jakarta Detachment (R.J. Brown 1975, pers. comm.).

Rama Dewa disease

This was first recognised in the Balinese trans-migration settlements in Lampung, south Sumatra in May 1976. The disease bore all the hallmarks of a virulent form of JD. Although there was free mixing of banteng with large numbers of Ongole cattle and other non-descript breeds, the disease was confined to the former. Attack rates in nine desa with a total population of 2596 banteng ranged from 2.4% to 71.6% (mean 36.5%). Case fatality rates were 12.5% to 72.0% (mean 50.6%).

In a controlled experimental study, rickettsial drugs such as chloramphenicol, terramycin and...
oxytetracycline given in daily doses alone or in combinations from the first day of inoculation failed to prevent deaths in 21 banteng. All developed clinical JD after a mean incubation period of 3.45 ± 0.2 days. Mortality was nearly 100% (two were killed in extremis). The mean day to death was 9.5 ± 1.2 post inoculation (Table 1). Analysis of clinical parameters revealed no significant differences in responses between treated and untreated animals.

**Banyuwangi Disease**

Banyuwangi, in east Java was the scene of several outbreaks of a JD-like syndrome from November 1978. Fourteen of 18 kacamats were affected. In the next six months, there were 497 deaths (31.2%) in a mixed population of banteng, Rambon cattle (cross between non-descript and banteng) and buffaloes. Tissue suspensions from one suspected case each in banteng and buffalo were inoculated into batches of two banteng at Denpasar. Only the buffalo suspensions caused a febrile response and equivocal clinical signs. Blood drawn during pyrexia from the reacting banteng was sub-inoculated into two other banteng, one being a known 'carrier' and the other susceptible. The latter developed pyrexia and leucopenia after an incubation period of four days. There was also regional adenopathy. This was the first isolation of JD agent from a buffalo. There was also histopathological evidence of JD infection in 10 of 17 'clinical' cases, two from banteng and four each from Rambon cattle and buffalo.

**Research**

Results of several laboratory studies conducted at the BPPH, Denpasar, during 1975–80 were as follows.

**Experimental transmission**

The causal agent of JD was readily transmitted to banteng, buffaloes, sheep and goats but clinical signs were manifest only in banteng. Pigs, guinea pigs, rabbits, mice, chicken and duck embryos were not infected. Although inoculated sheep and goats did not show clinical signs, they had viraemia which persisted for up to six weeks. Virus also persisted in the spleen for up to three months.

One of two experimentally-inoculated buffaloes developed fever and leucopenia on the fourth day. Fever lasted for five days. There were no other clinical abnormalities. When necropsied two months later, spleen was infective to banteng. Viraemia was demonstrable at earlier periods.

**Cell cultures**

The agent was not cultivable in primary banteng and pig kidney cells or chicken embryo fibroblasts. Cultured lymph node cells developed a CPE on the fifth day. There were foci of syncytia. Some individual cells showed eosinophilic cytoplasmic inclusions. The fifth day culture suspension induced an equivocal clinical response in a banteng. It had fever for two days but no leucopenia. Sub-inoculation of its blood into another banteng proved to be non-infective. Cultures of later ages were not infective.

**Persistence and relapses**

Recovery from clinical JD did not entail sterile immunity. Viraemia was demonstrable by inoculation into banteng, for several months. The longest period of virus recovery was 488 days from a clinically normal 'carrier'.

Relapses were common. In this regard, JD behaved like a rickettsial infection. Thirty-two of 35 carrier banteng (92%) relapsed. Nine relapsed twice, 4 thrice, 2 four times and 1 on six occasions. Sixteen of these cases died. During relapse infection, there was low-grade febrile response for two or three days and leucopenia of variable intensity. However, in many cases there was no leucopenia.

**Tick transmission**

In two separate trials in 1975–76, there was compelling circumstantial evidence of transovarian transmission of JD agent in Boophilus microplus ticks. Banteng used in these studies were obtained from Lombok, regarded as a JD-free island, and maintained in tick-proof premises at BPPH, Denpasar.

In the first study, female ticks that had engorged on the bodies of naturally-infected banteng were allowed to oviposit in test tubes. The larvae that emerged were placed on the bodies of three donor banteng and allowed to mature into adults in 21 days. Twelve, 21 and 25 days later, the three donors developed typical signs of JD and died at different intervals.

In the second trial, which was only a partial success, juvenile ticks that had engorged on the bodies of naturally-infected banteng were allowed to oviposit in test tubes. The larvae that emerged were placed on the bodies of three donor banteng and allowed to mature into adults in 21 days. Twelve, 21 and 25 days later, the three donors developed typical signs of JD and died at different intervals.

In the second trial, which was only a partial success, juvenile ticks were harvested on the 19th day from the bodies of three donor banteng and allowed to mature into adults in 21 days. Twelve, 21 and 25 days later, the three donors developed typical signs of JD and died at different intervals.
proving that it had been infected by larvae. The other two banteng did not develop the disease.

Haematology
This was a fruitful area of research leading to interesting findings. Absolute lymphopenia and marked thrombocytopenia were consistently observed in spontaneous and experimentally-induced JD. In the febrile phase, mature and immature lymphocytes underwent a series of degenerative changes leading to lysis. In the recovery phase, blood contained a significant proportion of abnormal lymphocytes. The abnormalities were related to their size, morphology and tinctorial properties. Large lymphocytes developed alternative foci of protoplasmic condensation, the so-called blebs and thinning of plasma membrane. The cytosol was vacuolated and weakly basophilic or amphoteric. Karyomegaly, swelling of nucleoli, eccentricity of nuclei, occurrence of binucleate cells and fusion into small syncytia were other abnormalities.

In the post-febrile lymphocytosis phase, there was a mixed population of medium-sized lymphocytes often binucleate, lymphocytes with mitotic figures and plasma cells. Medium-sized to large lymphocytes and monocytes had particulate inclusions. In electron micrographs, these were recognised as platelets in different stages of degeneration.

Lymphocytes and monocytes had ultrastructural changes. Mitochondria were swollen, the endoplasmic reticulum was disrupted and there was an abnormal increase in polyribosomes. In light microscopy, it was often difficult to differentiate large lymphocytes from monocytes. In the EM, the latter had more granules in the cytosol, more endoplasmic reticulum and a higher cytoplasmic–nuclear ratio.

Immunity
Jembrana disease is a persistent viral infection resulting in a profound immunological disorder. There was no evidence of humoral immunity. Subsidence of clinical disease did not constitute true recovery as relapses were frequent. Formalinised or B-propiolactone-treated infective tissue suspensions given alone or suspended in Freund’s adjuvant failed to protect banteng against challenge infection. Such animals did not develop neutralising antibodies.

Serology
Convalescent sera were anti-complementary. This interfered with the performance of a CF test. However, a precipitating antigen was demonstrable in the acute phase plasma. It gave a single line of precipitation in agar gels against concentrated convalescent sera. Sera required heat-inactivation (60°C for 30 minutes) to dissociate antigen-antibody complexes and ensure denaturation of free antigen. These interfered with the precipitation reaction.

Several samples of convalescent sera examined at the Naval Aerospace Medical Research Laboratory, Florida (USA) were reported to be hypogammaglobulinaemic (R.J. Brown, 1976, pers. comm.). This was attributed to a significant depression in IgG levels.

Characterisation of JD virus
The agent was filterable, the estimated particle size being 100–200 nm. It was inactivated after exposure to 55°C for 15 minutes and to extremes of pH (3.0 and 11.2). It was resistant to the action of sodium deoxycholate (1:1000), diethyl ether and a range of antibiotics. It was readily inactivated by formaldehyde and B-propiolactone. Infectivity in meat persisted up to 36 hours at 22–25°C and for 72 hours at ±4°C. The agent stored well for several months at −70°C.

Epidemiologic shifts
In the period since its discovery, JD has undergone marked changes in attributes such as virulence, rate of spread and species susceptibility. The virgin epizootic of 1964–67 was characterised by fast spread. Mass exodus of banteng might have acted as a contributory factor. However, excessive virulence might also be an associated factor.

In the early studies by the FAO team in 1974–75, rates of spread in the three village herds at Sesetan, Nagara and Panjer were moderate. The numbers of banteng with clinical disease were 22, 28 and 33 respectively and the periods taken for development of the disease in these cases were 5, 7 and 8 weeks respectively.

Whereas the 1964–67 outbreaks had affected buffaloes, this species was spared in subsequent outbreaks in Bali. However, there was evidence of natural disease in buffaloes in East Java where they outnumber banteng and crossbred cattle populations. Reduced susceptibility to tick infestation may be an important factor.

In Bali, general incidence of JD has declined significantly since 1976. Reported cases in five years from 1974 were 4584, 4610, 1970, 1188 and 1739 with a mean score of 1632 for 1976–1978. Mortalities and case fatality rates (in parenthesis) were 336 (7.3%), 345 (7.5%), 203 (10.3%), 125 (10.5%) and 55 (3.2%) respectively. The disease was recognised throughout the year. There was no correlation between incidence and climatic factors such as mean rainfall, relative humidity or diurnal temperatures. In a limited survey, spraying with insecticides significantly reduced the incidence of the disease.
A critical study of clinical parameters of experimentally-induced disease revealed that the infection had become progressively milder since 1977. For instance, mean duration of fever and mean peak temperature were significantly lower in later periods than in 1975–76. Whereas most infected banteng died in less than 11 days post-infection in 1975–76, the mean day-to-death was prolonged in 1977–78 (54 ± 12) and in 1978–79 (28 ± 9). In 1976–77, seven of 31 fatalities occurred in a mean period of 11.9 ± 0.6 days, six in 24 ± 2 days and the rest in 50 ± 4 days.

References
Ishitani, R. 1968. Some critical opinions on Jembrana disease occurred in the island of Bali, Indonesia, especially from the viewpoint of pathology. Folia Veterinaria Elveka, 2, 9–29.
Clinical Changes in Bali Cattle and Other Ruminants Following Infection with Jembrana Disease Virus

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Abstract

Clinical signs of Jembrana disease in Bali cattle (Bos javanicus), induced by intravenous inoculation of Jembrana disease virus (JDV), occurred after an incubation period of between 4 and 12 days. There was a linear relationship between the dose of virus inoculated and the incubation period. Clinical signs persisted for 5–12 days, followed by recovery of most affected animals. The major clinical signs were an elevated rectal body temperature persisting for seven days (range 5–12 days), lethargy, anorexia, enlargement of the superficial lymph nodes, a mild ocular and nasal discharge, diarrhoea with blood in the faeces and pallor of the mucous membranes. Not all of these clinical changes occurred in all affected cattle. The major haematological changes included a leucopenia, lymphopenia, eosinopenia and a slight neutropenia, a mild thrombocytopenia, a normocytic normochromic anaemia, elevated blood urea concentrations and reduced total plasma protein. The mortality rate in the experimentally-infected cattle was 17%.

Other cattle types were also susceptible to JDV infection. Infection of Friesian (Bos taurus) and crossbred Bali cattle (Bos javanicus × Bos indicus) induced clinical changes and lesions consistent with those detected in Bali cattle, although they were milder and consequently would have been difficult to detect under field conditions. Ongole cattle (Bos indicus) and buffalo (Bubalus bubalis) also developed a mild febrile response after inoculation with JDV but no other overt clinical signs of the disease; no haematological investigations were conducted on these animals after they were inoculated.

Jembrana disease virus was present to high titres in the blood of Bali cattle during the febrile period. The titre of virus in blood then declined concurrent with the regression of the clinical changes. The virus persisted at low titres in the blood of recovered cattle for at least 25 months. Buffalo, Ongole cattle, Ongole × Bali cattle, and Friesian cattle also developed a persistent infection; although the virus was recovered from blood or spleen tissue of infected buffalo for at least nine months after infection, it persisted for only 3–6 months in the other species. Sheep and goats did not develop a febrile response after infection with JDV. Sheep, however, developed a viraemia that persisted for 3–6 months after infection. Some pigs inoculated with JDV developed a fluctuating febrile reaction after infection but JDV was never recovered from the inoculated animals. Seventeen of 18 Bali cattle in which Jembrana disease had been experimentally-induced up to 22 months previously did not develop clinical signs when re-challenged with JDV.

WITH the recognition that Jembrana disease was caused by a bovine lentivirus designated Jembrana disease virus (JDV) (Kertayadnya et al. 1993; Chadwick et al. 1995a,b), the disease has been recognised as an unusual lentivirus disease: an acute disease with a short incubation period (Chadwick et al. 1995b).

Under field conditions, the clinical signs of Jembrana disease are difficult to define and are frequently complicated by the occurrence of secondary infections such as bacterial pneumonia (Teuscher et al. 1981). The disease can be readily transmitted to susceptible Bali cattle by inoculation of blood or spleen tissue from infected cattle (Soeharsono et al.
1990), and the pathological changes in experimental and naturally occurring disease were reported to be very similar (Teuscher et al. 1981). The authors examined the sequential clinical and haematological and virological changes occurring in Bali cattle experimentally infected with Jembrana disease virus (JDV) in order to define the changes that do occur during the disease.

There have been no confirmed reports of clinical disease, typical of Jembrana disease in Bali cattle, in other cattle types or other animal species. Experiments were undertaken to confirm this apparent host specificity, and are described in this paper. These experiments involved the experimental infection of other cattle types and ruminant species with JDV and a detailed examination of the sequential clinical and haematological changes that occurred in these animals.

Materials and Methods

Experimental animals

Bali cattle used for experimental infection studies were approximately 18 months of age. They were obtained from Nusa Penida, a small island adjacent to Bali, where Jembrana disease had not been reported. Experience has shown that these cattle are consistently susceptible to experimental challenge with the Jembrana disease agent. On arrival, the animals were kept in screened animal houses and given free access to *Penisetum purpureum* grass and water. They were kept under observation for a minimum of seven days prior to use.

Other ruminant species and pigs used in the experiments were purchased from various areas of Bali or Java: Friesian cattle, buffalo, Indonesian sheep, goats and pigs from Bali, Ongole cattle (*Bos indicus*) from East Java, Rambon cattle (developed by crossbreeding *Bos indicus* × *Bos javanicus*) from East Java, and the Madura breed of cattle (also derived by crossbreeding *Bos indicus* and *Bos javanicus*) from Madura island. All animals were clinically normal at the time of purchase and were monitored for several days prior to use.

Jembrana disease virus

Three isolates of JDV were used: Klungkung/85, Singaraja/86 and Tabanan/87. Unless otherwise stated, the Tabanan/87 isolate was used. All isolates were obtained from naturally-infected animals exhibiting typical signs of Jembrana disease: 10 mL of heparinised blood from the affected animal was injected intravenously into susceptible Bali cattle, the recipient animals were killed two days after the development of fever, spleen tissue was aseptically collected and distributed into aliquots, stored frozen at −70 °C and used as a source of infectious agent. When required, the spleen tissue was thawed, a 10% homogenate was prepared with a mortar and pestle with medium 199 (Flow Laboratories) as a diluent, and then clarified by centrifugation at 650 g for 20 min.

Method of infection and examination of experimental animals

Bali cattle were inoculated with various dilutions of whole blood or plasma obtained from experimentally-infected Bali cattle two days after they developed a febrile reaction in excess of 39.5 °C, as previously described (Soeharsono et al. 1990). Prior to infection and for up to 28 days after infection, the animals were examined daily: clinical signs were recorded and heparinised blood samples were obtained for haematological examination.

Clinical signs were recorded on a subjective scale of from 1 to 3 (1 = mild, 2 = moderate, and 3 = marked). Any animals that became recumbent and unable to rise were killed. A complete pathological examination was conducted on any animals that died, on animals that became recumbent and unable to rise, and all animals at the conclusion of the experiment. The results of the pathological examinations will be reported separately.

Haematological examinations

Blood samples were collected with heparin as an anticoagulant. Thin blood smears were made immediately after collection and stained with Giemsa for differential leucocyte counts. Total leucocyte, erythrocyte and thrombocyte counts, packed cell volume (PCV), haemoglobin (Hb), erythrocyte sedimentation rate, mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH) and mean corpuscular haemoglobin count (MCHC), blood urea, creatinine concentrations, total plasma protein and fibrinogen were determined as described by Soesanto et al. (1990).

Titration of infectious virus in blood and plasma

To quantitate the infectious agent present in the blood of infected Bali cattle, animals were infected intravenously with 1 mL of a 10% homogenate of spleen from an animal infected with the Tabanan/87 isolate of the Jembrana disease agent. Two days after the rectal temperature of the recipient animal exceeded 39.5 °C, a heparinised blood sample was obtained and serial 10-fold dilutions of the blood were made in antibiotic-free medium 199 with 10% foetal bovine serum (FBS). One mL of each dilution was inoculated intravenously into two Bali cattle.
Control animals were inoculated with 1 mL of medium 199 with 10% FBS.

All infected animals were examined daily after infection for evidence of Jembrana disease infection. The principal signs used were an increase in rectal temperature in excess of 39.5 °C which persisted for more than two days and a concurrent leucopenia (less than 4000 leucocytes per uL). Confirmation of the clinical diagnosis was made in most cases by the detection of typical gross and microscopic pathological changes (Teuscher et al. 1981) after the animal was killed. An approximate 50% Bali cattle infectious dose (ID_{50}) was determined as the highest dilution of the inoculum producing Jembrana disease in at least one of the two inoculated animals.

Infection and detection of persistent infections in ruminants and pigs

Ongole and Friesian cattle, buffaloes, sheep, goats and pigs were infected with JDV obtained from Bali cattle with experimentally-induced Jembrana disease as described above. They were infected with either blood collected and inoculated immediately after collection, or with a 10% suspension in medium 199 of frozen and thawed spleen from Bali cattle which were killed during the febrile period. The blood used to inoculate the goats and pigs was concurrently titrated in Bali cattle as described above and the dose per inoculated animal determined as 10^8 ID_{50}. All inoculated animals were examined for clinical signs of Jembrana disease at daily intervals for 28 days post-inoculation. To detect persistent JDV infection in the inoculated animals, at intervals after infection, tissues were collected and sub-inoculated into Bali cattle which were then monitored for typical clinical signs of Jembrana disease. Tissues used were either heparinised blood samples or 10% suspensions of spleen prepared from animals which were killed.

Resistance of Bali cattle to re-infection

Eighteen Bali cattle which had previously been experimentally infected with the Klungkung/85 or Singaraja/86 isolates of the Jembrana disease agent were challenged at various intervals after infection (Table 1) with 1 mL of a 10% suspension of spleen from a Bali animal infected with the Tabanan/87 isolate. Eight control animals were also challenged with the same inoculum. The rectal temperature was determined daily for 21 days after challenge, and blood samples were obtained from any animals exhibiting a febrile reaction to confirm the presence of haematological changes typical of Jembrana disease (Soesanto et al. 1990).

<table>
<thead>
<tr>
<th>Animal</th>
<th>Strain used in initial infection</th>
<th>Time since initial infection (months)</th>
<th>Susceptibility to challenge with Tabanan/87 strain</th>
</tr>
</thead>
<tbody>
<tr>
<td>442 K1ungkung/85</td>
<td>20</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>451 K1ungkung/85</td>
<td>22</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>186 K1ungkung/85</td>
<td>22</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>477 Singaraja/86</td>
<td>7</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>484 Singaraja/86</td>
<td>9</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>485 Singaraja/86</td>
<td>6</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>491 Singaraja/86</td>
<td>4</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>501 Singaraja/86</td>
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</tr>
<tr>
<td>502 Singaraja/86</td>
<td>4</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>510 Singaraja/86</td>
<td>5</td>
<td>—</td>
<td></td>
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<tr>
<td>511 Singaraja/86</td>
<td>4</td>
<td>—</td>
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</tr>
<tr>
<td>512 Singaraja/86</td>
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<td>—</td>
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</tr>
<tr>
<td>523 Singaraja/86</td>
<td>3</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>492 Singaraja/86</td>
<td>2</td>
<td>—</td>
<td></td>
</tr>
</tbody>
</table>

Analysis of data

As Bali cattle were inoculated with varying dilutions of blood and there was a linear relationship between the dilution of blood and the incubation period before the development of clinical signs, the clinical signs and haematological data recorded at daily intervals after infection were analysed on the basis of time after the development of a febrile response. The results are expressed as a mean of the individual results in animals that developed a febrile response within 14 days after infection, and include the data from animals that died or were killed in extremis.

Results

Experimentally-induced disease in Bali cattle

A febrile response was detected in 18 animals inoculated with various dilutions of whole blood from cattle which had been experimentally infected with JDV. As shown in Figure 1, the incubation period (time from inoculation to the onset of the febrile period) varied from 4 to 12 days, and there was a linear relationship between the duration of the incubation period and the dose (dilution of the blood or plasma) inoculated into each animal. The febrile response persisted for seven days (range 3–12 days) but there was no relationship between the duration of
Figure 1. Relationship between the dilution of blood used to inoculate cattle and the incubation period before the onset of clinical signs. The blood was obtained from two experimentally-infected cattle (A and B) on the second day of the febrile reaction. In animal A, $r=0.95$ ($P<0.01$); in animal B, $r=0.8$.

Of the 18 animals that developed Jembrana disease, two died and one was found recumbent and unable to rise and was considered to be in extremis and was killed. Including the recumbent animal, the mortality was 17%. The three animals died from 9 to 20 days after the development of a febrile response; one died during a febrile period which had persisted for eight days before death, and the other two died 4 and 13 days after recovery from a febrile period of 9 and 8 days, respectively.

All animals developed a leucopenia which coincided with the febrile period and was principally due to a lymphopenia, although there was also a moderate neutropenia towards the end of the febrile period persisting for approximately seven days (Fig 2). There was almost a complete absence of eosinophils 2 to 12 days after the start of the febrile period, which then slightly increased but remained below normal until the observations were discontinued 21 days after the start of the febrile period. No change was detected in the number of monocytes or basophils.
Table 2. Severity and duration of clinical signs detected in Bali cattle after experimental infection with Jembrana disease virus.

<table>
<thead>
<tr>
<th>Clinical sign</th>
<th>Per cent of 18 cattle affected</th>
<th>Severity of clinical signs* in relationship to onset of fever</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Days after onset of fever</td>
</tr>
<tr>
<td></td>
<td></td>
<td>-2  -1  0  1  2  3  4  5  6  7  8  9  10  11  12  13  14  15  16</td>
</tr>
<tr>
<td>Anorexia</td>
<td>94</td>
<td>1  1  1  2  2  2  2  2  2  2  2  1  1  1  1</td>
</tr>
<tr>
<td>Lethargy</td>
<td>89</td>
<td>1  1  1  2  2  2  2  2  2  1  1  1  1  1  1</td>
</tr>
<tr>
<td>Enlargement of prescapular LN</td>
<td>100</td>
<td>1  1  1  2  3  3  3  3  3  3  2  2  2  2  2  2</td>
</tr>
<tr>
<td>Enlargement of prefemoral LN</td>
<td>100</td>
<td>1  1  1  1  2  3  3  3  3  3  3  2  2  2  2  2</td>
</tr>
<tr>
<td>Enlargement of parotid LN</td>
<td>100</td>
<td>1  1  1  1  2  2  2  2  2  2  2  1  1  1  1  1  1</td>
</tr>
<tr>
<td>Erosions of oral mucosa</td>
<td>72</td>
<td>1  1  1  2  2  2  2  2  2  1  1  1  1  1  1  1  1</td>
</tr>
<tr>
<td>Hypersalivation</td>
<td>13</td>
<td>1  1  1  1  1  1  1  1  1  1  1  1  1  1  1  1  1</td>
</tr>
<tr>
<td>Diarrhoea</td>
<td>13</td>
<td>1  1  1  1  2  1  1  1  1</td>
</tr>
<tr>
<td>Blood in faeces</td>
<td>56</td>
<td>1  1  1  1  1  1  1  1  1  1  1  1  1  1  1  1  1</td>
</tr>
<tr>
<td>Pallor of mucous membranes</td>
<td>33</td>
<td>1  1  1  1  1  1  1  1  1</td>
</tr>
</tbody>
</table>

* Results are a mean of the severity of the signs observed and are expressed on a scale of 1 to 3 (1 = mild, 2 = moderate and 3 = marked severity). Blanks indicate that lesions were not detected.

There was a reduction in the PCV, number of erythrocytes and Hb values (Fig. 2) which commenced at the start of the febrile period and persisted throughout the period of observation (up to 21 days after the onset of the febrile period). The MCV, MCH and MCHC remained within normal values indicating a normocytic normochromic anaemia. There was a reduction in the number of thrombocytes commencing 1–2 days prior to the onset of the febrile period and persisting until three days after the end of the febrile period (Fig. 2). No change was detected in the erythrocyte sedimentation rate.

A decrease in the amount of plasma protein occurred from the onset of the febrile period but it returned to normal approximately 15 days later. Blood urea concentrations increased markedly commencing three days after the start of the febrile period but in those animals that recovered they returned to normal 9–10 days later (Fig. 2); in 2 of the 3 animals that died or which were killed in extremis, blood urea concentrations in excess of 160 mg per dL were detected. Similar high blood urea concentrations were seen in only 2 of the other 15 animals that survived the infection. No significant change was detected in the concentrations of fibrinogen or creatinine.

Experimentally-induced disease in Ongole cattle

Six of 7 Ongole cattle inoculated with 1 mL of a 10% suspension of spleen tissue from an affected animal developed a febrile response (a rectal temperature in excess of 39.5 °C) of 2 to 5 days duration after an incubation period varying from 6 to 17 days. No other clinical signs were detected and no haematological examinations were conducted. All the Ongole cattle survived the infection.

Experimentally-induced disease in Friesian cattle

Two Friesian cattle infected with JDV developed similar clinical and haematological changes. A febrile period began four days after infection and persisted for five days (Fig. 3). There was a slight reduction in appetite during the febrile period, and slight enlargement of the prescapular lymph nodes was observed on the first day of fever and persisted until 10 days after infection (Fig. 3). Haematological changes included a transient leucopenia, due to lymphopenia and neutropenia, during the febrile period (Fig. 3). There was a reduction in the number of thrombocytes extending from the start of the febrile period to the termination of the experiment 11 days after inoculation (Fig. 3). No change was detected in the PCV (Fig. 3) or number of erythrocytes.

The two Friesian cattle were killed 11 days after inoculation. In one animal (F81) the only macroscopic pathological change detected was slight enlargement of the spleen and lymph nodes; histopathological changes were detected in the spleen (a slight parafollicular proliferation of lymphoreticular
Figure 2. Changes in rectal temperature, peripheral blood leucocytes, RBC concentrations, PCV, haemoglobin concentrations, thrombocyte concentrations, and urea concentrations in Bali cattle during the course of experimentally-induced Jembrana disease. The results are the mean of results from 18 affected cattle.
Figure 3. Clinical and haematological changes in Friesian cattle at intervals after infection with JDV. Shown are the changes in rectal temperature and degree of subcutaneous lymph node enlargement (A), peripheral blood leucocytes (B), PCV (C), and thrombocyte concentrations (D). The results are the mean of results from two inoculated Friesian cattle.

cells), liver (mild infiltration in the portal triads with lymphocytes), kidney (moderate infiltration of lymphoreticular cells into the intertubular areas), lungs (mild interstitial pneumonia with thickening and infiltration of the septa with lymphoreticular cells, and mild leucostasis in small blood vessels), and uterus (mucosa infiltrated with mononuclear cells). These changes in F81 were similar to those in mild cases of Jembrana disease in Bali cattle. In the other animal (F82) there were no macroscopic changes and histopathological changes were detected in only the lungs (alveolar septa slightly thickened and infiltrated with polymorphs), bronchi (erosions of the epithelial surface surrounded by polymorphs), and intestine (focal necrosis of Peyer's patches with polymorph infiltration). There was a good follicular response in the spleen and lymph nodes. These changes in F82 were not typical of lesions in Bali cattle with Jembrana disease but were those of a mild suppurative bronchitis and pneumonia. In neither
Table 3. Detection of virus in blood and/or spleen at various intervals after infection from Jembrana disease.*

<table>
<thead>
<tr>
<th>Species/breed</th>
<th>Tissue</th>
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<th>2</th>
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<th>11</th>
<th>19</th>
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<tbody>
<tr>
<td>Bali</td>
<td>Blood</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
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<td>-</td>
<td>+</td>
<td>+</td>
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<tr>
<td>Ongole</td>
<td>Blood</td>
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<td>+</td>
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<td>Madura</td>
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<tr>
<td>Rambon</td>
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<tr>
<td>Friesian</td>
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<td>-</td>
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<td>-</td>
<td>+</td>
<td>-</td>
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<td>+</td>
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<tr>
<td>Buffalo</td>
<td>Blood</td>
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<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
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<td>+</td>
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<tr>
<td>Sheep</td>
<td>Blood</td>
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<td>+</td>
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<td>-</td>
<td>+</td>
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<tr>
<td></td>
<td>Spleen</td>
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</table>

*Detection of the virus involved subinoculation of the tissue into susceptible Bali cattle and subsequent observation of the inoculated animal for clinical signs of Jembrana disease.

Experimentally-induced disease in Madura cattle

Eight of 10 Madura cattle inoculated with JDV developed a rectal body temperature more than 39°C for two or more days after infection (Fig. 4). The mean febrile response in these eight animals began five days after inoculation and persisted for four days (range 2–5 days). In pyrexic animals moderate enlargement of the superficial prescapular, pre-femoral and parotid lymph nodes was detected (Fig. 4) but there were no further clinical changes and all animals survived the infection.

In pyrexic cattle there was leucopenia (less than \(4 \times 10^9\) leucocytes per \(\mu\)L), lymphopenia and neutropenia for 3 days during the febrile period (Fig. 4). The number of thrombocytes also decreased during the febrile period but was always greater than 500 \(\times 10^9\) per \(\mu\)L (Fig. 4). There was a reduction in the PCV during the febrile period (Fig. 4) but no significant change was detected in the number of erythrocytes, concentration of total plasma protein or blood urea. No haematological changes were detected in the cattle that did not show a febrile response after inoculation.

Two of the Madura cattle were killed at the end of the febrile period, 10 days after inoculation. The only macroscopic change detected was a slight enlargement of the visceral and subcutaneous lymph nodes, and spleen. Histopathological changes consistent with a mild form of Jembrana disease in Bali cattle were detected and included mononuclear cell proliferation in the non-follicular areas of the lymph nodes and spleen, alveolitis and leucostasis in the lungs, and mononuclear cell infiltration in the kidneys and adrenal medulla.

Reinfection of the remaining eight Madura cattle with JDV 48 weeks after the initial infection did not cause a febrile response.

Experimentally-induced disease in Rambon cattle

Six of 10 Rambon cattle inoculated with JDV showed a rectal body temperature exceeding 39°C for two or more days (Fig. 5). The mean febrile response in these animals began 5 days after inoculation and persisted for four days (range 2–8 days). In the febrile animals there was enlargement of the subcutaneous prefemoral, prescapular and parotid lymph nodes from the start of the febrile period and persisting in some animals until 19 days after infection (Fig. 5); the persistence of the lymph node enlargement was greater but the degree of enlargement was less than in the Madura cattle. No other clinical changes were detected and all animals survived the infection.

In the six Rambon cattle that showed a febrile response, there was a mild reduction in the total number of leucocytes but the mean number did not decrease below \(4 \times 10^9\) per \(\mu\)L (Fig. 5). The mild leucopenia was due to a transient lymphopenia.
Figure 4. Clinical and haematological changes in Madura cattle at intervals after infection with JDV. Shown are the changes in rectal temperature and degree of subcutaneous lymph node enlargement (A), peripheral blood leucocytes (B), PCV (C), and thrombocyte concentrations (D). The results are the mean of results from 8 of the 10 inoculated Madura cattle that developed a febrile reaction.
Figure 5. Clinical and haematological changes in Rambon cattle at intervals after infection with JDV. Shown are the changes in rectal temperature and degree of subcutaneous lymph node enlargement (A), peripheral blood leucocytes (B), PCV (C), and thrombocyte concentrations (D). The results are the mean of results from 6 of the 10 inoculated Rambon cattle that developed a febrile reaction.
during the febrile period and a transient neutropenia in the immediate post-febrile period (Fig. 5). Febrile animals showed a transient reduction in the number of thrombocytes, similar to that detected in Madura cattle (Fig. 5). There was a progressive decline in the PCV (Fig. 5) from the start of the febrile period and continuing until the end of the observation period (27 days after infection). No change was detected in the number of erythrocytes, concentration of total plasma protein or blood urea in any of the cattle that developed a febrile response, and no haematological changes were detected in the four cattle that did not become pyrexic.

One Rambon animal with a febrile response was killed 14 days after inoculation and one 19 days after inoculation. In both animals, consolidation and oedema of the lungs were detected macroscopically and histopathologically. Other changes included lymphocyte proliferation in the follicular and non-follicular areas of the lymph nodes and spleen, lymphocytic infiltration into other organs including the liver, kidneys and lungs, and a severe bronchopneumonia. The changes were consistent with mild lesions of Jembrana disease in Bali cattle, complicated by secondary bacterial pneumonia.

Reinfection of the remaining eight Rambon cattle 46 weeks after the initial infection did not produce a febrile response.

Experimentally-induced disease in buffalo

Five of eight buffalo developed a rectal body temperature of greater than 39.5°C of 2-4 days duration after an incubation period varying 8-16 days. No other obvious clinical signs were detected but no haematological examinations were conducted. All buffaloes survived the infection.

Experimentally-induced disease in sheep, goats and pigs

No consistent febrile response or any other clinical signs of Jembrana disease were detected in inoculated sheep (n=9), goats (n=6) and pigs (n=6).

Persistence of the virus in infected animals

The persistence of the Jembrana disease virus in the blood or spleen of Bali cattle and the other inoculated species is shown in Table 3. Virus was recovered from the blood of Bali cattle for 25 months after recovery from clinical disease, from Ongole, Rambon and Madura cattle for 3 months but not after 6 months, from Friesian cattle for 1 month but not after 4 months, from buffalo for 9 months and from sheep for 4 months after inoculation. The agent was not detected in any of the tissue samples from inoculated goats or pigs, or from the buffalo before they were inoculated.

Inoculation of blood or spleen tissue from the persistently-infected Bali cattle produced a disease in subinoculated Bali cattle that was indistinguishable from that induced by subinoculation of tissues from animals with acute Jembrana disease.

In sheep the agent was detected by subinoculation of spleen tissue 1, 2 and 4 months after infection, but was not detected by subinoculation of whole blood obtained seven days after infection (Table 3). In Ongole cattle the agent was detected in spleen tissue one month after infection but not detected in whole blood obtained at the same time.

The approximate titre of JDV in blood of infected Bali cattle was determined at intervals after infection and the results are shown in Table 4. The titres (ID₅₀ per mL) in blood were <10⁴ until one day before the onset of fever when they increased to >10⁴. Titres of 10⁸ were detected on the second and third day of the febrile reaction, and 10⁵ one day after the end of the febrile period. The titre decreased to 10² 32 days after infection and a low titre of about 10¹ was detected 47 and 72 days after infection.

Resistance of recovered Bali cattle to re-infection

Only one of 18 Bali cattle previously affected with Jembrana disease that was induced experimentally with one of either two strains of JDV developed clinical signs of Jembrana disease when they were re-challenged with a third Tabanan/87 strain of JDV. This animal that reacted had been infected four months earlier with the Singaraja/86 strain, when it had developed a febrile reaction four days after infection which persisted for six days. After rechallenge, this animal developed a febrile reaction six days post-inoculation which persisted for six days. The animal was recumbent and unable to stand

Table 4. The titre of infectious virus in peripheral blood of Bali cattle at intervals after experimental infection with JDV.

<table>
<thead>
<tr>
<th>Days after infection*</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>7</th>
<th>8</th>
<th>12</th>
<th>32</th>
<th>47</th>
<th>72</th>
</tr>
</thead>
<tbody>
<tr>
<td>Titre (log ID₅₀/mL)</td>
<td>&lt;4</td>
<td>&lt;4</td>
<td>&lt;4</td>
<td>&gt;4</td>
<td>&gt;4</td>
<td>8</td>
<td>8</td>
<td>5</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>

*The febrile period in the sampled animals commenced six days after infection and persisted for six days.
two days after the rectal temperature had returned to normal, and was killed. Macroscopic and microscopic lesions typical of Jembrana disease were detected. All of the eight control animals challenged with the same inoculum developed typical clinical signs of Jembrana disease.

Discussion

The titre of infectious virus in blood and plasma on the second day of the febrile reaction was consistently about $10^8$ per mL and it was estimated that all the animals other than Bali cattle were inoculated with at least $10^7$ ID$_{50}$ of JDV. Bali cattle were inoculated with doses of virus varying from $10^1$ to about $10^8$ ID$_{50}$ and although there was a linear relationship between the dose and the incubation period there was no detectable relationship between the dose inoculated and the severity or duration of the disease induced.

The most common clinical changes encountered in the cattle experimentally infected with JDV were anorexia, lethargy, fever, erosions of the oral mucous membranes and enlargement of the superficial lymph nodes. Other signs less frequently observed were hypersalivation, nasal discharge, diarrhoea with blood in the faeces, pallor of the mucous membranes and a mild serous ocular discharge. These signs were accompanied by leucopenia principally due to a lymphopenia but also partly due to a neutropenia, an eosinopenia, a normocytic normochromic anaemia, thrombocytopenia, elevated blood urea concentrations and decreased plasma protein. Most of these haematological changes occurred during the febrile period, although the nadir of the neutropenia occurred after the febrile period had ended and the eosinopenia persisted until the observations were discontinued 21 days after the start of the febrile period. These clinical and haematological changes are similar to those reported in Jembrana disease by Teuscher et al. (1981); however, these authors reported that the thrombocytes practically disappeared during the febrile phase whereas the thrombocytopenia detected in the current study was only partial, and also that the erythrocyte sedimentation rate showed a considerable increase and this was not detected in the current study.

The high concentrations of blood urea in excess of 160 mg per dL in two of the three animals that died suggest this may have been a contributing factor to the death of these animals. These high values for blood urea are likely to be associated with the kidney lesions consistently detected in animals with Jembrana disease (Teuscher et al. 1981; Dharma et al. 1991).

The specific diagnosis of Jembrana disease in the field is difficult and is based upon clinical signs, principally anorexia, lethargy, fever, diarrhoea with blood in the faeces, enlargement of the superficial lymph nodes and "blood sweating" (Teuscher et al. 1981). These changes are very similar to those detected in the experimentally-infected cattle except for the so-called "blood sweating". "Blood sweating", the appearance of pin-point drops of blood on the cutaneous surfaces, especially of the limbs, was not observed in any of the 18 experimental animals examined in this report and it does not appear to be directly related to the disease process; although the aetiology is unknown it may be related to arthropod bites (Teuscher et al. 1981) and is perhaps associated with the thrombocytopenia, which may contribute to delays in the clotting mechanism.

The differential diagnosis of Jembrana disease in Bali cattle includes the sheep-associated form of malignant catarrhal fever (MCF), an enzootic disease in Indonesia to which Bali cattle are susceptible (Sudarisman et al. 1986). The clinical signs of MCF can also include anorexia, fever, depression, lymph node enlargement, oral erosions, and diarrhoea or dysentery (Plowright 1986). Although the clinical disease associated with MCF in Bali cattle may often be peracute, the clinical signs of MCF may vary considerably (Hoffmann et al. 1984), and individual cases of MCF and Jembrana disease may be difficult to distinguish. However, the clinical signs of Jembrana disease such as the ocular and nasal lesions and diarrhoea are always mild and never progress to the severe forms sometimes observed in MCF (Selman et al. 1974); the erosive lesions in the oral cavity are also mild and generally confined to the postero-lateral surface of the tongue. Nervous signs observed in MCF (Selman et al. 1974; Plowright 1986) were not observed in the animals with experimentally-induced Jembrana disease. Other features distinguishing the two diseases are that with Jembrana disease the incubation period after experimental infection was consistently 12 days or less whereas in MCF the incubation period is highly variable, ranging from 18 days to over 100 days (Liggitt et al. 1978). The mortality rate observed in the present study of experimental Jembrana disease was 17%, whereas in MCF the disease is progressive and the mortality rate is virtually 100% (Plowright 1986).

The inability to induce clinical signs of Jembrana disease other than a transient febrile response in Ongole cattle, Ongole x Bali cattle, Friesian cattle, buffalo and pigs, and the lack of a febrile response in inoculated sheep, goats and pigs, is consistent with field observations that clinical signs of Jembrana disease occur only in Bali cattle and no other cattle.
breeds or other animal species. However, while these other species did not develop an overt clinical disease they did develop mild clinical and haematological changes that were consistent with Jembrana disease in Bali cattle, and these same bovine species and sheep were shown to be infected with JDV for considerable periods after infection. However, the period of persistence of JDV in Friesian cattle, Ongole cattle and crossbred Bali x Ongole cattle was much less than in Bali cattle. In two of these animal species there may have been a longer period of persistence: in sheep, even though they did not develop any clinical response to JDV infection, virus was recovered from spleen tissue for at least four months after infection; the Indonesian buffalo, which are common draught animals in Indonesia, developed a persistent infection that persisted for at least nine months. The results suggest that species of cattle and particularly buffalo may be capable of maintaining the infectious agent in populations of animals in areas where there is only a low population of Bali cattle. It is also possible that if Bali cattle were not present, the Jembrana disease virus could persist in cattle populations without being recognised.

Sheep did develop a persistent infection, even though they showed no clinical reaction after they were inoculated with the infectious agent. However, the inability to recover the infectious agent from the blood of sheep seven days post-inoculation, and the need to inoculate spleen material to recover the agent, suggests that in sheep the number of infectious virus particles persisting after infection was very low. The apparently low titre of virus persisting in sheep, and the inability to detect the virus in goats and pigs after inoculation, suggests that sheep, goats and pigs are unlikely to act as significant vectors of the virus.

All Bali cattle developed clinical signs of Jembrana disease after infection but only some of the Ongole, Ongole x Bali cattle, and buffalo developed a febrile response. For example, only 80% and 60% of the Madura and Rambon cattle, respectively, developed clinical or haematological signs. However, although a febrile response may not have been detected, most of these animals were infected. As reported separately in these Proceedings by Hartaningsih et al. an antibody response to JDV was detected in all the Madura cattle (including two that did not show a febrile response) and 7 of the 8 Rambon cattle tested (including 3 of the 4 animals that did not show a febrile response). These serological results indicated the absence of clinical signs in some animals was not due to a failure to infect them with JDV.

Only limited studies were undertaken of the infection in Ongole cattle, buffalo, sheep, goats and pigs but more extensive studies were undertaken in the Friesian and crossbred Bali (Madura and Rambon) cattle. The clinical changes in Friesian and crossbred Bali cattle were a febrile response and concurrent lymphadenopathy. The extent and duration of the febrile response in these cattle types were less than in Bali cattle (Fig. 6) and the enlargement of the superficial lymph nodes during the febrile period, a striking characteristic of Jembrana disease in Bali cattle, was also less than that in Bali cattle (Fig. 6). Other clinical signs detected in Bali cattle with Jembrana disease were also of lesser severity or absent in Friesian and crossbred Bali cattle: lethargy and anorexia, common in Bali cattle, were detected only in the Friesian and these changes were mild; oculo-nasal discharge, pallor of the mucous membranes and bloody diarrhoea, which occurred in some Bali cattle with Jembrana disease, were not detected in any crossbred Bali or Friesian cattle.

More extensive clinical and haematological studies were undertaken in the Friesian and crossbred Bali cattle than in Ongole cattle, buffaloes, sheep, goats and pigs. Changes in the crossbred Bali cattle occurred only in those animals that developed a febrile reaction after infection. The changes detected in Friesian and crossbred Bali cattle, including the leucopenia, lymphopenia, neutropenia, and thrombocytopenia, were also less severe than those reported in Bali cattle. Some haematological changes consistently detected in Bali cattle were not seen in the Friesian and crossbred animals: decreased plasma protein concentrations were not detected. Likewise, increased blood urea concentrations, consistently detected in Bali cattle and in severe cases associated with marked glomerular swelling and hypercellularity in the kidneys (Dharma et al. 1991), were not detected; decreased PCV and erythrocyte concentrations consistently detected in infected Bali cattle, indicating a normocytic normochronic anaemia, were not detected in the Friesian and crossbred Bali cattle. There were some changes indicating anaemia in the other animal species but these were mild: in the crossbred Bali cattle there was a small decrease in the PCV, which was transient in Madura cattle but persisted for the observation period in Rambon cattle. No marked change occurred in the PCV of infected Friesian cattle, and the erythrocyte count remained normal in all crossbred and Friesian cattle.

Although only two Madura, Rambon and Friesian cattle were examined post mortem, all had developed a febrile response and were killed during the immediate post-febrile period, when striking lesions were consistently detected in Bali cattle (Dharma et al. 1991). Histological lesions consistent with a mild form of Jembrana disease in Bali cattle were
Figure 6. Comparative temperature (A) and lymph node changes (B) in Bali cattle, Friesian, Rambon and Madura cattle at intervals after infection with Jembrana disease virus. The changes were more severe and of longer duration in Bali cattle than the other cattle types.

detected in both the Madura cattle, but they were seen in only one of the two Friesian cattle, and in one of the two Rambon cattle.

The predominant histological lesions reported in Bali cattle with Jembrana disease (proliferation of lymphoreticular and lymphoblastoid cells in the parafollicular regions of the spleen and lymph nodes) occur in lymphoid organs (Dharma et al. 1991). This was also true of lesions detected in the crossbred Bali and Friesian cattle. However, whereas in the spleen and lymph nodes of Bali cattle there was follicular atrophy and a scarcity of plasma cells in the lymph nodes and spleen until five weeks after infection (Dharma et al. 1991), there was a marked follicular response in the spleen and lymph nodes of the crossbred Bali and Friesian cattle during the immediate post-febrile period. Infiltration of mononuclear cells into non-lymphatic organs such as the liver, kidneys, adrenal, heart and choroid plexus was common in Bali cattle (Dharma et al. 1991); however, when detected in the Madura, Rambon and Friesian cattle, it was less intense. Pulmonary histological lesions were consistently detected in Bali cattle with Jembrana disease (Dharma et al. 1991).
the crossbred Bali and Friesian cattle, similar although mild pulmonary lesions consisted of infiltration of lymphoreticular cells into the septa and mild leucostasis in small blood vessels. As in Bali cattle, pulmonary lesions were sometimes complicated by histopathological lesions of bacterial pneumonia: Pasteurella pneumonia is common in Indonesian cattle (Dharma et al. 1991) and possibly the pneumonia pre-existed or was exacerbated by JDV infection.

Friesian and crossbred Bali cattle, unlike Bali cattle (Dharma et al. 1991), showed no parafollicular proliferation of mononuclear cells in intestinal lymphoid tissue. Haemorrhagic lesions, seen in infected Bali cattle (Dharma et al. 1991) were also not detected. Glomerular swelling and hypercellularity in the kidneys associated with increased blood urea concentrations, suggested as a possible cause of death in some cases of Jembrana disease in Bali cattle (Dharma et al. 1991), were not seen in the present study.

Although the lesions detected in the two types of crossbred Bali cattle were similar, they were of greater severity in the Madura than in the Rambon cattle. In Madura cattle, a higher percentage of animals became febrile, enlargement of the subcutaneous lymph nodes was greater, the leucopenia was of greater magnitude, and the antibody response to JDV was greater. The differences in magnitude of the response in the two types of crossbred cattle may be due to genotypic differences between them. Madura cattle belong to a stable breed derived by crossbreeding Bali and Bos indicus cattle. They are possibly more closely related genotypically to Bali cattle than are animals of the Rambon type (F1 generation Bali × Ongole (Bos indicus)). There is anecdotal evidence that F1 male progeny of Bali × Ongole cattle are infertile (Rouse 1972) and development of the Madura breed would have required further crossbreeding with male Bali cattle.

Although JDV has an antigenic (Kertayadnya et al. 1993) and genetic (Chadwick et al. 1995a, b) relationship with bovine immunodeficiency-like virus (BIV), there were major differences between the disease detected in the Friesian (Holstein) cattle infected with JDV and those that have been reported for BIV. In the Friesians leucopenia due to a lymphopenia and a neutropenia was detected. In contrast, the R29 strain of BIV inoculated into Holstein-Friesian calves was reported to cause a mild transient leucocytosis, lymphocytosis and lymphadenopathy (Van der Maaten et al. 1972), and mild follicular hyperplasia in lymphoid tissue five to six weeks after inoculation (Carpenter et al. 1992). Direct comparison of the histological changes in JDV and BIV-infected Friesian cattle is difficult as these have not been described for BIV-infected cattle until about five weeks after infection, at which time lesions in JDV-infected cattle are regressing and there is a marked increase in the lymphoid follicular reaction (Dharma et al. 1991). In the one Friesian animal infected with JDV that showed pathological changes 11 days after infection, there was a good follicular response in the lymph nodes and the degree of proliferation of lymphoreticular cells in the parafollicular areas of the spleen, a marked characteristic of the disease in Bali cattle (Dharma et al. 1991), was less than in Bali cattle. Further comparative studies of the earlier changes in Bos taurus cattle infected with BIV and later changes after infection with JDV are warranted.

Only one of 18 animals in which Jembrana disease had been experimentally induced up to 22 months previously developed Jembrana disease when re-challenged with the infectious agent. These results indicate that immunity does develop after primary infection with JDV.

In conclusion, it was demonstrated that JDV can replicate and produce lesions in a range of cattle types in addition to Bali cattle. However, while infection caused a severe disease in Bali cattle, it resulted only in a mild disease and sometimes a subclinical infection in the other cattle types. The disease in these other cattle types would be difficult to detect under field conditions. These experimental observations were consistent with serological evidence that in areas of Indonesia where the disease is endemic, cattle other than Bali cattle are sometimes infected (Hartaningsih et al. 1993).

References


The Pathology of Jembrana Disease

D.M.N. Dharma

Abstract

The pathology of experimental and natural field cases of Jembrana disease is similar. The striking gross lesions during the acute phase, approximately 2-4 weeks post-infection, are generalised lymphadenopathy and splenomegaly.

Microscopically, in the first week post-infection (Phase 1), there is a general lymphoreticular reaction affecting both follicular and non-follicular compartments. The acute phase (Phase 2), 2-4 weeks post-infection is signified by follicular atrophy and an intense non-follicular lymphoreticular hyperplasia and a similar infiltrative and proliferative process in liver, kidneys, adrenal medulla and elsewhere. Leucostasis, mainly occurring in the smaller blood vessels of the lungs, has a very high diagnostic value. In the third phase, starting from the fifth week of infection, there is a marked lymphoid follicular reaction and plasma cell formation. Residual lesions occur up to 60 days post-infection.

Temporary immunosuppression appears to occur during the acute phase of the disease as indicated by follicular atrophy, a decrease in the immunoglobulin G-containing cells in the lymphoid organs and a decrease in the BoCD4:BoCD8 T-lymphocyte ratio in lymph node follicles.

Pathological Changes of Field Cases of Jembrana Disease

The major pathological changes of field cases of JD from Bali (1983-95), Lampung (1976), West Sumatra (1992), South Kalimantan (1994) and East Java (1978) are principally the same. The general post mortem picture is dominated by reactive changes in the lymphoid system namely lymph nodes and spleen, and haemorrhages in various tissues such as epicardium, endocardium, serous and mucous membranes. The striking histopathological changes consist of proliferation of lymphoreticular cells in the lympho-haemoipoietic system and infiltrative changes in other organs such as lungs, adrenals, liver, kidneys and choroid plexus. Leucostasis is consistently detected in the lungs and other tissues. Using an electron microscope, the large mononuclear cells with abundant cytoplasm causing leucostasis were proven to be intravascular macrophages (Budiarso and Rikihisa 1992). There are no significant changes observed in the central nervous system. It is concluded that the general pathological changes observed in recent cases of JD are basically as same as those described by Adiwinata (1967), Pranoto and Pudjiastono (1967), Ishitani (1968), Budiarso and Hardjosworo (1976, 1977) and Teuscher et al. (1981).

Sequential Pathological Changes in Bali Cattle Experimentally Infected with Jembrana Disease Virus

This experiment was conducted to understand better the pathogenesis of JD. In this experiment, animals were killed at specified intervals after intravenous
inoculation of the Jembrana disease virus (JDV). Twenty Bali cattle were allotted into 10 groups of two animals each and were all intravenously inoculated with 1 mL of a 10^{-5} dilution of 220 nm filtered plasma from a donor animal. Two animals were then killed and necropsied at various intervals after infection. In addition, four animals were intravenously inoculated with 1 mL of medium 199 containing 10% foetal bovine serum and kept in close contact with the experimental animals, then killed and necropsied at 2 and 10 days after inoculation. They served as in-contact control animals.

This experiment revealed that infection of Bali cattle with plasma from a JD-infected donor animal caused a well-defined pathological response. The general gross picture was dominated by signs of vascular damage in the form of mild exudates and haemorrhages, and reactive changes in the lymphoreticular system especially. The most striking gross changes were lymphadenopathy and splenomegaly. Enlargement of lymph nodes was first observed at day 6 post infection (PI), becoming severe from days 10 to 15, and still apparent up to day 30 PI. Splenomegaly was first observed two days PI and became most marked from days 8 to 15 PI. Mild splenomegaly was still seen at day 42 but not at day 60 PI. Petechial and ecchymotic haemorrhages and oedema were observed in various visceral organs including the gastrointestinal tract, heart and kidneys. Mild apical consolidation of the lungs and multifocal greyish foci of the heart and kidneys were also constantly present.

Microscopic examination revealed three distinct phases of JD. The initial phase, occurring in the first week of infection, was indicated by a general lymphoreticular reaction involving both follicular and non-follicular compartments of the lymphoid organs. The second phase, occurring from 8 to 21 days PI, was signified by an intense non-follicular proliferative response by reticular and lymphoblastic cells in lymphoid organs, with a similar infiltrative and proliferative process in the lungs, heart, liver, kidneys, adrenal medulla and choroid plexus. The third phase, which developed from the fifth week of infection, was signified by a marked lymphoid follicular reaction and plasma cell formation which was prominent in the medullary cords of the lymph nodes and the non-follicular areas of the spleen. The cytology and histological distribution of proliferative changes in the lymphoid system suggested that during the acute phase of the disease, a predominantly T-lymphocytic reaction took place, perhaps associated with transient humoral immunosuppression. Residual lesions occurred up to day 60 PI. Detailed results of this experiment have been published (Dharma et al. 1991).

Sequential Immunohistochemical Findings in the Lymphoid Organs of Bali Cattle after Inoculation with JDV

This study was carried out using the peroxidase-antiperoxidase test for immunoglobulin-containing cell (ICC) assessment and the indirect test for lymphocyte subset assessment.

Assessment of ICC

For ICC assessment, formalin-fixed lymphoid tissues (spleen and lymph node) from animals used for the sequential study of the pathology of experimental JD were used. Tissue preparation and staining procedures are described by Dharma (1992). Briefly, this study revealed that the prevalence of immunoglobulin--containing cells (IgG-CC) in the follicular and medullary cord compartments of the lymph nodes, and follicular and non-follicular compartments of the spleen, dropped from day 2 PI until day 21 PI, then increased sharply thereafter until day 60. This pattern was similar to the pattern of antibody detected in the blood (Hartaningsih et al. 1993). Immunoglobulin G-CC were most prevalent in the medullary cords of the lymph nodes and the non-follicular compartment of the spleen. ImmunoglobulinM- and IgA-CC were, however, scattered in all compartments of the lymphoid organs but the numbers were low and the standard deviations were high.

Assessment of lymphocyte subsets

To assess lymphocyte subsets in the lymph nodes of Bali cattle after infection with the JDV, animals were allotted into three groups of three animals each and were all intravenously inoculated with 1 mL of the 10^{-5} dilution of 220 nm filtered plasma from the donor animal. All animals were subsequently killed by exsanguination and necropsied on days 4 (incubation phase), 10 (acute phase) and 42 (recovery phase) PI. Three animals were intravenously inoculated with 1 mL medium 199 containing 10% FBS and killed and necropsied at day 14 PI to serve as control animals. Tissue preparation and staining procedures are described by Dharma (1992).

Briefly, this study revealed that BoCD4 and BoCD8 lymphocytes were detected in all lymph node compartments in all sections from every experimental animal. They were found in the largest numbers in the parafollicular and paracortical areas of the lymph nodes. In this study no significant differences in the ratio of BoCD4:BoCD8 lymphocytes in the paracortex were found between different clinical phases of the disease. The numbers of BoCD4 and BoCD8 lymphocytes in the follicles
varied remarkably between follicles within the same section. In this study, a significant decrease (P < 0.05) in the mean BoCD4:BoCD8 ratio in the follicle was noted during the acute phase of the disease. Scattered BoCD4 and BoCD8 lymphocytes were also seen in the sinuses and the medullary cords of the lymph nodes but no clear numerical difference was apparent. Detailed results of the experiment on the immunopathology of JD have been published (Dharma et al. 1994).

Conclusions and Recommendations

Jembrana disease appears to be unique in Bali cattle and seems to have no obvious parallel in any other species. The pathology of JD has been well documented and can be used as diagnostic tools. The pathology of experimental and natural field cases of JD is basically the same. It induces severe lymphoreticular hyperplasia in the non-follicular areas of the lymphoid organs in a neoplastic-like fashion but regression eventually follows. Prominent 'leucostasis', such as seen in the lungs of an infected animal has not been described in other bovine diseases. Without complication by secondary bacterial infection, the acute phase is usually followed by recovery.

Knowledge of the pathogenesis of JD at this stage is far from complete. Therefore, more detailed sequential lymphocyte subset studies in the lymphoreticular tissues and peripheral blood are recommended to understand better the immunosuppressive mechanism in JD. Identification of the precise target cells, for example, by a double-labelling immunoperoxidase test, is suggested to provide more information on the kinetics of JDV, particularly during the early immune response which is related to the antigen presentation mechanisms. The recently developed in situ hybridisation technique may later be applied to detect minute amounts of viral nucleic acid in the tissues, to explain the persistence of the virus.

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Bali Cattle: Origins in Indonesia

J.W. Copland

THE origin of Bali cattle, *Bos javanicus* d'Alton, in Indonesia, is part of the complex evolution of all cattle over a long time. The evidence is clouded in antiquity. There is some information from fragments of fossils and limited evidence from anthropological and archaeological findings, from ancient and modern history. Although there is no formal basis on which to make a statement on the origin of Bali cattle, there are intriguing clues which suggest a possible process of evolution.

**Origin of Cattle**

The earliest known type of wild cattle, *Bos acutifrons* comes from a part of a fossil found in India (Payne 1970). The wild cattle (*Bos acutifrons*) then developed into two streams: the aurochs (*Bos primigenius*) that inhabited the forests of Asia, North Africa and Europe; and the urus (*Bos namadicus*) found in India and other parts of Asia. One view is that the centre of origin of the cattle family, the Bovidae, was the old world tropics or subtropics of Asia, North Africa and Europe (Pilgrim 1947). Most views expressed on the origin of cattle focus on the auroch as the foundation of most of the cattle species and breeds developed in the world. The auroch, although an ancient cattle type, has survived into historical memory as the last recorded animal was killed in a Polish forest in the seventeenth century (Payne 1970). They were large animals with enormous horns, the latter a characteristic found in modern cattle species.

Very little is known of the origin of *Bos (Bibos)* type cattle in Southeast Asia. They appear to have become separated from the *Bos* type in the Upper Pliocene period (Zeuner 1963). Cattle of this type superficially resembled Zebu, as they possessed a hump, although the bone structure of the head is quite different. It has been suggested that the modern *Bos (Bibos)*, such as Bali cattle, is more similar to ancestral cattle than other modern types.

The widespread distribution of wild species of cattle in the temperate and subtropical climatic zones of the ‘old world’ and findings in the Upper Pleistocene period make it particularly difficult to discover the original centre(s) of domestication (Payne 1970). Domestification could have occurred at one point or several points at the same time. It is thought that there must have been at least two centres of domestication of cattle in Southeast Asia. The geographical distribution of *Bos (Bibos)* types of cattle suggests that the centre of domestication was Indo-China and Malaysia, later spreading to Bali. The other centre of domestcification was probably Assam-Burma and led to the development of the gayal.

The centre of domestication is a geographical region where a type of cattle migrated and evolved into another type due to natural selection and the influence of man. The cattle may have migrated naturally or as a result of man’s role as a hunter. In these centres, the results of natural selection over a long time transformed the surviving animals into a type which best suited the biophysical environment. In the first instance, natural selection was the force as it is most unlikely to have been influenced by man. However, as man became a sedentarised farmer, he started to utilise all the resources around him, including cattle. The process of domestication integrated cattle into the agricultural system and developed an interdependence between man and animals.

A tentative and general picture of the origins of Bali cattle is given in Figure 1 and the global location of the main cattle breeds of today in the world is given in Figure 2.

The evolution and origins of Bali cattle could be summarised as follows (adapted from Friend 1970).
Figure 1. Distribution in the tropics and subtropics of the major types of cattle (Payne 1970).

Figure 2. Possible migration routes of domestic cattle in Asia (Payne 1970).
Common Ancestors

Pliocene period: some 7 million years ago

Natural selection and migration leading to the following ancient breeds

Pleistocene period 2 million years ago leading to

- *Bos bonanus* European bison
- *Bos gaurus* Guan of India
- *Bos grunniens* Yak
- *Bos nomadicus* Wild cattle of India
- *Bos primigenius* Wild Aurochs
- *Bubalus bubalus* Indian Buffalo
- *Bos banteng* Banteng of Burma

Domestication and migration of man and *Bos banteng*

Domestication period of *Bos banteng* 10 000–5000 years ago in Indonesia

- *Bos javanicus* Indonesia, Thailand, Indo-China, Australia

Not all cattle types have been domesticated. For example, the American bison, in spite of many attempts by American Indians, have not been domesticated. Bali cattle in one sense are not fully domesticated, as they have the capacity to revert readily to the wild state, called the banteng, at any stage of life. Conversely, they can be domesticated relatively easily. The ability to move between the wild and domestic states is a possible reflection of recent domestication in Indonesia.

Indonesia probably became the centre of Bali cattle domestication some ten to five thousand years ago. The process of domestication probably started in prehistoric times on Bali and Java, according to Meijer (1962). Bali cattle migrated in the 19th and 20th centuries from Indonesia into Malaysia (Devendra et al. 1973) and into Australia (Lett 1962; Calaby 1975). Often the number imported was small. The 20 animals imported into Australia in the early 1800s resulted in a wild feral population of more than 2000 cattle 150 years later (Lett 1964). Interestingly, the importation of Bali cattle from Timor did not introduce Jembrana disease, but it may have resulted in the introduction of cattle tick and buffalo fly into Australia.

Indonesia has two types of Bali cattle, a domestic type, called Bali cattle and a wild type named banteng. There are not many wild bantengs left in Indonesia. They are now mainly found in the national parks such as Ujung Kulon and Baluran (Wind 1978).

Taxonomy of Bali Cattle

The taxonomy of a species can reflect its origins. The scientific name of Bali cattle has been loosely used in the scientific literature during the past century, perhaps the result of wild and domesticated examples of the species existing at the same time. The taxonomy was resolved by Hooijer (1956) in a study of the authenticity of earlier reports and observations.

The scientific name of Bali cattle is *Bos javanicus* (d'Alton); d'Alton identified the genus and species and described the taxonomy of Bali cattle in 1823 which precedes all other descriptions and names.

The formal classification of Bali cattle is as follows:

- Kingdom Animal
- Order Artiodactyla Cloven hoofed animals
- Class Ruminantia Animals with a rumen
- Family Bovidae
- Genus Bos
- Species javanicus (d'Alton)

Synonyms of *Bos javanicus* are:

- *Bos leucopyrnumus* by Quoy and Gaimard (1830). This is rejected because it was based on hybrid and was after d'Alton's description.
- *Bos sondaicus* by Muller (1840). Rejected as it was after d'Alton's description.
- *Bos banteng* by Temminck (1840). Rejected.
- *Bos bantinger* by Schlegel and Muller 1855. Rejected.
- The holotype of the species is located in the Leiden Museum in the Netherlands. It is a male skeleton from Java and described by d'Alton and subsequently confirmed, although misnamed by other taxonomists (Schlegel and Muller 1845, and Rutimeyer 1867).

Without any doubt, Bali cattle (*Bos javanicus*) are an independent and distinct cattle species.

Indicators that banteng and Bali cattle are the same species of *Bos javanicus* d'Alton

Morphological evidence is given by Hayashi (1981) in a study of the multiserial craniometric analysis of the relationships between wild banteng and five types of native Asian cattle in Indonesia, Philippines and Korea. In this study, Bali cattle and wild banteng were found to have similar cranial relationships.

The chromosomes of the two types are identical and consist of 29 acrocentric chromosome pairs and two submetacentric sex chromosomes, with a diploid of $2n = 60$ (Fischer 1971).

In a global survey of the skin of cattle types and breeds, Jenkinson and Nay (1973) examined the sweat glands of Bali cattle and banteng and found...
that they were similar. The shallow hair follicle in *Bos javanicus* is part of the reason for their superior heat tolerance.

The Australian experience described by Letts (1962, 1964) of importing domestic Bali cattle (*Bos javanicus*) from Bali and Timor and then letting them become feral has shown that the domestic Bali cattle can revert to the wild banteng quickly and become indistinguishable from the native banteng as seen in Indonesia.

The uniqueness of Bali cattle

Bali cattle are different from all other species of cattle as a result of their origin and evolution. They will crossbreed with European cattle, *Bos taurus*, but the male offspring as reported by Jellinek et al. (1980), is usually infertile. Bali cattle do have some distinct characteristics which have made them the cattle breed of choice in many parts of Indonesia. Differences between Bali cattle and *Bos taurus* and *Bos indicus* may influence their responses to physiological and pathological events such as Jembrana disease and may explain the variation seen in the susceptibility observed in the field.

Some of the unique differences of Bali cattle from other cattle which make them a valuable asset in Indonesia are:

**Size** Smaller than Zebu and European cattle (Anon. 1983)

**Colour** Strikingly uniform markings (Payne and Rollinson 1973)

**Behaviour** Timid and can become wild readily

**Reproduction** High conception rates

**Lactation** Ability to stop lactating and survive a bad dry season

**Feed utilisation** Better able to utilise low quality feed base

**Heat tolerance** Better heat tolerance than buffalo and other cattle (Moran 1973)

**Water turnover** Lower water turnover than *Bos taurus/Shorthorns* (Siebert and Macfarlane 1969, Jenkinson and Nay 1973)

**Meat quality** Marked fat deposition sites and limited fat in muscle mass

**Disease pattern** Stated to be resistant to external and internal parasites except liver fluke, *Fasciola gigantica*. Increased resistance to malignant catarrhal fever and Jembrana disease.

Due to the difference in origin, Bali cattle are genetically unique compared to other cattle, and are likely to have a subtle and different spectrum of characteristics of production and disease responses. They are well adapted to the Indonesian environment, particularly in Eastern Indonesia, and once Jembrana disease is controlled, they have the potential to be a major national asset for the livestock sector.

**References**


Bali Cattle — Their Economic Importance in Indonesia

S. Wiryosuhanto

Abstract

Indonesia covers an area of 9.8 million km² of which 1.9 million is land. The population was about 195 million in 1995, and Gross National Product (GNP) per capita was US$900 in 1994. The agricultural sector has made a major contribution to the Indonesian economy, growing more than 4% per year during the past decade, with the contribution of livestock to agricultural GNP increasing from 6% in 1969 to 11.5% in 1994. This paper examines the role of livestock in Indonesia from the point of view of its contribution to food production, and as a means of generating income and employment, of sustaining agriculture and the environment, and of reducing poverty. The economic importance of Bali cattle is discussed, as well as their role in livestock development in Indonesia.

INDONESIA is an archipelago of more than 17,000 islands extending 5100 km from west to east and 1888 km from north to south. Administratively, Indonesia consists of 27 provinces, 243 districts, 60 municipalities, 3839 subdistricts and 65,554 villages. Out of a population of about 195 million (1995), about 109 million (54%) live on the island of Java, which accounts for only 7% of the total land area. Gross National Product (GNP) per capita in 1994 was about US$900.

Thirty years ago, Indonesia was one of the poorest countries in the world, with a GNP per capita of US$50, half that of India and Bangladesh. In 1969, the Government of Indonesia (GOI) adopted a new strategy for development, the First Twenty-Five Year Long-Term Development Plan (PJP 1), and pursued it through successive Five-Year Development Plans (Pelita) for broad-based economic growth, particularly related to rural development. Agricultural development was given one of the highest priorities, with food self-sufficiency as a target.

The agricultural sector made a major contribution to the Indonesian economy, growing more than 4% per year during the past decade. A large part was due to a rapid growth in rice production, but also to steady growth in livestock, estate crops and fisheries. The result was that poverty among rural smallholders was reduced.

Although the contribution of agriculture to GNP decreased during the past 25 years from 42% in 1969 to 18% in 1993, the role of agriculture in the Indonesian economy is still important, given that 51.1% of households are still engaged in agriculture. On the other hand, the contribution of livestock to agricultural GNP increased from 6% in 1969 to 11.5% in 1993.

Role of Livestock in the Indonesian Economy

Livestock for food

The contribution of livestock to food supplies in Indonesia is increasing at a high rate. Meat production rose by 375% between 1969 and 1994, from 309,300 tonnes to 1,469,200 t. During the same period, egg production increased by 905% from 57,700 tonnes to 580,300 t, and milk by 1244% from 28,900 t to 388,600 t.

As income determines the protein intake of people, particularly in urban areas, higher incomes result in higher demand for animal products. Of the different animal species, meat production from monogastric animals (poultry and pigs) increased faster than that of small ruminants (sheep and goats) or large ruminants (cattle and buffalo). Trends in meat production for different animal species in Indonesia are presented in Table 1.
Table 1. Meat production by species 1969–1994 (Indonesia) (‘000 tonnes).

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*Preliminary figures.

Livestock for income and employment

At the farm level, livestock provide income and so increase the economic stability of households because small stock act as a cash buffer, and large stock as a capital reserve. They also act as a hedge against inflation and reduce the risk associated with crop production in mixed farming systems.

At the national level, livestock products represent 11.4% of total agricultural output. The livestock sub-sector has achieved the greatest growth in production during the past 25 years and it is expected to increase by 6.4% in Pelita VI, with food crops increasing by 2.5%, estate crops by 4.2% and fisheries by 5.2%.

Livestock are a source of general employment. Milk production can be very labour-intensive at the farm level, and sheep, goats and poultry provide an important source of part-time jobs for rural families.

During Pelita V, livestock generated 328,000 jobs, and during Pelita VI, a target of 456,000 jobs has been set.

Livestock in sustainable agriculture and the environment

Mixed farming systems that include livestock offer many advantages by providing the basis for a more profitable and sustainable agricultural system by adding value to crop residues, and by providing manure. Large animals also provide draught power.

The use of livestock to graze vegetation under coconut, oil palm and rubber plantations increases production and reduces the costs of weed control. Such systems also safeguard the environment and avoid pollution while supplying additional organic matter to the soil.
Feeding crop residues to livestock and using their manure as fertilizer and as a soil conditioner benefits the environment directly. Soils treated with manure have better structure, water retention and draining capacity. As a result, crops grow faster, provide good ground cover and reduce erosion.

The number of cattle and buffalo used as draught animals, as well as for meat and milk production, has increased by 70% and 7% respectively during the past 25 years. Trends in livestock populations for the different animal species are shown in Table 2.

**Contribution of livestock to poverty reduction**

In general, livestock can best contribute to poverty reduction through improving efficiency in integrated farming systems other than through the addition of free standing intensive enterprises.

Experience under the recent GOI — Smallholders Cattle Development Project indicates that the income of farmers with cattle was higher than those without them, primarily because of the capacity to use cattle as draught animals, use of crop residues for fodder, grazing on land otherwise not used, and the contribution of manure to soil fertility.

The importance of livestock as a source of income for poor farmers is demonstrated by the fact that in Bangladesh, the Grameen Bank, which assists the “poorest of the poor”, provides about 50% of its loans for the purchase of livestock. The same applies in Indonesia where 70% of funds for “desa tertinggal” under “Inpres Desa Tertinggal” (IDT) were used for purchasing livestock.

**Economic Importance of Bali Cattle**

The role of Bali cattle in agriculture is almost the same as that of livestock in general agriculture. The species contributes to meat production, acts as a source of income, generates employment, provides

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<td>330</td>
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<td>3109</td>
<td>11886</td>
<td>6485</td>
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*Preliminary figures.
manure for improved soil fertility, converts roughage into high value products, and provides draught power.

In Indonesia, Bali cattle are considered an original species with several economic advantages, compared to other species.

**Historical background**

Bali cattle (*Bos sondaicus*) is a domesticated species of banteng (*Bibos banteng*) which was known from ancient times in Burma, Thailand, Indochina, the Malaysian peninsula, Sumatra, Java and Bali.

Wild banteng are still found in West Java (Ujung Kulon Wildlife Reservation) and East Java (Baluran Wildlife Reservation). Bali cattle are similar in type and appearance to wild banteng, but where and when their domestication took place is still a matter of scientific debate. However, a Special Veterinary Conference in Pamekasan Madura in 1934 concluded that domestication of wild banteng did take place in Indonesia, sustaining a certain species purity on Bali Island.

**Distribution**

Bali Island is recognised as the main source of pure Bali cattle. From Bali, they spread to other parts of Indonesia, and to Malaysia and Australia. Bali cattle were imported to Lombok island by the ancient monarch and Lombok subsequently became the second source of pure Bali cattle. They were introduced to South Sulawesi in the 1890s and to Java in 1907. They performed satisfactorily in South Sulawesi and Mojoagung, East Java, but unsuccessfully in West Java, due to malignant catarrhal fever, a virus infection carried by sheep. Bali cattle were then introduced to Timor, Sumbawa, the Moluccas, Irian Jaya and other parts of Sumatra and Kalimantan. Trends in Bali cattle numbers and distribution are shown in Table 3.

**Table 3. Bali cattle and other breed populations and distribution in Indonesia (1988).**

<table>
<thead>
<tr>
<th>Provinces</th>
<th>Ongole</th>
<th>P.O</th>
<th>Bali</th>
<th>Madura</th>
<th>Other</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>DI. Aceh</td>
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<td>3107</td>
<td>84</td>
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<td>179024</td>
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<td>6867</td>
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<td>620</td>
<td>9709</td>
<td>126179</td>
<td>151080</td>
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<td>116349</td>
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<td>DI. Yogyakarta</td>
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<td>26949</td>
<td>185</td>
<td>797</td>
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<td>185353</td>
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<tr>
<td>Jawa Timur</td>
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<td>42234</td>
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<td>113239</td>
<td>611855</td>
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<td>57139</td>
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<td>35203</td>
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<tr>
<td>Sulawesi Utara</td>
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<td>230627</td>
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<td>Sulawesi Tengah</td>
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<td>Sulawesi Selatan</td>
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<td>67559</td>
</tr>
<tr>
<td>Irian Jaya</td>
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<td>0</td>
<td>7654</td>
<td>0</td>
<td>25422</td>
<td>33076</td>
</tr>
</tbody>
</table>

Total in Indonesia (Percent of total cattle in Indonesia) 260094 (2.64) 773165 (7.85) 2632125 (26.75) 1131371 (11.5) 5045393 (51.26) 9842148 (100)

Comparative advantages

Pioneer cattle

Because Bali cattle were seen to thrive in South Sulawesi, Kalimantan, Irian Jaya and other areas where cattle had not previously been developed, they were regarded as a pioneer species that could go wild or semi-wild if left in the jungle, but could be easily domesticated.

High fertility rate

The fertility rate is the percentage of normal calving produced by a group of mated female animals within a year. Some researchers found that the fertility rate of Bali cattle was very high.

Some observations by scientists in a number of locations were as follows:
- Aalfs (1934) on Bali island, 83%;
- Wardoyo (1950) in South Sulawesi, 82%;
- Davendra (1973) in Malaysia, 82%;
- Moran (1971) in northern Australia, 90%;
- Kirby (1972) in northern Australia, 100%.

Normal length of pregnancy and ease of calving

The average length of pregnancy of Bali cattle observed during the Bali Cattle Breeding Project at Pulukan, Bali, was 287.6 days, with male calves taking 2.7 days longer than female calves. In general, all cattle breeds have a similar length of pregnancy, between 275 and 298 days.

Observations of 1000 calvings of Bali cattle (in Bali) showed that more than 90% of foetuses were in the anterior position which enabled calving to take place easily; the rest were in the posterior position. Dystocia cases caused by malposition (parturition abnormalities) were rare.

Pregnancy rate at first mating

Pregnancy rate at first mating is a very important determinant of the fertility of a group or breed of cattle. As already noted, the pregnancy rate of Bali cattle is high.

Observations in Penebel, Marga and Sukasada for the three years 1978–1980 of 2000 pregnancies showed an average rate of 88%. Other researchers found rates between 80% and 86%. The pregnancy rate of Bos taurus is between 50% and 70% while that of Bos indicus is lower, ranging between 35% and 61% (Cloufa 1955).

Higher calving percentage

The calving percentage per year of a group of cattle is the number of calvings divided by the number of adult cows, multiplied by 100. Darmadja (1980) found the calving percentage of Bali cattle was 52.15%. Other research in Indonesia indicated the calving rate for Bali cattle on Timor was 64% ±1.6%, Flores 78% ±12.8%, Sumbawa 72% ±21.5%, Lombok 74.4% ±11.3%, with the average 72.1% ±14.3% higher than Ongole cattle 40.4% ±21.4%.

The Bali Cattle Breeding Project reported that by strict selection (culling of infertile and unproductive cows) the fertility rate of Bali cattle is increasing from 74.08% to 93% with the calving interval reduced from 538 days to 421 days. It is expected that the calving percentage per year will increase by about 29%, from 52.15% to about 80.6%.

High adaptability

As has been mentioned, Bali cattle have a high adaptability, being able to survive in any area, climate or condition, such as the wet, arid and semi-arid areas of Indonesia, from Sumatra to Irian Jaya.

High response to fattening

Research in Bali by Nitis (1979) on the effect of replacing 30% of forage with concentrated feed showed that the body weight of Bali cattle fed on concentrates was 2.7 times heavier than those given green roughage, or 1.7 times heavier than cattle raised by traditional farmers. On such findings, it could be concluded that Bali cattle have a high response to fattening.

High carcass percentage

Researchers have recorded the following carcass percentages for Bali cattle: Aalfs (1934) 52%; Moran (1978) 55%–56%. Compared with other original Indonesian cattle, the carcass percentage of Bali cattle is the highest, with Ongole cattle at 45%, and Madura cattle at 47.8% (Tajib 1956).

Comparative disadvantages

Slow growing cattle

Bali cattle are known to be slow growing, but males especially continue to grow until they reach a considerable slaughter weight. The live weights of male and female Bali cattle, from birth to slaughter age, are presented in Table 4.

Growth of the female is slower than that of the male, and the older the female, the slower the growth. At 5.5 years of age, female growth stops. Weight gain between 2.5 years and 3 years was only 8.1 kg and between 5.5 and 6 years, only 1 kg.
Males grow fairly fast until weaned, grow slower for a period, then grow faster from 1–1.5 years on. Good quality bulls grow well until 4.5 years old and can achieve a high body weight.

Bali cattle given high quality feed produced an average daily gain (ADG) of only 660 grams over a period of 154 days, lower than for Grati and Ongole cattle, and buffalo, which produced a ADG of 900 grams, 750 grams and 730 grams respectively (Moran 1971).

**High calf mortality**

Darmadja (1980) reported average calf mortality up to 6 months of age (182 days) was 7.33% and Sumbung et al. (1977) reported calf mortality of Bali cattle in South Sulawesi was 7%.

The Bali Cattle Breeding Project recorded mortality rates of young cattle (205–550 days) at 3.95%, while the percentage on traditional farms was 6%.

Several observations on adult cattle mortality have been made both on Bali and outside Bali. The Bali Cattle Breeding Project has recorded adult Bali cattle mortality at 4%, while Sumbung et al. (1977) recorded adult mortality at 2.7% in South Sulawesi. The high mortality of adult Bali cattle is caused by Jembrana disease which is threatening the cattle industry in Indonesia. Other causes are bacterial diseases and toxic pesticides. The causes of adult mortality recorded by the Bali Cattle Breeding Project are shown in Table 5.

### Susceptibility to Jembrana and other specific diseases

Jembrana is the name given to a disease affecting Bali cattle which was first recognised in 1964 in the Jembrana district of Bali. The disease spread rapidly to surrounding districts and by August 1965 had occurred throughout Bali and an estimated 26,000 Bali cattle died out of a total population of 300,000. In the Jembrana district, the reported mortality was 19,000 out of a total of 31,000. Another outbreak of a similar disease occurred in Tabanan district of Bali in 1971 and 1972, with a mortality rate of 13%.

A more endemic condition of Bali cattle disease, currently known as Jembrana disease (JD) has been the topic of research since 1972.

Currently, JD or a Jembrana-like disease has been reported in three areas: in Bali, JD is now endemic; in Lampung Tengah, Rama Dewa disease is now endemic; and in Banyuwangi, East Java, the disease is known as Banyuwangi disease.

Malignant catarrhal fever (MCF) affects cattle and buffalo sporadically and with low incidence, but its case fatality rate is very high, up to 95%. MCF is caused by a virus. The first report of MCF in Indonesia was in 1894, affecting buffalo in Kediri (Partadiredga et al. 1988). Bali cattle are the most susceptible to MCF, followed by Madura cattle and buffalo, while Ongole and Friesian Holstein are relatively resistant (Peranginangin 1988).

### Table 4. Production and reproduction performance of Bali cattle.

<table>
<thead>
<tr>
<th>Items</th>
<th>Sulsel</th>
<th>NTT</th>
<th>NTB</th>
<th>Bali</th>
<th>P3 Bali*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Puberty age (month)</td>
<td>20</td>
<td>22</td>
<td>22</td>
<td>20</td>
<td>18</td>
</tr>
<tr>
<td>Av. weight at first mating (kg)</td>
<td>165</td>
<td>170</td>
<td>170</td>
<td>180</td>
<td>185</td>
</tr>
<tr>
<td>Calving percentage (%)</td>
<td>76</td>
<td>70</td>
<td>72</td>
<td>69</td>
<td>86</td>
</tr>
<tr>
<td>Calving interval (days)</td>
<td>475</td>
<td>520</td>
<td>510</td>
<td>530</td>
<td>430</td>
</tr>
<tr>
<td>Av. birth weight (kg)</td>
<td>12</td>
<td>12</td>
<td>13</td>
<td>16</td>
<td>18</td>
</tr>
<tr>
<td>Av. weaning weight (205 days) kg</td>
<td>70</td>
<td>75</td>
<td>72</td>
<td>86</td>
<td>94</td>
</tr>
<tr>
<td>Av. weight at 1 year (kg)</td>
<td>112.5</td>
<td>115</td>
<td>115.4</td>
<td>127.5</td>
<td>140</td>
</tr>
<tr>
<td>Av. weight at 2 years (kg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>— male</td>
<td>210</td>
<td>220</td>
<td>222</td>
<td>235</td>
<td>260</td>
</tr>
<tr>
<td>— female</td>
<td>170</td>
<td>180</td>
<td>182</td>
<td>200</td>
<td>225</td>
</tr>
<tr>
<td>Av. weight at 5 years kg</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>— male</td>
<td>350</td>
<td>355</td>
<td>300</td>
<td>395</td>
<td>484</td>
</tr>
<tr>
<td>— female</td>
<td>225</td>
<td>235</td>
<td>238.5</td>
<td>264</td>
<td>300</td>
</tr>
</tbody>
</table>

Source: Ismed Pane, 1980.

*P3 Bali = Bali Cattle Breeding Project.

NTT = Nusa Tenggara Timur province.

NTB = Nusa Tenggara Barat province.

Sulsel = Sulawesi Selatan
Table 5. The cause of adult mortality at Bali Cattle Breeding Project.

<table>
<thead>
<tr>
<th>Mortality causes</th>
<th>1978 ND %</th>
<th>1979 ND %</th>
<th>1980 ND%</th>
<th>1981 ND %</th>
<th>Average ND %</th>
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<tbody>
<tr>
<td>Accident</td>
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<td>1 1.85</td>
<td>3 4.47</td>
<td>6 2.90</td>
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<tr>
<td>Feed poisoning</td>
<td>2 6.25</td>
<td>2 3.70</td>
<td>2 2.78</td>
<td>9 4.48</td>
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</tr>
<tr>
<td>Pesticide poisoning</td>
<td>2 6.25</td>
<td>5 9.26</td>
<td>9 12.50</td>
<td>20 9.5</td>
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<td>Jembrana</td>
<td>17 53.13</td>
<td>27 20.00</td>
<td>33 45.83</td>
<td>94 46.77</td>
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</tr>
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<td>Coryza</td>
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<td>2 4.65</td>
<td>2 3.70</td>
<td>1 1.39</td>
<td>7 3.48</td>
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<td>Bacterial diseases</td>
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<td>5 11.63</td>
<td>10 13.89</td>
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</tr>
<tr>
<td>Internal parasites</td>
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<td>2 4.65</td>
<td>8 14.81</td>
<td>7 9.72</td>
<td>17 8.46</td>
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<td>4 7.40</td>
<td>6 8.33</td>
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<td>2 4.65</td>
<td>2 3.70</td>
<td>4 5.56</td>
<td>14 6.97</td>
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</tbody>
</table>

Source: Bali Cattle Breeding Project. ND = Number of deaths.

Another specific disease affecting Bali cattle is Bali Ziekte (BZ), which attacks sporadically and with clinical signs similar to photosensitivity caused by liver function disorder. Ressang (1984) suspected that a virus or poisonous plant was the causal agent. Sobari (1980) stated that Lantana camara is a poisonous plant which causes BZ. He proved that the clinical signs of BZ occurred in Bali cattle fed on 1 kg of dried Lantana camara leaves. Dharma et al. (1982) also said that the clinical signs and necropsy was indistinguishable between BZ and the disease caused by poisoning with Lantana camara.

Role of Bali Cattle in Livestock Development

Bali cattle as draught animals

Bali cattle grow faster than any other breed. In 1967, the proportion of Bali cattle was 11% of total cattle numbers and this has now increased to 26.8% (Table 6). In line with this, the role of Bali cattle as draught animals has become more important.

Bali cattle and meat production

According to a sampling carried out by the Directorate of Livestock Programming in 1991, the percentages of different cattle species slaughtered for meat were Bali cattle 38%, Peranakan (crossbred) Ongole (Bos indicus) 25%, Ongole 14%, Madura cattle 5%, local cattle 16% and dairy cattle 2%. Based on this study, the contribution of Bali cattle to beef production in Indonesia was the highest.

Table 6. The cattle population of different breeds in Indonesia (1984–1994) in '000s.

<table>
<thead>
<tr>
<th>Year</th>
<th>Ongole</th>
<th>PO</th>
<th>Bali</th>
<th>Madura</th>
<th>Others</th>
<th>Total</th>
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<tbody>
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<td>765</td>
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<td>1108</td>
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<td>1989</td>
<td>267</td>
<td>781</td>
<td>2753</td>
<td>1154</td>
<td>5139</td>
<td>10094</td>
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<td>1990</td>
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Distribution of Bali Cattle through GOI projects

The Government of Indonesia (GOI) has distributed Bali cattle throughout Indonesia through several projects.

(1) The Second Kalimantan Livestock Development Project (ADB Loan No. 706 — INO) has provided loans for the procurement of 58,000 female and 4900 male cattle, of which 21,000, or 33% were Bali cattle.

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Legend: Bali = Bali Cattle, BC = Brahman Cross, SC = Sahiwal Cross, SO = Sumba Ongole, PO = Peranakan (crossbred) Ongole, Madura = Madura Cattle.

Bali cattle related projects

There are two main projects related to the development of Bali cattle:

1. The Bali Cattle Breeding Project in Bali (Pulukan), Lampung, West Nusatenggara (Dompu) and South Sulawesi (Bone). The objective of this project, which began in 1978, was to improve the genetic potential of Bali cattle. It was conducted with New Zealand technical assistance (1978–1995).

2. The Bali Cattle Disease Investigation Unit (BCDIU) is part of the Disease Investigation Centre (DIC) at Denpasar, Bali, which was established a part of the Smallholder Cattle Development Project. The objectives of the study...
are to determine the basic characteristics of the disease and the aetiological agent involved, to develop a methodology for detection and diagnosis, and to determine the distribution and method of transmission.

Acknowledgment
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References