# Buffalo genetic resources in Latin America: constraints and treats on its sustainability

**Prof. Dr. WILLIAM G. VALE**
Professor of Animal Reproduction
Universidade Federal do Oeste do Pará
Campus Tapajós
68.035-110 Santarém, Pará – BRAZIL
wm.Vale@hotmail.com

## Abstract

This paper deals with hereditary diseases and/or birth defects diagnosed in buffaloes observed in Brazil and some Latin American countries. Related aspects of possible development and control of hereditary components are addressed. For some of the diseases described, the genetic origin has been definitively stated; however others are still only hypothesis which has yet not to be confirmed. It has been described some epidemiological, clinical and pathological inherited/hereditary diseases already observed, as albinism, achantolytic mechanobulous dermatosis, arthrogryposis, myotomy, hydronencephaly, chondrodysplasia and birth defects that do not have a cause yet proven as cheiloschisis e cheilognatoschisis, megaesophagus, heart defects (persistency of the ducts arteriosus), dermatoparexia, defects in the reproductive tract such as uterus unicorns, uterus partial segmental aplasia, gonadal hypoplasia (ovary and testis), cryptorquidism and other defects. It was observed that breeds most affected by genetic diseases nature are those that came from Asian Continent (Murrah, Jafarabadi and swamp type), probably as a result of existing inbreeding in herds due to ban the importation of buffalo breeds, semen and embryos from that continent. The diagnosis of some these diseases as albinism, arthrogrypose and myotomy which has seen in different parts of Brazil as an outcome of undesirable genes steadily disseminate in some breeds of buffaloes population. The accurately identification of these genes by molecular techniques associated to breeding of this species with the highest health, reproductive and livestock control can minimize the damage the damage caused in some buffalo herds. Then this paper intent and reiterate warns on the problem and identifies some of the challenges that need to be addressed in the future.
**INDEX TERMS:** Buffaloes, congenital defects, genetic/hereditary diseases.

## Buffalo introduction in Latin America

There are strong evidences that buffaloes were introduced in Latin America through Brazil. In fact it seems that the first animals landed in the fluvial-marine Marajo Island in the mouth of the Amazon River which took place in 1890. A ship with political refugees from French Guyana exchanged with local farmers buffaloes for cereals. Those buffaloes were swamp type and probably originated from French Indochina or Eastern Dutch Indies. However the Brazilian Buffalo Breeders Association considered 1895 as the official date of buffalo introduction in Brazil, when a flock of Mediterranean buffaloes arrived from Italy. Further importations were performed from India and Italy, not only to Brazil but also to other countries of Latin America.

## Inbreeding in some buffaloes herd has brought the occurrence of genetic diseases

In Brazil very few animals were imported from India. As a result the risk of reintroducing rinderpest, which had already occurred in 1921 (Riet-Correa 2007), the importation of animals from Asia was suspended in 1956. In 1962 this ban was broken and some breeders have brought few Murrah buffaloes from India (Miranda 1986). After that there was no official importation of genetic material from Asia. It was allowed to import from Europe only. In 1989 there were imported some Mediterranean's from Italy where, even today, there are semen import. Semen of Murrah and Jafarabadi breeds were imported from Bulgaria at the same time. This material was only released by the Ministry of Agriculture Livestock and Supply (MAPA) in 1991. For these reasons the Brazilian buffalo herd has a narrow genetic base (***founder element***) and consequently, inbreeding, especially in flocks of pure origin, where has been identified as the main factor appearance hereditary inherited diseases.

## What´s happen when inbreeding is used

Genetic disorders in humans and animals include illness caused by abnormalities in genes or

chromosomes, those abnormalities typically existing at birth. Disorders can be passed down from the parents' genes, or caused by *de novo* mutations to the DNA *loci* (Zhao, 2012). Thus, selection in animal breeding systems uses genetic diversity/variation to improve the population by selecting superior animals for desirable traits. The more genetic variation observed in a population, the bigger the potential genetic gain possible in each generation. Conversely, decreased genetic variation increases the similarities of the population. inbreeding reduces the amount of genetic diversity in a population. Thus, explanation of inbreeding in animal populations relies on a few basic genetic principles (Lesley 1970). Genetic information is stored in chromosomes are made up of *DNA*. Genes are sections of DNA and occur in pairs. A particular gene will occur at a particular site (*locus*, plural is *loci*) in the DNA of a particular chromosome. The inbreeding coefficient (I.C.), (as proposed by Sewell Wright in 1922) is the probability that two alleles at a randomly chosen locus are *identical by descent*. Note that alleles may be identical for other reasons, but the IC is just looking at the mathematical probability that the alleles have come from a common ancestor. Example of calculating an IC for an animal that has a common great grandparent:

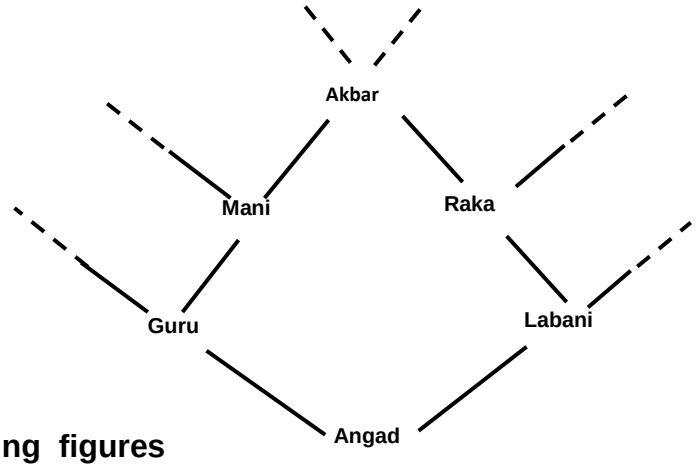


**Figure 1.**Calculating inbreeding coefficient (I.C.), according Tomar(2014).**Angad** is inbred because **Akbar** is a common ancestor.There are five ancestors in the inbreeding loop:**Mani – Guru–Akbar – Raka – Labani** then **Angad** inbreeding coefficient = $(1/2)^5$ = 1/32 = 3.1%.**(If any of Akbar's ancestors were themselves inbred, then Angad's inbreeding coefficient would be higher)**

**Inbreeding figures**

The IC is expressed as a percentage value. A low IC means a low level of inbreeding p. ex. 3%. In most beef cattle breed societies, the vast majority of animals have an IC of less than 10%, inbreeding coefficients over 30% are unusual, and over 40% are rare, Table 1.

**Table1. Inbreeding coefficients for various inbred relationships.**

| Type of crossing | Per cent |
|---|---|
| •Father x daughter | 25,00 |
| •Full brothers | 25,00 |
| •Half brothers | 12,50 |
| •Father x grand daughter | 12,50 |
| •Son x grand daughter of the same father | 6,25 |
| •Grandson x grand daughter of the same father | 3,13 |

*****Minimum value, will be higher if ancestors are themselves inbred

Then typical IC for various relationships are given in Table 1. The IC will be higher if the ancestors in the pedigree are also inbred themselves. Not surprisingly, smaller populations tend to have proportionally more animals with higher inbreeding coefficients than larger populations - simply because there are fewer candidate animals to select from (Figure 1).

**Are some breeds genetically 'healthier' than others?**

Inbreeding, such as is used to fix traits within pedigree breeds, will inevitably increase the risk on inherited defects coming to light. Although inherited diseases can occur in both non-pedigree

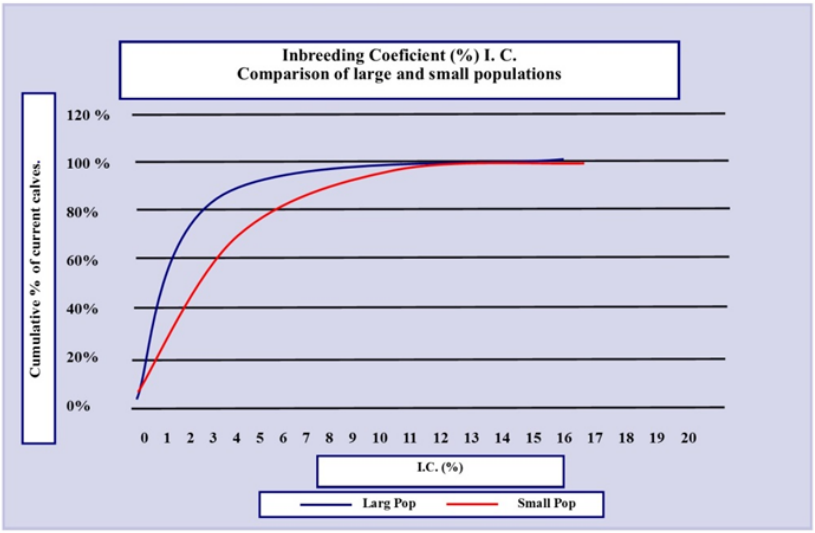


**Figure 1.** Inbreeding coefficients for various inbred relationships

and pedigree animals, they are generally more likely to occur in pedigree animals than in outbred domestic animals. Some pedigree breeds are much more inbred than others and so, in theory, would be at greater risk of having inherited diseases present. However it is difficult or impossible to say that some breeds are genetically 'healthier' than others. In some breeds of cattle, pig, dog and cat, a large number of different inherited diseases have been identified, but this usually reflects more widespread surveillance and testing within these species rather than necessarily a higher frequency of inherited diseases (Basrur, 1988; Zhao, 2012; Damé, 2013).

**Can inherited diseases be controlled?**

Where there is a relatively simple mode of inheritance, and where there is a DNA test widely available, controlling an inherited disease may be relatively straightforward. Currently, it is possible to use cytogenetic techniques (cosmid probes, alfa DNA probes satellite) or molecular techniques for genome determination like DNA genome sequencing (southern blotting,PCR-transcriptase reverse) in buffaloes, aiming to studies on productivity parameters, fertility as well as the hereditary nature of diseases. With the application of these techniques it can be identify economic traits for production and hereditary diseases and then mitigate the presence of the same deleterious genes in herds (Ohashi et al 1995, Dame et al 2012, Damé 2013, RolimFilho et al. 2013).

**Results and discussion**

Very few animals were introduced in Brazil for the formation of the different breeds with exception of the Mediterranean type. For the Murrah breed it has been noticed that only eight females and four males gave origin to the **POI** (***Pure of Origin Imported***) herd although in the recent years semen from Bulgaria and India has been introduced. It seems that inbreeding can be the main cause of the appearing of such abnormalities. However it has further has been proved experimentally in other species that many forms of congenital malformation, which appear spontaneously can be produced by different teratogenic agents (Basrur, 1988). Hence, this does not mean that those defects are nongenetic when they appear spontaneously. Not in the least it should be considered that spontaneous occurrence of defects is usually due to homozygosity for recessive genes, mainly in the herds where new germoplasm were not introduced. The same forms of diseases described in Table 2 have also been described in other domestic species (Lagerlof 1938; Spriggs 1946; Bonsma, Venter 1973; Leipold et al 1972,1983; Basrur 1988; Zhao, 2012; Ghalop et al .2014).The most interesting is that some of these diseases have the same origin genetic and pathophysiological features are just exactly the same as in mouse, primates and human beings, like albinism and polycystic kidney disease (PKD) in Persian cats and related breeds, monkey and mouse (Bosje et al. 1998; Carden et al

1988; Plesker & Schulze, 2006; Grattham 2008).In the past, up to 50% or more of Persian cats may have been affected by this disease, which will frequently result in chronic kidney disease and premature death of affected cats actually identified through modern molecular diagnosis for hereditary diseases. The first reports on the pathology hereditary in the domestic animals were published by Fincher; Williams, (1926), Lagerlöf (1939), Eriksson, (1943), Spriggs, 1946; Koch (1952), Götze, (1950;1952), Flechsig (1952), Lagerlöf & Settergren, (1952); Nordlund (1956).

**Table 2.**Occurrence of hereditary/congenital defects in buffaloes in Brazil.

| Typeofabnormality | Author |
|---|---|
| 1.Abnormal formation of scrotal sac | Vale (2005) |
| 2.Albinism – partialand total | Vale (2005);Damé et al (2013) |
| 3.Aglosia | Vale et al (1976) |
| 4.Amputee limbs | Vale (2005) |
| 5.Arrest of mesonephric (Wolff) ducts | Vale et al (1981); Vale et al (2002) |
| 6.Arrest of paramesonephric (Müllerian) ducts | Vale et al (1979;1981;1988) |
| 7.Arthrogrypose | Vale (2005); Damé et al (2013) |
| 8.Atresia ani | Laú (1995); Vale (2005) |
| 9.Calf ataxia | Barbosa et al (2002a-b) |
| 10.Cheilognatoschisis | Vale et al (1976); Barbosa et al. (2002a-b) |
| 11.Chromosome X fragile sites | Mota et al (2004) |
| 12.Chondrodysplasia | Barbosa et al. (2002a-b) |
| 13.Congenital muscular hyperplasia | Barbosa et al (2002a-b); Vale (2005) |
| 14.Cryptorchidism | Vale & Ohashi (1994) |
| 15.Dag defekt | Vale & Ribeiro (2002) |
| 16.Dermatose acantolítica mecanobolhosa | Reit-Correa et al (1994); Damé et al (2013) |
| 17.Embryonic mortality/Repeatbreeders | Vale et al (1989) |
| 18.Epitheliogenesis imperfecta | Láu (1999); Vale (2005) |
| 19.Epydidimal disfunction | Ohashi et al (1986) |
| 20.Gonadal aplasia and hypoplasia – male and female | Vale et al (1978); Ohashi et al (1995) |
| 21.Harelip andcleftpalate | Vale et al (1976) |
| 22.Hydronencephalia | Schild et al (2011); Damé et al (2013) |
| 23.Inguinal and umbilical hernias | Vale et al (1980); Láu (1999) |
| 24.Ovarian cysts | Ribeiro &Vale (1988); Vale & Ohashi (1994) |
| 25.Outward bowed pasterns | Vale (2005) |
| 26.Overshot jaw | Vale (2005) |
| 27.Patent ductus arteriosus | Ecco et al (2008) |
| 28.Renal hypoplasia | Pereira & Vale (1994) |
| 29.Spastic paresis | Láu (1999); Vale (2005) |
| 30.Tendon contracture | Láu (1999); Vale (2005) |
| 31.Twisted testis | Vale (2005) |
| 32.Undershot jaw | Vale (2005) |
| 33.Wry face | Vale (2005) |

Those articles described different forms of genetic abnormal conditions affecting different systems and organs like nervous, eyes, blood, locomotor and reproductive in different domestic species (Ladds, 1992).Further on an excellent publication was done by Bonsma& Venter(1973) dealing with the hereditary defects ofcattle. Of course, buffaloes are not free of such problems. There are in the international literature many reports on the occurrence of different forms of pathology hereditary in buffalo species that also occur in Brazil (Vale et al. 1976, 1979; 1981; 1991; Láu et al, 1991; Pereira; Vale, 1994; Iannuzzi et al., 2001a-b; Barbosa et al. 2001; Correale & Consalvo, 2003; Mota et al. 2004). Götze (1949;1952) stressed the importance of the hereditary health and control of the sire sused in artificial insemination programs. Survey on known or presumptive hereditary defects and undesirable hereditary dispositions in buffaloes raised in Latin America should be established specially for males. It isimportantto look at least for general deformities on the body shape, gait, libido, erection, copulation, as well as the absence of hereditary diseases, such as testis hypoplasia, cryptorchidism, epididymal dysfunction, spermiogenesis disturbance – semen pathology of hereditary origin like sperm dag defect (Vale & Ribeiro 2009).

**Conclusion and recommendations**

Within the buffalo production systems in Brazil, inbreeding has been considered the main factor for the onset of congenital diseases in this species, especially in the Murrah breed. Due to high rates of inbreeding in some herds, the genetic background of most inherited diseases diagnosed in buffalo in Brazil is due to autosomal recessive genes. Furthermore, among the hereditary disturbs one of the major disease, albinism is a consequence of a nonsense mutation in the tyrosinase gene, with the substitution of a single base at nucleotide 1431 (G to A) makes the stop codon for tryptophan 477.Besides albino buffaloes have an inadequate adaptation to the tropical climate, are also predisposed to the development of skin pathologies such as malignant melanoma. A identification of defective genes cause of albinism ocular-cutaneous and hereditary myotomy and the elimination of carriers of reproduction animals allows control of these diseases. Establish progeny test for buffalo bulls semen donor.

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