Ethics and Animal Breeding

By Eric Hoffman

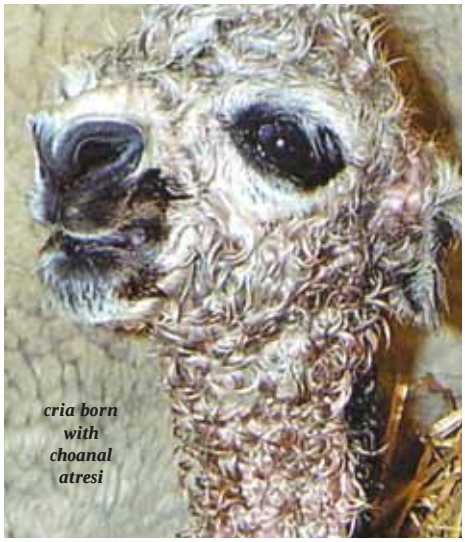
Have you ever known a gambler who cupped their sweaty palms around a pair of dice, uttered a prayer and let them roll? You may have never thought of it this way, but perhaps you've been that gambler, but instead of dice you banked on the union of two alpacas or two llamas to produce healthy offspring.

Sooner or later breeders of any kind of animal will learn that their roll of the dice can bring joy or profound sadness. The sales pitch for both llamas and alpacas is, "they are generally hardy animals and easier to take care of than many other kinds of livestock." This can be entirely true in certain herds and certain settings. But there is an Achilles heel to this tag line involving the identification and proliferation of congenital defects and the ambiguity around strategies to prevent them. Further confusing the issue is the lack of ethical standards for trading animals that may be carrying genetic problems.

Conversations with experienced veterinarians, case histories of the South American herds that created North America foundation stock, the marketing dominance of a small group of interrelated studs in a closed gene pool, the promotion of inbreeding and line breeding by marketers, combined with no industry-wide systems to identify carriers of genetic problems adds up to a familiar animal breeding scenario. Being part of "new age breeding strategies" may sound exciting, especially to people new to animal breeding, but rest assured the forces at work in the camelid business have been tried before. We only have to look as far as dog breeding and to other forms of livestock to find out that there are numerous examples of the best laid plans resulting in both positive outcomes and genetic disasters that plague some breeds for decades. If what I'm driving at isn't clear read The Dog and its Genome by Cold Spring Harbor Laboratory Press and you'll see parallels that should concern you.

Domesticating animals and selectively breeding them to serve mankind's needs and pleasures has gone on for centuries. The array of different kinds of horses, sheep, cows, dogs, cats and other animals attests to this effort. Breeds of sheep and goats have disappeared due to genetic problems that made it too difficult to continue. Often geneticists are called in to "save" breeds from the genetic problems rampant in a population or to rehabilitate a breed that is depleted of genetic diversity. Often the difference in the outcome for an entire breed can hinge on the general education of owners on how to best avoid creating a "prevalence" of genetic problems in a population of animals and the overuse of studs whose virtues are eventually overshadowed by an unintended genetic impact.

If you've been in the camelid business for more than a few years you've heard the stories. People buy some animals for top dollars because of their bloodlines and show ribbons. The proud new owners ready their paddocks, top off their barn with tons of hay and wait for their spring crias. Unfortunately, the first cria is born with choanal atresia, an abnormal bone growth over the nasal passages which makes it impossible to breath and nurse at the same time. The cria has to be destroyed. The breeder who sold the dam who birthed the afflicted


cria born with choanal atresi


alpaca with scoliosis


example of undershot jaw

cria offers to replace the cria but not the mother. The sire keeps on breeding too. When pressed about the wisdom of keeping animals who created an animal with a lethal congenital defect in a gene pool, the breeder's response may be, "It isn't known if it's genetic or not." This kind of answer to this kind of problem, underscores the perplexity in the mix of genetics and ethics.

It would help us all if genetic issues could be expressed in black and white, either the defect is genetic or it is not. However, with camelids, often sleuthing the exact causes of an undesirable genetic outcome carries some uncertainty for the simple reason that the kinds of exacting genetic studies that pinpoint carrier genes don't exist – though progress is slowly being made on some fronts.

In the large operations in South America, like Rural Alianza, obvious genetic defects are dealt with by culling, which assigns the animal to the meat market and thus ends its genetic input into the population. Still this is not an ideal solution. Often the carrier animals are not identified and are allowed to keep breeding, if they possess other desired characteristics such as a high fleece weight, fine fiber, and are reliable breeders.

Outside of South America, Australia is the only country with an alpaca meat market. In North America and Europe this end use is repugnant to many owners who view their animals as pets. Instead, there is ambiguity about culling. Some breeders neuter animals that have a history of creating crias with defects while others cull by selling suspect animals, allowing them to reproduce and continue to impact the gene pool. There are also prolific studs, who are known producers of numerous defects, that are allowed to keep producing offspring. The irony is that culling from the gene pool is probably more likely in South America than North America where DNA based registries are in place that could track defects accurately.

Understanding Genetic Defects.

Educating yourself about congenital defects is the first line of defense against them. First, knowing the entire array defects (probable genetic as well as other types) out there and being able to identify them is paramount. Knowing the basics about genetics helps too.

Simply put, the code carriers of inheritance are on genes which are carried by chromosomes. Sperm and egg carry the genetic material and combine to make a new individual. In the shuffle of genes sometimes-recessive genes, not known to exist in the parents, pair up and saddle the newborn with a defect that curtails its ability to live a normal life. In other instances harmful genes can be eliminated in the shuffle that occurs. In most animals genes causing problems may produce a particular trait by a simple pairing of two genes (monogenic). Less commonly, the process ending in a defect may be multi-factorial (involving complex combinations of genes).

Genetic flaws occur in all species. Genes in llamas and alpacas, like those of other species, sometimes mutate. However the frequency of change caused by mutation in a wide range of species is much rarer than those caused by the pairing of genes from parents, meaning that a defect that is probably genetic in origin is more likely to be transmitted through inheritance than mutation.

Nature's Way of Sorting Genes

In the wild animals deleterious genes, that lessen an offspring's ability to survive, provide a natural culling mechanism. In the struggle for "the survival of the fittest" which is a constant in nature, animals operating under a disadvantage are often the first to perish. In large wild populations lethal recessive genes are often swallowed up in vast gene pools that are so diverse that a pairing of lethal genes becomes highly improbable. The likelihood that two healthy parents carrying a rare lethal recessive gene will mate is small. Closed or shrinking gene pools that become saturated with harmful genes will likely lead to the loss of reproductive vitality and extinction.

Our Responsibility for Domestic Animals

With domestic animals the rules change. Often survival of the fittest is replaced by survival of animals with a certain appearance, which may or may not have allegiance to nature's principle of survival of the fittest. With domestic animals there are many documented examples of breeding disasters that occur when a certain appearance became the primary goal. Pedigreed dogs, for example, have 500 known diseases from the simple pairing of undesirable recessive genes and they have 1100 known genetic diseases. Pedigreed dogs offer classic examples of deliberately narrowing a gene pool to get a particular

look while ignoring accompanying traits that cause pain and suffering to the breed. For example, 75% of registered boxers in Europe have hip dysplasia, while other breeds are saddled with the prevalence of cataracts, blood disorders and immune deficiencies. In the boxer's case, a particular stud who won many ribbons and dominated breeding was the culprit. Unfortunately, his impressive appearance masked his downside and allowed him to have great influence and do much harm to the breed. Dalmatians have a high percentage of deafness, associated with the spots that define the breed's appearance. One of the astounding features of the dog genome is discovering how many different breeds ended up with a defining appearance, achieved at the cost of crippling genetic problems.

In comparison, large wild populations of wolves, the progenitor of all dog breeds, have much lower instances of problems. To some extent dog breeders have an excuse. Most of the dog breeds were created before genetic testing was a science. Dog breed associations now find themselves trying to fix or otherwise sweep away the problems caused in creating phenotype standards for their breeds that ignored soundness and health. The more responsible breed associations have spent large sums of money, and energy, to institute over fifty carrier-identifying tests to clean-up breeds, by identifying carriers with an over abundance of genetic problems. With camelids identifying genetic problems, and sleuthing them, is still in its infancy, while breeding practices similar to how dog breeds were created are underway. This should be reason for concern.

Reason for Suspicion and Concern in Domestic Camelids

Murray Fowler DVM, a former department chair at the UC Davis School of Veterinary Medicine and an early pioneer in camelid medicine, reported at the 1984 ILA Conference in Santa Cruz, California that twenty-nine congenital defects had been identified in South American camelids. Fowler was careful to call them congenital defects (present at birth) instead of genetic defects because no genetic studies existed that proved the cause of a particular defect was genetic, even though in many cases faulty genetics were suspected.

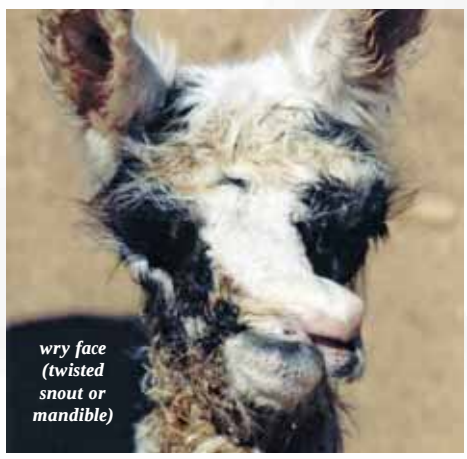
Multiple Sources for Defects Muddy the Waters

When it comes to defects, genetics is just one of several possibilities which can also include dietary imbalances and environmental causes such as drugs, chemicals that are either administered or present in the environment, as well as other agents such as viruses. All of these agents are called *teratogens* or are t*eratogenic*, and they result in defects that arise during fetal development. These defects are much better documented in cattle, sheep and horses than they are in South American camelids. And, because there are reasons for defects other than genetics, it is easy to explain any defect as being caused by unknown agents. But, explaining away all defects as not genetic is neither good for the breed, wise, or truthful. There is enough known to indicate a number of the most serious defects are consistent with how genetic diseases present themselves and there are case histories indicating genetic components. For example, LaRue Johnson DVM, Phd, specializing in camelids, believes more than forty-five defects are genetically transmitted.

Whatever the cause, the list of congenital defects keeps growing and, so far, all defects found in alpacas are also found in llamas. Well-known livestock geneticist at Virginia Tech, D. Phillip Sponenberg DVM, PhD wrote Chapter 24: Basic Principles and Implications for Feeding" for *the Complete Alpaca Book, Revised Second Edition (2006)*. In his chapter Dr. Sponenberg created a table (24.1) listing sixty-four known camelid defects, an increase of thirty-five defects from the presentation done by Dr. Fowler in 1984. Sponenberg offers comment on each defect based on research done on camelids and other species. Though the comments may not be definitive they are useful in steering the reader to informed decisions about particular defects. For example, for choanal atresia he points out, "Evidence of genetic control is mixed, so if genetic, it is not a simple single gene trait." This conclusion is also reached in early work done by researchers Brad Smith DVM, PhD and Karen Timm DVM PhD. Timm and Smith have since added to the genetic likelihood by carefully documenting a female llama that gave birth to crias with partial or full choanal atresias from five unrelated males. When the birth-mother died her necropsy revealed that she had a partial choanal atresia. A current Alpaca Research Foundation study underway at the University of Minnesota is attempting to determine the actual location of the genetic code that creates this lethal defect. In the case of choanal atresia the progress has been slow but steady with unrelated research efforts pointing in the same general direction. With most of the other defects no such research is underway, though in many cases there appears to be a high incidence of heritability (crooked tails, defective ears, etc.).

With some defects the experts appear to have conflicting views. Dr. Sponenberg points out that in some livestock wry face (twisted snout or mandible) is associated with environmental causes while Dr. LaRue Johnson believes the cause is primarily genetic in camelids.

At the 2009 International Camelid Health Conference held an Oregon State University LaRue Johnson DVM, PhD, a pioneer in camelid medicine in North America, presented a paper entitled "Camelid Congenital/Genetic Defects." documenting seventy-four congenital defects. A seventy-fifth defect (fused ears) was added during the presentation. Dr. Johnson went on to rate each defect in the order of likelihood of occurrence (Common, Occasional and Rare). He also offered his opinion as to the cause of the defect citing the late livestock geneticist Dr. Horst Leipold, formerly at the University of Kansas, Professor Emeritus Murray Fowler, from UC Davis, unnamed colleagues and his own experiences over



wry face (twisted snout or mandible)

many years as head of the camelid veterinary medicine program at Colorado State University. Dr. Johnson surmised that forty-five of the seventy-four defects he listed were genetic in nature. Though Dr. Johnson believes genetics cause most defects, he also lists many defects and their causes as "unknown." In time, providing there is funding for further research, the actual genes involved in the transmission of genetic problems may become known, as well as the accuracy of Dr. Johnson's hunches.

Telltale Signs

What kind of abnormalities are most likely genetic? The words of Dr. JoAnn Paul-Murphy, formerly of UC Davis, in a l988 interview on the subject of camelid genetics still make sense. " Often you look for abnormalities that are being reported across the nation that have no common suspected cause other than transmission through genes. For example, choanal atresia and arthogyrposis (abnormal attachment and formation of limbs) are reported in Florida, California, New York and Colorado in animals with different diets and no common deleterious environmental factors (toxins, extreme heat and cold etc.). These abnormalities are more likely to be genetic."

Do Llamas and Alpacas Have an Atypical Rate of Defects?

Dr. LaRue Johnson: "Hard data is hard to come by, but from my experience I'd say llamas and alpacas show more problems than other livestock. I had never heard of choanal atresia and certain digit problems until I became immersed in camelids." Pat Long DVM, a well-known camelid vet from Corvallis, Oregon: "Llamas and alpacas have a seemingly high percentage of congenital defects (defects present at birth) compared to other animal species. Some of these we identify at birth like choanal atresia or atresia ani and others we don't identify until later in life. Some defects, such as female reproductive disorders like segmental aplasia of the uterus, won't be detected until the animal is mature and ready for breeding. In my opinion, the high rate of congenital abnormalities is the biggest health concern for alpacas and llamas." Brad Smith DVM, Phd, now retired, ran the camelid medicine program at Oregon State University through the 90s. "There are significant problems in camelids with what appear to be heritable defects. Breeders need to realize that culling doesn't mean selling to the next guy down the road. Culling means destroying the carrier or rendering it incapable of reproduction."

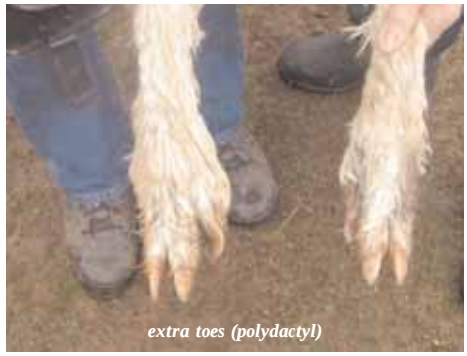
Case Histories of Zoo Populations & South American herds

In a 1988 Llama Life article, genetic records of llama herds from several zoos across the United States were obtained to see what was going on in these highly restricted gene pools. This was pertinent at the time because much of the foundation stock for the early privately owned llama herds in North America was from zoo stock. Six out of seven zoos had high incidences of stillborns, blindness, limb and facial deformities. The zoo with the worst log had twenty-six stillborn or neonatal deaths from forty-six births spread over ten years. In one zoo, twelve births from a single male ended in premature deaths, with "palate problems" being noted over and over again. The suspect male was sold to another zoo when a replacement male was purchased. It is worth noting that in recent years most zoos have attempted to improve their genetic diversity, but prior to this zoo stock was often notoriously inbred. These records illustrate what happened to reproductive viability in these highly inbred populations of camelids.

What Went on With the South American Herds

In South America herd records are hard to come by. Tracking lineages is rarely done. However culling to the meat market is done continuously. Most alpacas imported into the US, Canada, England, Australia and Germany were stringently screened using standardized objective criteria which was specifically aimed at eliminating congenital defects. This eliminated animals with overt defects but could not identify animals carrying defects in their genes.

In an interview with the late Don Julio Barreda in Peru in the 1990s, he recounted how he deliberately inbred his animals to achieve consistency. He



extra toes (polydactyl)

recounted using the same stud on three generations, or more, of females. The failures of these unions went to slaughter. In a study, Julio Sumar DMV, a Peruvian researcher and alpaca judge in North America, found high incidences of internal abnormalities in freshly slaughtered camelids at an abattoir he inspected. In the many herds visited by the author, during many years of travel and work in South America, he saw no attempt to curtail inbreeding and often a defect like extra toes (polydactyl) was seen as a sign of good luck! The author saw one herd in which at least half the herd had a condition called gopher ears (an abnormally short ear). However, animals that were slow to reproduce or had poor quality fleeces or low fleece weights were often culled.

The big picture is that breeding practices varied greatly from one compensino to the next. There were also a series of severe disruptions in the Andes causing genetic bottlenecks that impacted genetic diversity dating back to 1532. Two of the most recent ones involved radical property reforms in the l960s that resulted in thousands of animals being sent to slaughter and the move of the large fiber mills to paying more for white and fawn fleeces, which resulted in severe depletion of the darker colors. It is easy to conclude husbandry practices in recent times were not favorable for tracking and decreasing abnormalities, even though culling was occurring. In North America, and other parts of the world, tracking suspected genetic problems is entirely possible and was one of the reasons a DNA based registry was created. The will to do so has not materialized in most countries, with the exception of Germany. Australia has a Stud Certification Program that disqualifies studs with overt defects based on the aforementioned screening standards.

Russian Roulette via Inbreeding

In any species inbreeding is always hazardous because it exacerbates a gene pool and tends to allow the recessive lethal genetic material to play a more active part in ensuing generations.

In the l980s inbreeding was condemned by llama and alpaca breeders. Inbreeding is breeding to immediate relatives (daughters, sisters, mothers). Currently alpaca breeders seem willing to accept the risks inherent in decreasing the genetic diversity in national herds in order to get a desired look. The reward of creating the "correct" outward appearance as quickly as possible is used as a justification by advocates of inbreeding and line breeding. But, inbreeding also increases the risk of lethal expressions. In theory, if two carriers of a recessive lethal gene breed, the outcome for four offspring is: one cria fatally afflicted, two carry the trait but are healthy, and only one is free of it – which adds up to 75% of the offspring either carrying or expressing the gene. The more the inbreeding the narrower the combinations until everything has been expressed – in the worst scenario like the aforementioned zoos herds.

What About Line Breeding?

This has become increasingly popular with North American alpaca breeders. It is often said line breeding accentuates a certain characteristic. The definition of line breeding varies but in general it is seen as breeding to more distant relatives than mothers, fathers, daughters and brothers. Cousins and nieces are usually fair game. Those embracing this philosophy can be successful if, in their push to fix some traits in their animals, they don't emphasize one or two characteristics while ignoring soundness. Breeding strategies of all kinds carry some risk of unknowns coming to the surface. Usually there are greater risks when the gene pool is narrowed.

Line breeding restricts the gene pool. Dominance of one or two studs or their bloodlines further restricts it. At last check three alpaca studs in the US are directly related to 10% of the entire national herd. Does anyone know what defects, if any, these studs have produced and if they are contributing or detracting from the health of the gene pool?

It should be mentioned that what some breeders believe is line breeding may actually be inbreeding. With some imported populations the DNA markers utilized to differentiate between individuals were so similar, that distinguishing between individuals was sometimes challenging. The herd was more like identical twins than an assortment of unrelated individuals. Breeders, often unaware of the highly inbred aspect of some of the original imported stock, made breeding decisions that assumed each registered animal was unrelated to the next one, when in fact they may have been brother and sister or so highly inbred they were a virtual clone of many other animals.

A decade ago I was working on a story for another publication when I interviewed Dr. Oliver Ryder, a geneticist for San Diego Zoo's highly touted Center for Reproduction of Endangered Species. He had this to say, "It's not at all unusual to find genetic problems in closed populations. Island-bound human populations and the Amish community in the mainland US are good examples."

In the programs Dr. Ryder has worked on he sees the path to salvation through record keeping that involves not just the animal's appearance (or end product) but its overall health. "The only way to sort out genetic problems is through keeping a good pedigree. Incidences of anomalies must be recorded in a comprehensive and uniform fashion over the entire population. If only a portion of the population is covered, or there is missing information, a bias will occur and invalidate your efforts. It's very important to be accurate and comprehensive as possible. Once you develop your statistics accurately, predictions can be made in the outcome of mating particular animals." Dr. Ryder is motivated by success in breeding animals without defects.

However, this can be touchy territory. When a Cornell geneticist pointed out a particular breed of dog carried a particular defect he was threatened with a lawsuit for "defaming the breed." The geneticist learned that protecting an income stream was more important to some breeders than cleaning up a genetic problem that was affecting the entire breed.

Where do the Words "ETHICS" and "MORALS" Fit into the Process of Making Breeding Decisions?

Ethics are the system of moral principles for governing the appropriate conduct for an individual or group. Morals relates to issues of right and wrong and how individuals should behave. Morals are based on a person's conscience and their sense of what is right and what is wrong, rather than on what the law says should be done.

Are there appropriate "rules of conduct" for divulging the occurrence of congenital defects and identifying the animals involved ? In the process of selling animals should all past defects associated with the animal be divulged? Should there be methods of reporting defects so scientists can evaluate the frequency of certain defects to prioritize which ones deserve the most attention? Since there are different thresholds for determining "right" and "wrong" amongst camelid breeders, should breed associations or registries draft guidelines for reporting defects? Should there be a national or international strategy to decrease defects?

In the heyday of the North American llama business there was a push to use the registry to unmask lineages carrying harmful defects. This effort failed for a number of reasons, some of which involved breeders not wanting to be exposed to public scrutiny.

What action should a breeder take when a stud they have used forty times without a single defect being produced, suddenly produces a cria with a defect that may be genetic from a dam who was bred for the first time? Should the owner quit using the stud? What about the dam?

If the scenario involved a stud used for the first time to breed twenty females and five of the cria were born with extra toes and fused ears the answer to the ethical question on what to do would be much clearer for most people – castrate the male and monitor the offspring from the crias who were born without overt signs of the disease. What about the females producing the defects?

There are other unanswered questions muddying the ethical dilemmas facing breeders before a parental pair is condemned. Are any of the defects sex-linked i.e., only involving one parent? In simple monogenic pairing both parents contribute but what about multi-factorial situations involving an array of genes. 'Which defects are simple recessive pairing and which are multi-factorial?

So, What's the Ethical Thing to Do?

So what can we do now, as defects are being classified, yet determining the genetic mechanism causing them may be a ways off? Here are some suggestions:

1 Learn what the defects are and how to identify them.
2 Learn if a defect is most likely genetic, based on the criteria used by veterinarians and scientists.
3 Disclose all defects in a lineage to a potential buyer who can then decide their importance. (This one may seem naive in a market where many breeders will not disclose defects in a lineage for fear of losing a sale, and thus gain an advantage of the honest seller who makes a full disclosure.)
4. Don't declare a defect as not genetic when you don't know, and rely on science not hearsay to make decisions.
5 Urge national organizations to devise methods of reporting congenital defects to determine frequency and encourage anonymous participation in heritability studies.
6 Consider the wisdom of sacrificing genetic diversity for a fast track to particular look, especially when operating in a closed gene pool.
7 Have fun with your animals and think of breeding pairs that put the emphasis on trouble free offspring that live long, healthy, reproductive lives.

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About the Author

Eric Hoffman is the primary author of the second revised edition The Complete Alpaca Book, (Bonny Doon Press, 2006). He is one of the authors of The Alpaca Evaluation: A Guide for Owners and Breeders (book and dvd set) (Bonny Doon Press, 2009)He wrote the first scientifically based alpaca registry (today known as ARI) in the world and is the author of hundreds of articles on all four species of camelids. His articles have appeared in International Wildlife, Animals, Pacific Discovery, California Living, Wildlife Conservation and many other publications. His speaking engagements on camelids have taken him to many places including Australia, New Zealand, Sweden, Switzerland, Peru, Germany and England in recent times.

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