Genetics for Alpaca Breeders
(To appear in the forthcoming AOBA "*Alpaca Breeder's Handbook*")
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The Basics
Humans and alpacas share many things in common, along with the rest of the animal kingdom, including how they pass their genes on to the next generation. Humans have over 20,000 genes spread across 23 pairs of chromosome and some 3 billion base pairs of DNA. Alpacas likely have between 15,000 and 20,000 genes (just a guess at this point) spread across 37 pairs of chromosomes and unknown billions of base pairs of DNA. The first complete Alpaca genome was sequenced in 2008, so many of these statistics will shortly become known. Among these billions of base pairs of DNA are portions of DNA that code for proteins. These protein-coding regions of DNA are called genes. Proteins are what we, and all animal, plant and bacterial life on earth, are made of. Proteins like melanin help give color to skin and hair. Proteins such as insulin help control the sugar levels in our blood. Hemoglobin is a protein that carries oxygen to our cells and carbon dioxide back to our lungs. All of our tissues are made of proteins. All of our proteins are encoded for by the order of the base pairs of our DNA. Change a single letter in our DNA chains and the protein encoded for by that DNA may change or lose its function or appearance. Mutations in the gene for hemoglobin can cause sickle cell anemia or in the genes that control the cell cycle can cause cancers. Mutations are just random errors that occur when the body tries to copy its DNA (called replication) during cell division (called mitosis), or during the construction of gametes (called meiosis) like sperm and eggs. Mutations can also be caused by the environment, and if they are not repaired properly by the body's DNA repair systems, become permanent in the cells that had the mutation, and all the cells that are descended from those cells after cell division. Environmental causes of mutation can include chemical mutagens as well as radiation. This is why sun-tanning is not such a good idea, and why we slather our kids with SPF 45 sun-block lotion, and why many tanning salons make you sign a waiver form before letting you mutate yourself. Only the mutations that happen to the cells that will become sperm or eggs get passed on to the next generation. All the other (somatic cell) mutations do not get passed on, but can still cause disease to the bearer of the mutations.

We all know that our kids get half their genes from each parent. The process of meiosis leads to the production of gametes: sperm (in males) or eggs (in females). Sperm and eggs only have one copy of each pair of chromosomes. In humans, this means each sperm or egg has 23 chromosomes, in alpacas each sperm or egg has 37 chromosomes. When the sperm and egg unite during fertilization, the fertilized egg (or zygote) then has two copies of each chromosome, one copy from each parent (46 for humans, 74 for all camelids). Because each parent has 74 copies, and can only put 37 in each gamete, there are 2^{37} (over 13 billion) possible different combinations of the 37 maternal and paternal chromosome copies that can be made for each sperm or egg. This is not even accounting for the fact that recombination can scramble or mix the maternal and paternal copies to create mosaic chromosomes that contain parts of both parents mixed together. It should not be surprising therefore that full siblings rarely look alike. Indeed, it is amazing that siblings ever look alike given these odds.

When an individual is born that has two different versions of a gene (the different versions of a gene are called alleles), we say that individual is a heterozygote. An individual that gets the same version of a gene from both parents is called a homozygote for that gene. Not all alleles of genes are expressed equally. For some alleles, having one copy is enough to express the trait encoded by the allele. We say these alleles are dominant alleles. For other alleles, both copies of the trait must be the same for the trait to be expressed. We call these alleles recessive alleles. Dominant alleles mask the presence of recessive alleles, preventing them from being expressed. You may have heard in humans that brown eyes are dominant and blue eyes are recessive. It only takes once copy of the brown eye color allele to have brown eyes, but it takes two copies of the blue eye color allele to have blue eyes. We will discuss what this means for alpaca traits in later in this chapter. It is also possible that having two different alleles leads to a new trait (phenotype) altogether. This happens when traits are additive. In flowers there are examples where each copy of the R allele adds more red color to the flower. Plants with no R alleles (rr) are white. Plants with one copy of the R allele and one copy of the r allele (Rr) are pink. Plants with two copies of the R allele (RR) are red. We will discuss traits that may follow this pattern in camelids as well.

More Complicated Genes

Some genes or regions of DNA control the expression of one or more (sometimes hundreds) other genes. These are called homeobox genes, when these genes are turned on during different stages of development, or regulatory genes. Mutations in these genes can dramatically affect the appearance of an individual with just a single mutation causing many genes to behave differently. Some of these homeobox genes are involved in segmentation. We do not have a gene to create each separate finger. We have a gene that segments the developing hand. The longer the gene is turned on, the more segments (fingers) are made. In alpacas, this gene is not on very long, and only two digits are made. In humans, it is on longer and five fingers are made. In some humans and alpacas, the gene is left on too long and two many digits are created, or too short, and too few digits are created. The same is true for teeth (we don't have a separate gene for each tooth, but rather a gene for segmenting into teeth, and the longer it is on, the more teeth are created), and for vertebrae and ribs. The environment is often the trigger that turns these homoebox and regulatory genes on and off. Mammals make a special version of fetal hemoglobin while in utero, because they are not breathing air at that time. Once they are born, and oxygen hits their lungs for the first time, many developmental systems needed by the fetus are turned off (like the production of fetal hemoglobin) and many new genes needed for life in the oxygen-breathing, food-eating world are turned on. Every time an animal eats, the genes for many digestive enzymes are turned on. When we fast, they are turned off. While every cell has the same genes, most of one's genes are not turned on (making proteins) most of the time, and different genes are turned on in different cells and different tissues of the body at different times.

Pleiotropy and Polygenic Traits

Some genes make proteins that have many different functions in different parts of the body. Melanin is a great example of this. It is involved in giving all of our tissues pigment, but it is also a structural protein. When melanin is absent in the cells that will

become the inner ear, it can lead to deafness (as happens in blue-eyed white alpacas). When it is absent in the eyes it can lead to blue or red eyes. Genes that have these wide ranging effects are called pleiotropic genes. Other traits (or phenotypes) are caused by many genes acting togther. This can also be true for skin color. In humans, skin color is due to the amount and kinds of melanin present near the surface of the skin (which may increase due to exposure to sunlight), but it is also due to the translucence of the skin (translucent skin allows the blue veins under the surface to be more visible leading to a bluish tint), and even to ones diet (try drinking a carrot juice shake every day for a month, and you will turn orange). Traits caused by multiple genes are called polygenic traits. Diabetes and hypertension are two diseases in humans that appear to be polygenic (caused by multiple different genes acting together, plus the environment).

The Basics of Breeding (Suris and Huacayas)

Half of the genetic makeup of the cria will come from each parent. If anything, the dam provides slightly more input into the creation of the cria, as she provides all of the mitochondrial DNA, and provides the environment within which the cria develops. One set of traits comes in the sperm, another set comes in the egg. What determines what traits are visible in the cria depend primarily upon whether the alleles passed on are dominant or recessive or are additive. If either parent passes on a dominant allele, the cria will express that trait. Both parents must pass on a recessive trait for it to be expressed. The suri allele provides a good illustration of the phenomenon of dominance. The suri allele is almost certainly a dominant trait. The recessive trait can be called the huacaya allele. Every huacaya has two copies of the huacaya allele. Every suri has at least one copy of the suri allele. Indeed, every suri in the registry (with one possible exception) has at least one suri parent. This also means that two huacayas cannot throw a suri, but it is possible for two suris (if both are heterozygotes) to throw a huacaya. Additionally, a huacaya out of two suris cannot throw a suri cria unless it is bred to a suri. Bred to huacayas, a huacaya out of two suris will always throw only huacayas. People are often confused as to how a single mutation could possibly cause the many differences between suris and huacayas (fiber characteristics, crimp or the lack thereof, luster, saddle muscles, etc.). However, these differences could easily be under the control of one gene. The obvious explanations are a pleiotropic gene that effects many different systems (like the melanin example earlier). The second is it could be a homeobox or regulatory gene that controls the expression of many other genes during development.

Color Genetics

The following is my current interpretation of the registry data and data I have collected. It follows along the lines of what Philip Sponenberg has previously preposed (see this volume) as well as ideas proposed by Liz Paul and by my own ongoing research. While explaining all of the known color and pattern variants is fairly complex, understanding the rules for basic base color inheritance is fairly straightforward.

Base Color (solid color gene)

I have found Sponenberg's initial suggestion that light colors are dominant over darker colors to fit fairly well with the existing data. So white is dominant over beige and fawn. Beige and Fawn are dominant over brown, and brown is dominant over black and bay

black. I do not see evidence for dominance within a color, ie. Light fawn is not necessarily dominant over dark fawn. Thus, white is dominant over every thing. Every animal has two copies of the solid color gene. If one of the two copies is the allele for white, the animal will be white, regardless of what the second color is, since white is dominant over everything. Thus white animals may be white covering beige, white covering fawn, white covering brown or white covering black. You cannot tell by just looking at the animal. I will explain how you can determine the recessive color in a later section. By the same rules, black is recessive to everything, so all black animals have two copies of the black allele. So very often, the color an animal is is its dominant color, and its second (or recessive color) must be the same color or darker. Figuring out the second color is critical to making predictions of cria color from breedings.

White Spot Gene (and Blue-Eyed Whites)
[a version of this section is published on the AlpacaStreet.com site]

The white-spot gene (originally suggested by Sponenberg) has many variants (alleles). Allelic variants include markings anywhere on the animal, such as white-face, tuxedo, white legs, pinto, etc.. The white spot gene may also be responsible for the grey phenotype (at least for tuxedo patterned grey alpacas) and possibly even some of the multi-color phenotypes. These different phenotypes seem to behave as if due to dominant alleles. Remember, dominant alleles mask (override, cover or hide) recessive alleles. This means if both alleles are present, only the dominant allele's phenotype is seen. This means an alpaca only needs to get one copy of the dominant allele for the trait to show up in the phenotype. However, an alpaca born with two copies of these dominant alleles (like pinto, white faced, tuxedo) produces a new (additive) phenotype, a Blue-EyedWhite (BEW), or may not be born at all (Liz Paul has suggested that grey by grey mating produce a lethal combination 1/4 of the time, and these are aborted, Paul 2003). It seems that BEWs occur when a cria receives a white-spot allele from both parents. To see what possible outcomes exist for any breeding, it is easiest to create a Punnet Square that shows the possible allele combinations for the offspring of any mating. In this case, we are ignoring the base color and just concentrating on the white spot alleles.

In a mating of a white faced black dam and a solid black sire:
S = solid colored (S/S)
W = white faced animal (W/S)

	Female Gamete		
Male Gamete		W	S
	S	W/S	S/S
	S	W/S	S/S

So half the offspring will be white-faced and half will be solid colored. Something different happens when both parents have white-spot alleles. Some offspring will receive two copies of dominant alleles. These will have the blue-eyed white phenotypes.

In a mating of a white faced black dam and a tuxedo silver grey sire:
S = solid colored animal (S/S)
W = white faced animal (W/S)
G = tuxedo grey animal (G/S)

	Female Gamete		
Male Gamete		W	S
	G	W/G	G/S
	S	W/S	S/S

So 1/4 will be BEWs (the W/G), 1/4 will be grey (G/S), 1/4 will be white-faced (W/S) and 1/4 will be normal (S/S, solid colored). That is how BEWs can be created. It is important to note that some all-white animals have white spots on them, but you cannot see white markings on a white animal. Also, one version of the white spot gene is an all-white animal (think of it as a white spot that covers the whole body). So it is possible to get BEWs from all-white's bred to white-spot or grey animals. Also, it is possible that an animal has a TINY white marking that you cannot see, or easily see. I have seen animals that threw a BEW that appeared solid, but upon close examination, a tiny white spot was found between the toes.

Breeding Blue-Eyed Whites

The question then remains, what happens when you breed a BEW? Should you use one in your breeding program? If the above scenario is correct, then BEWs should always contribute a white-spot allele to a breeding. Therefore, if you breed a BEW to a non-white, solid animal, you should not get a BEW cria from the breeding, but all cria are likely to have white-spot phenotypes.

For example: in a mating between a BEW female (created in the breeding above) with a solid colored black sire:
BEW = Blue eyed white animal (W/G)
S = solid colored animal (S/S)

	Female Gamete		
Male Gamete		W	G
	S	W/S	G/S
	S	W/S	G/S

The result would be 1/2 white-faced cria, 1/2 grey cria. No BEWs. Remember apparently solid animals may hide a white spot, and all-white animals often hide white markings or are a white spot variant (all-white) themselves..

You can use this information to predict the outcomes from your breedings, and avoid making BEWs if you don't want to make them.

What causes the BEW phenotype? This is conjecture based on what we know from other species. We know that most fiber and skin color is due to the presence of

melanin (phaemelanin and eumelanin) in the tissue. Melanin migrates through the body during development. Melanin is also a critical structural component of cells, including hairs in the cochlea in the inner ear. Failure of melanin to reach these cochlear hairs results in their death soon after birth, and deafness. Similarly, failure of pigment cells to reach the cells that will become the eyes leads to blue eyes. Melanin is distributed by the growing neural crest during embryonic development. Melanin is produced from cells called melanocytes that migrate outward in the expanding neural crest. Melanin is produced in melanocytes from tyrosine (an amino acid) by the enzyme tyrosinase as part of a complicated biochemical pathway that ends with the Melanocortin-1 receptor which decides whether phaeomelanin or eumelanin should be deposited (the yellow and black colored pigments that give the skin and fibers their color). You will note that the white markings on animals tend to be at the extremities (head, neck, feet, legs, tail…). This is because the melanocytes migrate from the core outward along the neural crest. Wherever they do not reach is white. So if the cells migrate all the way up the neck, but don't make it to the face, you get a white face.

White spot alleles may or may not include graying and multi-color phenotypes. Most greys are also white-faced (many with tuxedos, white legs, and other patterns). Some greys however have no white markings at all (these are less common, and I do not think they can throw BEWs). It is therefore possible that the white spot gene and the roan/grey gene are not the same gene, but two genes very close to each other on the same chromosome. They are so close that the alleles of these two different genes are almost always transmitted together (so grey and white faced usually occur together). Occasionally, during meiosis, recombination occurs (crossing over between homologous maternal and paternal chromosomes) and the grey and white spot alleles can be separated, leading to the rare whiteless greys. However, I think it is more likely that greys with no white on them are really due to a different gene altogether, and are transmitted as a recessive trait. This is because greys with no-white markings almost never produce grey cria themselves unless bred to grey.

Returning to the risks of using white spot alleles in a breeding program. BEWs themselves should not produce more BEWs unless they are bred to BEW or other white-spot allele animals. To eliminate BEWs from the gene pool completely, we would have to eliminate all greys, white-spotted and pattern animals. Then a good fraction of the white animals would still have white spot alleles. Do we want to eliminate whites, greys, and white spotted animals? I don't. Animals with white-spot often attract many farm visitors because of their distinctive markings. Greys are beautiful and some of the highest selling alpacas. Some other countries' breeding programs are breeding for all-white fiber animals (which can be dyed to any color). There is already some prejudice in some other breeding programs against any animals with non-solid markings because it is difficult to process for fiber mills (colors must be separated prior to spinning and processing). Since the US is not yet a fiber market, but rather a rare-livestock market, we are not necessarily breeding for all-white solid animals. Some in the US have been pushing natural colors, rather than dyed colors.

Environmental Causes of White Spots:

There are some ways you can end up with white spots that are not genetic in nature. Frostbite kills cells and leads to white fibers. Wounds, especially abscesses can lead to small white spots. I often see these on males who have been bitten by other males while fighting and have small dime-sized white spots along their backs. I also see these small white spots where injections are commonly given (since injections can lead to abscesses). Note that since genetic white spots typically occur at the extremities, isolated white spots in the torso are less likely to be genetic white spots, or at least less likely to be alleles of the white spot gene, and therefore may not increase the risk of producing a BEW.

Blue-eyed Non-whites:
Non-white animals with blue eyes are almost never deaf. Blue eyes on their own do not appear to be a BEW risk factor. Every blue-eyed non-white I have come across has been out of a grey or a white animal that has grey in the background. I believe ALL greys can produce blue-eyed non-white offspring. As they are not deaf, I think this is an irrelevant trait, although blue eyes will devalue some animals in the US marketplace (for no logical reason). Blue-eyed non-whites do not seem any more prone to producing blue eyed offspring than tuxedo greys do, but the data is exceedingly small on these animals, so it is hard to be too sure of the inheritance patterns of the trait at this time.

For myself, I don't usually use herdsires with white-spot since offspring that have white-spot have more limited opportunities for breeding due to the risk of making BEWs when breeding them to gray, and white). I always breed to make a herdsire-desireable male in every breeding. I try to minimize combinations that are hard to sell, and maximize ones that are sought after. In Europe, white marked animals are devalued on the market (even though no-one processes the face fiber anyway where the white spot often ends up). Other than greys and suris, there are very few white-spot herdsires in widespread use in the US. AOBA fleece and full-fleece halter judging also penalize for color variation in the blanket. You can certainly use white faced females in a breeding program (we have a lovely white faced DSG Nic-Nac daughter in our foundation herd that is spectacular). Bred to solids, they will produce 50% solid cria and 50% white spot cria. Greys are very trendy right now in the US market, and often command top-dollar at auctions, and they almost all have white markings. Greys are also the rarest color combination, increasing their value for both males and females, despite having white markings.

Breeding Rule to Prevent Making BEWs
Breed them to only solid-colored, non-white animals if you don't want to risk BEWs. One member of every breeding pair should be solid and non-white to avoid making any BEWs. The exception is if you want to make whites, than you should obviously breed to whites, and you cannot eliminate some risk of making BEWs.

BEWs and Greys
Since many BEWs are made by a tuxedo grey parent bred to a white-marked parent, BEWs out of greys can make grey cria themselves. Indeed, a BEW out of greys should produce grey cria just as often as a grey animal would. Since BEWs are often heavily devalued in the marketplace, it can be an inexpensive way to make greys. Note that not all BEWs are made by a grey parent, so not all BEWs can produce greys.

So it is up to you and your breeding program goals. Personally, I would happily breed BEW females to solid non-white males. I would not likely breed a BEW male at all. Eliminating a BEW from the breeding program removes two copies of the white-spot gene from the gene pool, but unless we are also removing greys and other white-spot animals from the breeding program, it will have a negligible effect on the overall level of white markings and BEWs in the North American gene pool. The impetus to avoid making them is primarily financial (as BEWs are badly devalued in the US market) and medical (deaf animals may require extra care and can occasionally be harder to breed and behavior test as they cannot hear the male's orgling).

Lethal Grey

Liz Paul has suggested, based up the Australian Registry data, that receiving two copies of the grey allele is lethal, and these animals are never born. The evidence for this is circumstantial population genetic data. There are three scenarios for a breeding between two grey alpacas. 1) If two copies of grey makes grey, then there should be 75% or more grey offspring from grey to grey breeding, and some (homozygous) animals should produce grey no matter what color they are bred to. 2) If two copies of grey make a blue-eyed white, then 25% of grey to grey breedings should yield BEWs. 3) If two copies of grey is lethal, than we are left with1/4 not born double greys, 2/4 grey, and 1/4 solid animals (or 66% grey and 33% solid animals born).

For example: in a mating between a tuxedo grey female with a solid tuxedo grey sire:

G = tuxedo grey allele

S = solid allele

GG= ??? [scenario 1. = grey, scenario 2. = BEW, scenario 3. = lethal]

	Female Gamete		
Male Gamete		S	G
	S	S/S	S/G
	G	G/S	G/G

The US registry and the Australian registry show pretty close to 66% grey cria when two greys are bred, exactly as predicted if the lethal grey theory is correct. Further, I have found no greys in either registry with >20 cria on the ground with 100% grey offspring, or even 100% grey OR white offspring (since white seems to be dominant over grey). I have collected a large body of anectdotal evidence for lethal grey. We lost a grey-to-grey bred cria at 5 months gestation, and many have emailed me similar tales of woe. I do think that most of the time, the breeding just does not take. And since you get homozygotes only 25% of the time, 75% of the time you will have no problems. This is why I always council breeding greys to solid non-white mates. Then you will get 50% greys (on average) without the added 25% reproductive loss of grey to grey breedings. Further, I would argue that you could always find a MUCH better fleeced fawn animal to breed to than most grey mates you would choose, so you can increase the fleece much more quickly breeding grey to solid animals with great fleece (than you can breeding

grey to grey). Personally I love breeding my grey girls to fawn accoyo males that cover black, but any great fleeced non-white will do the trick.

Dilution Gene

Sponenberg and others have proposed a dilution gene exists in alpacas, as it seems to do in many other mammalian species. My interpretation of this gene is that it dilutes (lightens) the color of animal animal's base color to a lighter color. This is how we sometimes get fawns or browns out of two blacks. The dilution gene appears to act as a recessive, meaning both parents must pass it on for the cria to be diluted to a lighter color. It does not appear to be exceedingly common, and is traceable through pedigrees. Evidence that an animal carries one copy of the dilution gene is if two parents produce a cria lighter than either parent. In this case, lighter means a lighter color, not a lighter shade within a color (ie. Throwing a light fawn out of two dark fawns is not necessarily evidence of dilution). The diluted animal should then be carrying two copies of the dilution allele and should always pass it on. This does not mean all of its' cria will be dilute though, as the animal it is mated two must also pass on a dilution allele for the offspring to be diluted versus the parents. It is most easily spotted when mating dark animals, because they have so many colors lighter themselves. It would be harder to discern when breeding a beige and a fawn for example.

UV Fading Gene and Tipping Genes

Some animals fleeces fade when exposed to the ultraviolet light of the sun. It is the same reason many of us get blond highlights in our hair when we spend a lot of time outside in the summer. The fleece exposed to the sun "tips out" to a lighter color. It should be noted though that many animals that have tips do not appear to be due to the sun. Suris often demonstrate this. Since suri fleeces hang down, the fleece along their toplines is exposed almost all the way to the roots, yet in many of these animals, only the tips are lighter. This indicates that some animals that tip-out in a different color do so for reasons other than UV fading. The Chinchilla and Agouti coat patterns in mice and cats put different color stripes or tips on each fiber, so there is ample precedent for such a gene in alpacas.

Dark Spotting Gene

Dark spots are caused by a different gene than are white spots. Dark spots appear to be dominant and can appear on any color back ground from white to black. The size, number and locations of the spots can vary wildly from generation to generation. I have noticed that it is common for more spots to appear with age, and for the sizes of the spots to also increase with age. Not in all animals, but I have seen the number and sizes of spots increase with age in many animals, including several I have owned.

How to Determine Recessive Colors

first the basic rules:

1) for the basic color gene, light colors are dominant over darker colors

2) every animal has two copies of each gene, but only one gets passed on in the sperm or egg. Usually the color the animal is is one color, and the second color (often called the recessive color) is the same color or darker. So an animal inherits two colors, the lighter of the two it inherits is the color it will be.

3) black is recessive to everything, white is dominant over everything
4) greys with white on them (often called tuxedo greys) are silver grey if they are a black animal with the grey allele of the white spot gene, and are rose grey if they are a beige/brown/or fawn animal with the grey allele.
5) some small percentage of whites are actually a darker animal with a white spot overriding everything (not the normal dominant white)
6) getting two copies of the dilution gene dilution allele in one animal will lighten the color (you can tell this happened if you get a cria lighter than both parents)

Tricks to finding the recessive color:
1) an animal covers black if
   a) it has produced black or silver grey cria
   b) is out a black or silver grey parent

2) If you breed a white animal to a black animal and you get a non-white color, the non-white color is the recessive color of the white animal (unless the diluting gene has diluted a darker color to this color).

For example, I have an alpaca (Princessa) who is out of a white sire that covers dark brown and white dam with unknown recessive color. Princessa is light fawn. This tells me she is light fawn covering dark brown. I know this because if she had gotten white from her sire, she would be white. She is not white, therefore she got her sire's recessive color (which I had already figured out from his pedigrees and offspring). I also now know that the dam is a white covering light fawn. If she had passed on white, Princessa would be white. Light fawn is dominant over dark brown, so the light fawn color of princessa had to come from her dam, and the dark brown from her sire is her masked/hidden/covered/recessive color.

It is not always possible to figure out recessive colors, as there may not be enough data to figure it out. Just remember that the second color must be the same color or darker than then animal's visible color. By tracing through the pedigrees it may be possible to assign second or recessive colors to many ancestors and in doing so, narrow the possible second or recessive colors for the animal you are investigating.

Evolution and Domestication of Camelids
[updated from a poster I presented at the American Association of Physical Anthropologists meeting in Philadelphia, PA in April 2007]

Background

Alpacas were domesticated from vicunas in Andean South America over 6000 years ago. Llamas were domesticated from guanacos around the same time. Since that time there has been introgression between llamas and alpacas, as evidenced by (currently 3) STR (Short Tandem Repeat) markers that are mutually exclusive in their allele size ranges between guanacos and vicunas, but whose ranges partially overlap between alpacas and llamas. This admixture was accelerated with the conquest of the Inca empire

and the subsequent decimation of the Incan alapca and llama herds and breeding programs by the Spanish.

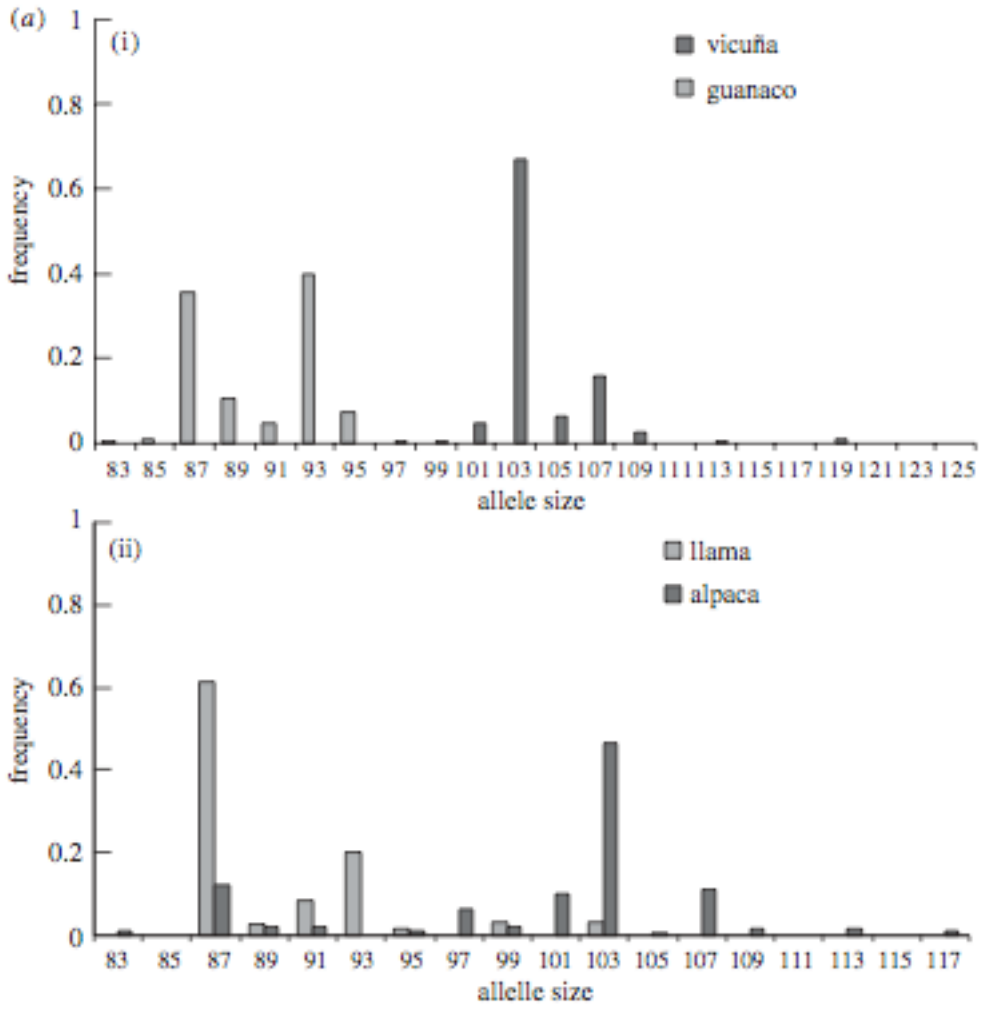
Creation of the North American Herd

North American farmers imported alpacas into the United States and Canada in the 1980s (primarily from Chile) and in the 1990s (in carefully judged imports primarily from Peru, but also including some from Bolivia and Chile). The US herd now exceeds 100,000 animals. I examined whether the phenotypic traits (chosen to avoid llama-like traits) the judges used to screen the imports actually reduced the number of guanaco (and thus llama interbreeding) alleles in the alpacas that were screened, versus the unscreened animals from the 1980s imports, and as compared to populations in Andean South America alive today and from the fossil record.

Markers from a panel of 200 STRs we are using to map fiber characteristic genes in camelids may be a useful tool in trying to recreate the original alpaca and llama phenotypes from the time of the Inca and earlier.

Admixture and Hybridization in South American Camelids

Kadwell et al., 2001 identified two dinucleotide repeat markers (LCA19 and YWLL44) in camelids that had mutually exclusive ranges of allele sizes between guanacos and vicunas (the wild parental species of the domesticated species llamas and alpacas, respectively). Kadwell et al., 2001 showed that the allele size ranges of alpacas and llamas did overlap, indicating admixture /hybridization between the two species (see histograms from Kadwell et al., 2001, figure 3a. below).



Selection of animals in the US herd was done before the discovery of these markers. Many of the animals selected for import in the 1990s (primarily from Peru, but also some from Bolivia and Chile) were screened to avoid selecting animals with llama-like features. Llama features that differ from alpaca include high tail set, large banana shaped ears, guard hair second coat, bare legs or poor leg coverage, large body size and taller height at the withers). So screening judges eliminated alpacas with any of these characteristics as being likely of having llama admixture. Imports from Chile prior to 1990 were typically not judged. We hypothesized that Peruvian Imports would have fewer llama-sized alleles than the Chilean imports.

Variation in the Import Generation of the US Herd

Using the DNA Detail STR typing data from 145 imported alpacas from the Alpaca Registry Inc database data sent to D.A.M. by alpaca owners, heterozygosity of each locus was estimated for the 9 primary loci used in the registry. The loci screened in this study are listed in the table below with heterozygosities by country of origin.

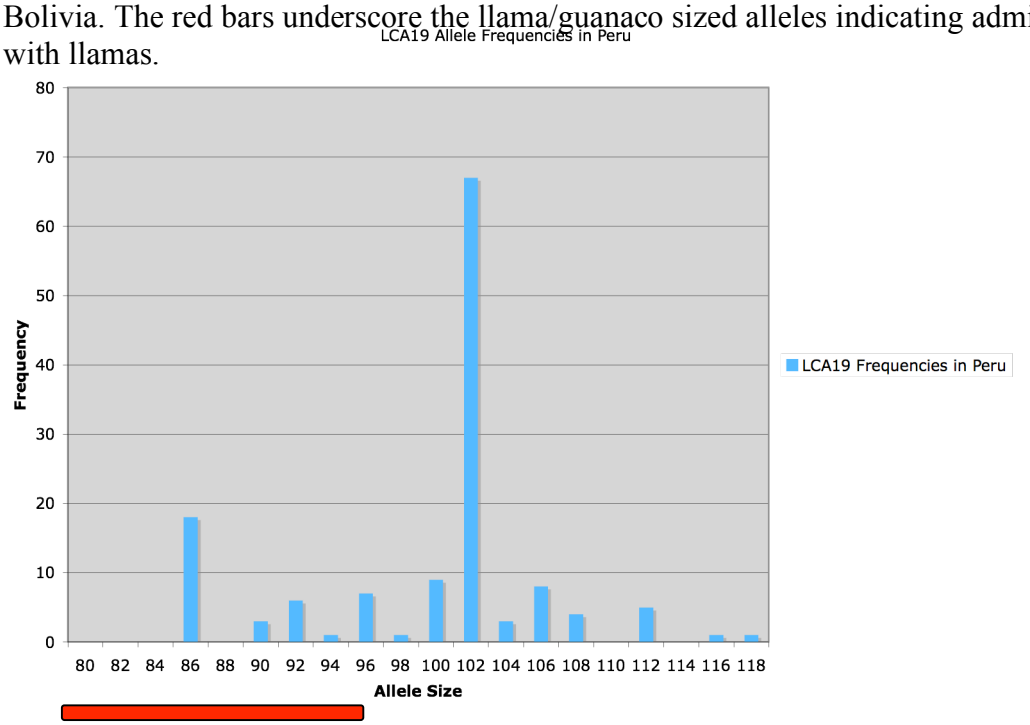
Pop.name/ Heterozygosity	LCA19	LCA37	LCA5	LCA66	LCA8	YWLL08	YWLL29	YWLL36	YWLL40
Bolivia (n=12)	0.833333	0.916667	0.833333	0.75	0.833333	1.0	0.916667	1.0	0.416667
Chile (n=66)	0.681818	0.757576	0.484848	0.742424	0.878788	0.893939	0.606061	0.80303	0.454545
Peru (n=67)	0.746269	0.880597	0.671642	0.686567	0.791045	0.910448	0.772727	0.861538	0.569231

Regardless of country of origin, alpacas from all three regions were remarkably variable, as evidenced by their high heterozygosities above (calculated using the MVA program (Dieringer and Schloterer, 2002). This is strong evidence that there was not a significant bottleneck in the variation imported into the US, or for that matter that existed in South America prior to import. Similarly, genetic distances (Nei et al.'s 1983 Chord Distance) between the three groups were relatively small but nonetheless appreciable, as shown below, indicating some isolation between the populations in South America.

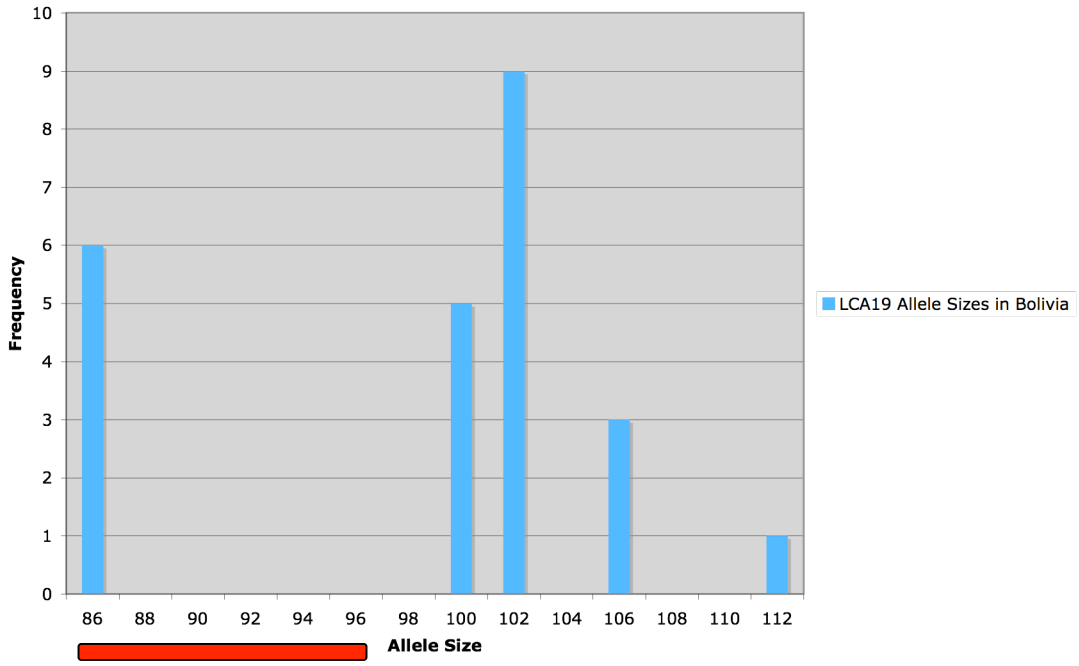
Nei's Chord Distance (Nei et al., 1983)

	Bolivia	Chile	Peru
Bolivia	0.00000	0.13136	0.12674
Chile	0.13136	0.00000	0.05359
Peru	0.12674	0.05359	0.00000

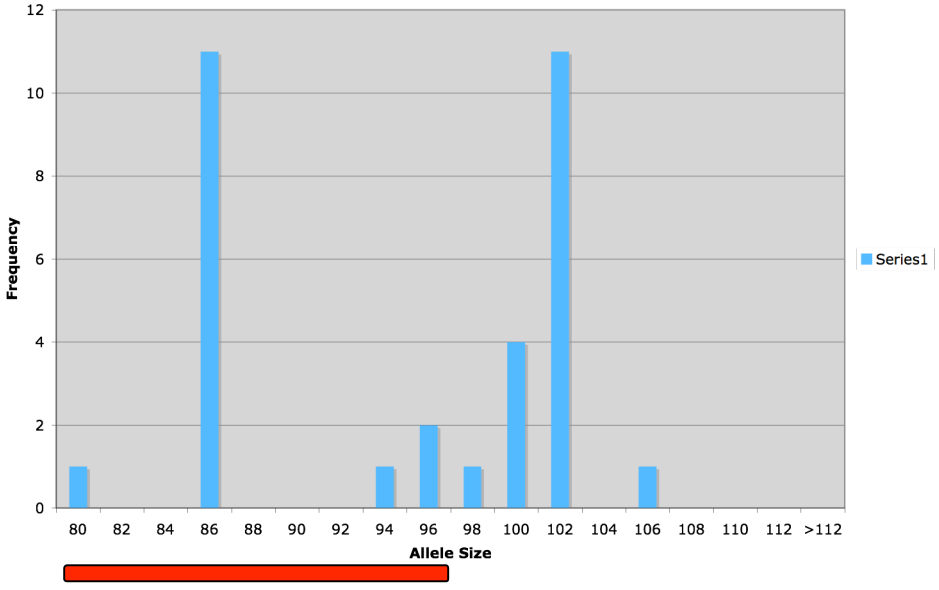
The small sample size of Bolivian Imports must be noted, and frequencies and distances may change with larger data sets. Below are the histograms of the frequencies of STR alleles sizes for LCA 19 in 145 alpacas imported into the US from Peru, Chile and Bolivia. The red bars underscore the llama/guanaco sized alleles indicating admixture with llamas.



LCA19 Allele Frequencies in Bolivia



LCA19 Allele Frequencies in Chile



Population	# Guanaco/Llama Alleles	Total # Alleles	% Guanaco/Llama Alleles
Alpacas: Bolivia	6	24	25.00%
Alpacas: Chile	15	32	46.85%
Alpacas: Peru	35	134	25.12%

As we can see from the three histograms of Imported alpacas into the US from Chile, Bolivia and Peru, there is quite a bit of llama admixture in the US alpaca genome. The results are summarized in the table above.

Conclusions:
The US herd exhibits tremendous variation as evidenced by the high heterozygosity of the 9 STR loci examined in this study. Further, while all alpacas surveyed had a substantial amount of llama admixture (all had over 20% llama sized alleles), the screening process in the Peruvian Imports seems to have reduced the amount of llama admixture successfully by selecting against llama-like traits in alpacas. Peruvian and Bolivian imported alpacas have less than 1/2 the llama-sized alleles at LCA19 than did the Chilean alpacas. We are still collecting and assimilating variation in South American Alpacas to see if the levels of llama admixture in the national herds of Chile, Bolivia and Peru differ. It is possible Chilean Imports had more llama admixture because Chilean alpacas had more llama admixture, or it is possible that the levels were the same everywhere, but the screening of alpacas from Bolivia and Peru eliminated many llama alleles while the earlier unscreened Chileans brought in more llama alleles. It is predicted that Chileans that came in via judged imports (some came in in 1998 in the Peruvian import) would show reduced llama admixture as well. We have insufficient samples of the judged Chilean Imports to test this hypothesis, but hope to have the full data in the coming year and will test it as soon as we have the data in hand. Kadwell et al., (2001) estimated llama admixture at 82-90% in the South American herd overall, so this would imply all of our import selections greatly reduced the level of admixture, even if just by selecting the most phenotypically correct alpacas.

Please note that these markers are not useful to estimate the level of admixture in an individual animal. An estimate of individual admixture would require a large number of such markers. These markers are useful for estimating population admixture. It

Future Work:
My lab is screening 200 STRs across a broad range of camelids to identify the genes involved in domestication, including fleece color, fleece character, and inherited diseases of alapcas and llamas. We are also involved in the first complete genome of an alpaca which should be a blueprint and a road map for future studies. We hope to have access to all 12,000 founding animals for the US registry to test these results with a much larger sample size. We will also be able to break down variation by region and by farm.

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