

The Genetics Journal  
Entry #1

This column, for the foreseeable future, will be devoted to providing the elementary building blocks of genetics to alpaca breeders who have never been taught about genetics. After having written two columns for AM over the last year on the more advanced topics of the genetics of animal breeding it has come to my attention that there are many breeders out there who do not have the fundamental background in genetics that I assumed they did. Add to that the fact that over one quarter of our membership just became alpaca owners/breeders in the last eight years and it seems prudent to go back to the beginning for those who will benefit from such an approach. Hopefully it can be a useful review for others. If anyone has any basic topics or questions they want information about relating to the genetics of animal breeding please write to me and I will try to include them in this column when appropriate. For now, we will follow Lewis Carroll's advice, "Begin at the beginning and go on till you come to the end; then stop."

Genetics is the name given to the study of how qualities and characteristics of the parents are inherited by their offspring. It has as its foundation some very basic principles. Just as numbers, counting, addition and subtraction must be mastered before one can learn multiplication, division and square roots, and all of these must be in hand before attempting to learn algebra, geometry, trigonometry, calculus and so on; the basic genetic principles must be mastered so they can become building blocks to understand more complex areas of genetic science such as population genetics, gene frequencies, the quantitative genetics of polygenic traits etc. This column is about learning the numbers, counting, addition and subtraction of genetics as it were

As alpaca breeders our work is to take a pair of alpacas, male and female, and produce a new alpaca offspring known as a cria. It is important and useful for us to be able to predict as much as possible about what the outcome of that mating will be. The genetic code of this newly produced cria is different from either of its parents, totally unique, and determined entirely at conception. All of the genetic information that cria has or ever will have is determined at the time the sperm cell joins with the egg to form a single cell called a zygote. All of this genetic information is stored in molecules called DNA (deoxyribose nucleic acid). These DNA molecules, and several other molecules that form a framework to hold these coiled molecules, form the structures we call a chromosome. This newly formed life, consisting at first of a single cell called a zygote, has 74 chromosomes in 37 pairs. Thirty-seven of these chromosomes came from the sire by way of the sperm cell and 37 came from the dam in her egg cell. Although the duplication of this material begins immediately as that first cell makes an exact copy of itself, and as this duplication process takes place literally trillions of times as all of the cells of this cria are formed and then trillions more times during the lifetime of the cria as its body cells replace themselves, the genetic information never changes. No new information is added or taken away from that which was put together at conception.

One half of this unique genetic combination came from the sire and one half from the dam. Each parent donates 37 chromosomes of identical structure and composition to make 37 pairs of the 74 total chromosomes, with the position of the genes for each trait at the same location on each one of each pair of chromosomes. Genes are distinct and discreet units of information about specific traits. Each gene is transferred as a whole unit. Before genetics was understood people believed that inheritance resulted from the mixing together of the blood of the sire and the dam, sort of like pouring two colors of paint together and getting a result that is the combination of both. This misconception about inheritance coming from the blood still lingers in our language. We often hear of “bloodlines”, “mixed blood”, the “pureblood” animal, etc. The fact that each gene is distinct and unchanged as it is passed to an offspring is important to understand and we will see why when we discuss the Mendelian concept of gene segregation. The actual genes donated may in fact be the same or different from each parent. For example, the genes controlling eye color will be in exactly the same location (called a **locus** in genetic terminology) on the chromosome from the male and on the one from the female, but it might be coding for blue eyes on the female’s chromosome and for brown eyes on the male’s chromosome. The different types of gene that can occur at one locus on each of the two chromosomes in the pair, in this case blue eye color or brown eye color, are called **alleles**. At any one locus the two chromosomes can have the same allele, for example two genes for blue eyes, or two different alleles. We will discuss later how these two alleles interact to determine the expression of a particular trait for the individual animal in question. There may be many possibilities of alleles that can be present at a given locus, but the individual organism can have at most two of the different available options, one on each chromosome. In our French Angora rabbits for instance, there are five different genes or loci (plural of locus) that affect the rabbit’s coat color. These five genes are labeled A, B, C, D, and E. The C gene locus on each chromosome can have any one of five different alleles: C for full color expression, chd for a dark chinchilla expression, chl for a light chinchilla pattern, ch for the himalayan allele and c which blocks out all color and produces a white rabbit with red eyes. However, even though there are five possible alleles for this locus, each rabbit can have at most two of them, one at that locus on each chromosome. It is possible that a given rabbit will carry only one of these alleles, if they have the same one on each chromosome. In that case, when the alleles are the same, this condition is called **homozygous**. When the alleles are different it is called **heterozygous**.

The interesting and exciting thing is that, at least for our basic discussion, each parental contribution is a random sampling of that parent’s total genetic code. I don’t know for sure how many different genes exist in the genetic code for an alpaca, but in the mouse for instance there are over 30,000 pairs of genes on its chromosomes. Each one of those pairs sorts randomly when a new egg or sperm cell is formed. This Mendelian principle is known as **independent assortment**. As a result of independent assortment the number of possible different combinations that could be formed is the number two raised to the 30,000<sup>th</sup> power, which means multiplying two, times two, times two... thirty thousand times! You can see then that the chance of the genetic information passed from a parent to any two or more offspring ever being exactly the same is almost zero. Each one will be unique and may theoretically differ at only one gene locus or at all thirty thousand or

anywhere in between. This is why no two offspring from the same parents are ever exactly alike, with the exception of course of identical twins, which in fact however still came from just one sperm and one egg.

For our introductory discussion however let us just consider one gene. This makes the possibilities much more manageable to discuss and will allow us to better understand the basic principles of genetics and inheritance. Some characteristics of an organism, or traits as geneticists refer to them, are governed by only one gene.

For a classic example we will look at the work of Gregor Mendel, a monk who was the father of genetics as a science, to explain the inheritance of traits from parents to their offspring. Among other things, Mendel bred and recorded observations about his pea plants. One that he became aware of right away was that the plants were either tall or short. He found that when two short plants were allowed to fertilize each other, 100% of the offspring were short plants also. He found out that a certain strain of tall plants that when bred together always produced 100% tall plants. There were also some strains of tall plants that looked identical to the others, but when they were bred they always produced a crop of offspring that consisted of 25% short plant and 75% tall plants.

He then experimented by taking the tall strain that produced all tall offspring and crossed them with plants from the strain that always produced short plants. From this mating scheme he got 100% tall plants in the first generation, F1. Therefore, he said that these tall plants were dominant to the short plants, since all of the first generation offspring were tall. He then undertook the interesting experiment to breed these F1 tall offspring together and discovered that they would produce an F2 generation that consisted of 25% short and 75% tall plants. It is important to note as an aside here that F2 not only represents a second generation, but more specifically means a second generation produced from breeding together only the individuals in the F1 generation. He realized then that his tall strain of plants that produced this same result must have come from a combination of the genetics of true breeding tall plants and true breeding short plants. After a lot of study, observation and thought Mendel figured out a mechanism to explain these results that we call a simple dominance relationship of a single gene trait.

Here is what was happening. For each trait in question, in this case the tall or short style pea plant, the offspring gets one copy of the gene for that trait from each parent. This is the Mendelian principle of **gene segregation**. There are two genes or alleles for every locus on a pair of chromosomes, in this case for the locus that will program the plant to be either a tall or short style pea plant. The alleles that an individual carries are referred to as its **genotype**, while the traits caused by those alleles are referred to as its **phenotype**. Let us use a capital T to represent the gene (allele) that causes a plant to be tall and a lower case t for the allele that causes a plant to be short. If each parent contributes a t, then both copies of the gene at this locus will be t and the plant will be short in its appearance (phenotype) and will be homozygous (the same) in its genotype, which we can represent with two lower case letters like this tt. If both parents contribute a gene for the tall characteristic, the plant will again be homozygous for tall TT. When either of these homozygous plants prepares to reproduce, one of these two copies of this allele will

be randomly chosen to be passed on and there is a 50% chance that the offspring might get either one. Since in the homozygous circumstance both copies are the same, the offspring will always receive the same genetic contribution from this parent. The homozygous tall plant (TT) will always pass on a T and the homozygous short plant (tt) will always pass on a t.

What if a plant has one copy of each however (Tt), which is referred to as heterozygous? The plant will appear tall, exactly like the TT plants since the T exerts simple dominance over the t. We cannot tell by looking at a plant if it is TT or Tt, they both look the same. All we know for certain about the tall plant is that it has at least one T gene to make it appear tall. When we look at a short plant however, we know that it must be homozygous for the recessive (opposite of dominant) gene and thus can be represented as tt, because if it had even one T it would have a tall appearance (phenotype).

Let's go back to Gregor Mendel's experiments now. His short strain of pea plant always produced more short plants as offspring. This is because they were tt, and could only each contribute a t to the offspring. Therefore all of the offspring were tt short as well. The tall strain of plants that could only produce tall offspring together must have been homozygous for the T gene, or TT as well. Therefore the only outcome of a cross between these plants was TT tall. The tall strain that produced both tall and short plants must have had the alleles present for both tall and short traits. The offspring that got a copy of the short gene, t, from each parent were short. Any time either parent passed on a T to the offspring it would appear tall, regardless if it received a T from both parents and became TT or a T from one parent and a t from the other in which case its genotype would be Tt (heterozygous) but it still would exhibit the tall trait. The result was that this strain produced both tall and short plants, but the tall were ones much more numerous.

When Mendel went on to cross the "true breeding" strain of tall plants, which were homozygous TT with the "true breeding" short plants, homozygous for tt, he got only tall plants. Now we can understand why. The tall strain could only contribute one T, the short strain could only contribute one t, therefore all of the first generation of offspring, the F1, were heterozygous, Tt, and all appeared tall due to the simple dominant effect of the one T gene.

In our next issue of the journal we will examine an important tool called a Punnett square which can be used to clearly illustrate what Mendel discovered and we have studied here. We will also look at how to use that same tool to predict possible outcomes of different matings. In the mean time, if anyone has general questions about basic genetics that you would like to see discussed in the Genetics Journal please e-mail them to the author at : [journal@alpacagenetics.com](mailto:journal@alpacagenetics.com)