

BY THE NUMBERS

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The Uncontrollable Randomness of Breeding

Richard Bourdon wrote, “As animal breeders, our task is not to change individuals, but populations.”

This idea is still true in animal breeding today. Fundamental properties of animal breeding extend from two different ideas: Mendelian inheritance and population genetics. While Mendelian inheritance explains the genetic mechanisms of individuals, population genetics extends those principles from the individual to the population.

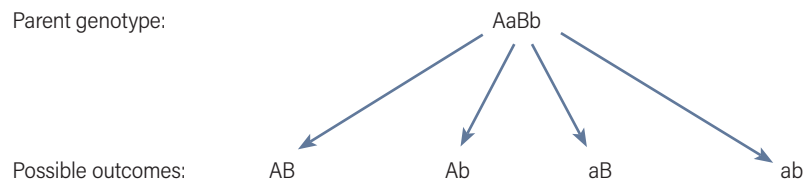
Mendelian inheritance, named after Austrian monk Gregor Mendel, refers to the pattern in which genes are passed down from parent to offspring. First discovered through the now-famous pea-plant breeding experiment, Mendelian inheritance is broken into two different parts: the *law of segregation* and the *law of independent assortment*. These laws help explain why variation exists from mating to mating.

Mendelian Law

It is important to understand cattle, like humans, are diploid organisms, meaning half of the DNA in a progeny’s chromosomes will come from its sire, and half will come from its dam.

The *law of segregation* refers to a trait being influenced by a pair of alleles, or genes, but each parent can only pass down a single random allele to its progeny.

Fig. 1: Possible inheritance outcomes of a two-locus genotype



The *law of independent assortment* is the idea these alleles will independently combine within a gamete (sperm or egg). This will cause the random half of DNA passed down from parent to offspring to vary as well.

Take the example in Fig. 1 above. In this simplistic example, a trait is controlled by a two-locus genotype, meaning two different pairs of alleles (four alleles in total) work together to control the same trait. By rule of nature, a single parent passes down one-half of its alleles that control this trait. In this case, the parent is heterozygous for both pairs of alleles (AaBb), and, as a result, can pass on four different combination pairs.

The exceptions

While Mendel’s original work in peas followed these two laws very specifically, exceptions to the law of independent assortment arise. These exceptions are caused by linkage, or

the idea two genes can be linked if they are in the same area as one another in the genome.

Linked genes tend to be passed together during gamete (sperm or egg) formation. In the example above, let’s assume A and B are linked, as are a and b. This would support these two allele pairs would be passed down together, unless a recombination event occurs.

Recombination is the formation of a new combination of alleles in the event the alleles cross over during gamete formation. This process creates even more genetic variation from parent to offspring.

How much variation is caused by Mendel’s laws?

These two laws combine to cause 50% of the variation seen in an individual progeny, known as *Mendelian sampling*.

Not only does Mendelian sampling capture the variation that exists in the random sampling of parental

genes caused by segregation and independent assortment of genes during gamete formation, but it also describes randomness of the selection of those gametes to be used for the formation of the embryo. It determines the genetic makeup of the offspring.

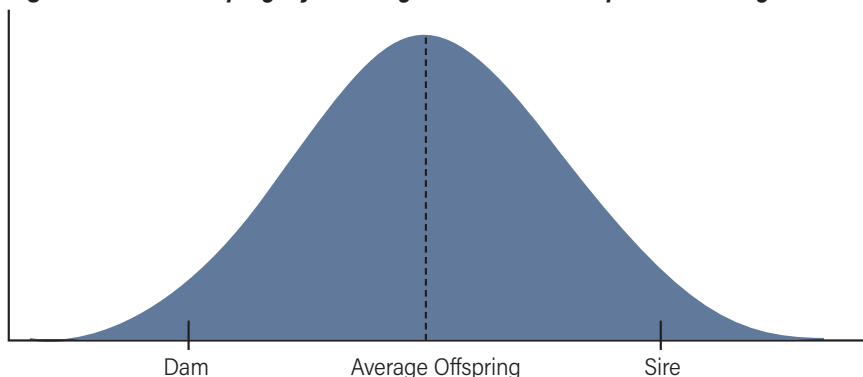
All in all, the effect on variation due to Mendelian inheritance is large, and can multiply very quickly.

Gametes (sperm or egg) will receive a favorable sample of given genes from the parent, while others will not. This process is equally random when it comes to embryo production. Not only are gametes made up of a random assortment of DNA, but then it is totally random as to which sperm and egg will sync up for embryo creation.

This variation in the population is crucial for the success of genetic selection through artificial mating. If no variation existed within a population, there would be no room for breeders to make genetic improvement. On the flip side, the variation induced by Mendelian sampling is oftentimes the largest nemesis as well. Just because a mating works one time does not guarantee the same result will persist in the next identical mating. Likewise, just because a mating does not work the first time does not mean it will not work on the second try.

While tools exist that allow for the tracking of the random sampling of genes (i.e., genomics) that get passed down from parent to offspring, there is no way to control what genes are passed down. The randomness of these events causes the resulting genetic merit of individual progeny to fall below or above the parental average (PA) expected progeny differences (EPDs) calculated prior to breeding. If the same sire and dam are mated hundreds of times, and those matings resulted in hundreds

Fig. 2: Distribution of progeny breeding values around the parental average



of progeny, the average breeding value of those animals would be equal to the average of the parental breeding values. However, due to the random sampling of genes passed down, an individual animal can fall above or below the average.

In the end, the distribution of the progeny will follow a normal distribution, or bell-shaped curve, depicted in Fig. 2. As you can see, some of the resulting offspring will be inferior to both the sire and dam, others will be superior to those parents, and most will fall somewhere in the middle.

The random draw that creates those superior genetics allows for those individuals to continue to drive genetic progress. In doing so, this shifts the distribution, or bell curve, of the entire population in a positive direction over time, creating genetic progress in a population.

A bit of luck

In some ways, the random sampling of genes passed down by chance from one mating to another is like a random hand of cards being dealt by the dealer. Sometimes a player receives a good hand; other times, it is a complete bust.

Breeding cattle is somewhat luck of the draw, as well. It really can become a decision of weighing one's tolerance of risk and reward. Using proven genetics that have genetic

predictions backed by mounds of data can add consistency to the calf crop. However, just because genetics are considered proven doesn't mean genetic variation no longer exists. Even matings between high-accuracy parents are subject to the unpredictable randomness of Mendelian sampling.

Understanding this phenomenon cannot be controlled allows breeders to go into mating situations with realistic expectations. Creating breeding objectives and staying true to your breeding goals will increase your chances of success. Using tools like EPDs to track the variation in your population helps support genetic improvement over time. Mating superior genetics to superior genetics will increase your chances of getting superior offspring. However, the randomness introduced by Mendelian sampling can humble breeders in a hurry.

Breeders should not fret over this variation. It's when variation ceases to exist that breeders should start to worry. **A**

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Editor's note: If you have questions, please contact the AGI team at 816-383-5100.