Pattern Interaction

In the last installment of Hoist the Colors, I discussed the what is often referred to as the two-gene theory of appaloosa coat color inheritance. That theory proposes that there is one gene that “sets the stage” and another gene (more likely a number of other genes) that then adds the white patterning. That first gene, referred to as leopard complex, is responsible for the appaloosa coloring most of us know as varnish roan. The second gene (or genes) adds the white patterning necessary to make the higher contrast patterns like leopard and blanket.

Appaloosa patterns are therefor a bit more complex than patterns like tobiano and frame overo that are the result of a single dominant gene.

This situation is made even more complex by the fact that both leopard complex and the appaloosa patterning genes interact with the different base colors. Appaloosas can appear quite different depending not only on the pattern, but on the base color of the horse.

A Quick Review of Base Color Genes

Beneath the wide variety of dilutions and patterns, all horses can be said to be one of three basic colors: black, chestnut and bay.* These three colors are the result of two different genes.

The first gene, extension (E), determines whether or not the horse can produce black pigment. The default pigment in horses is red, so if the horse cannot produce black, he will be red. The dominant form of the gene (E) means the horse will be black; the recessive form (e) means the horse will be red.

The second gene, agouti (A), determines where the black pigment will go. (Red horses do not have black pigment, but the instructions are still there.) The dominant version (A) restricts black to the mane, tail and legs, leaving the body red (ie., a bay horse). The recessive version (a) does not restrict the black pigment, so the horse is all-over black.

This is important to the understanding of appaloosa patterns because these two genes - extension and agouti - interact with white patterning genes. That interaction can alter the appearance of those patterns.

Although the chestnut horse appears to have louder markings than the black, differences in how base colors interact with patterning genes might mean that the black horse had more, genetically speaking.

* I have lumped wild bay and brown horses in with bay for the sake of simplicity.

Base Colors Influence White Patterning

Because extensive studies were done on leg and face markings, it has long been known that the average amount of white on a horse varied depending on the base color. Black horses have the least amount of white, while bays have a little more, and chestnuts have the most.

What's more, the level of white markings are influenced not just by the visible base color of the horse, but also by the colors the horse carries hidden. So while all black horses are more resistant to white markings, black horses that are homozygous for the black gene (EE) are even more resistant than black horses that carry chestnut (Ee).

Because bays have more white on average than black but less than chestnut, it is believed that the dominant form of agouti (A) boosts white. This boost isn't enough to overcome the difference between black (E) and red (e), so bays still have less white than chestnuts. But it is enough to make a difference between bay and black.

And just as carrying red (Ee) makes a black horse less resistant to white, a bay that carries black (Aa) will have less white (on average) than a bay that does not carry black (AA).

This leads to the following ranking of the colors, with the most white-resistant first, and going down to the least resistant.

Base colors ranked by resistance to markings		
Rank	Color	Code
1	Black (no red, pure for black)	*EE aa*
2	Bay (no red, carries black)	*EE Aa*
3	Bay (no red, pure for bay)	*EE AA*
4	Black (carries red, pure for black)	*Ee aa*
5	Bay (carries red, carries black)	*Ee Aa*
6	Bay (carries red, pure for bay)	*Ee AA*
7	Chestnut (red, carries black)	*ee aa*
8	Chestnut (red, carries bay and black)	*ee Aa*
9	Chestnut (red, carries bay)	*ee AA*

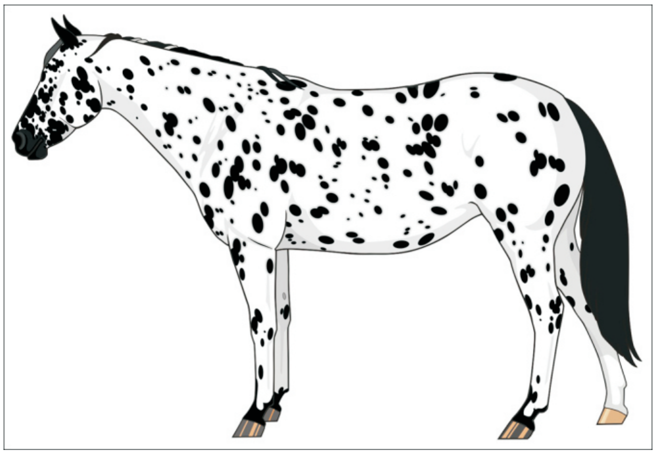
As can be seen on the chart, the extension gene - whether the horse is black or red - is the biggest factor in resistance to white. Horses that do not carry red (first group of three, shaded in tan) tend to have the least amount of white. The group of horses that are bay or black but carry chestnut come next (shaded pink), followed by the chestnuts (shaded green).

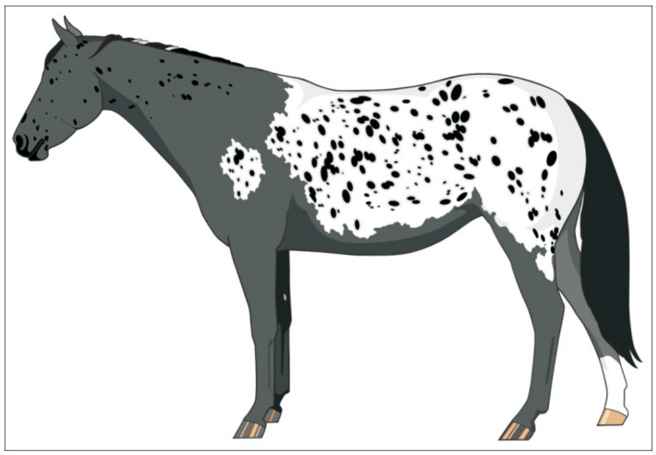
Within each extension gene group, the three possibilities are ranked by their agouti genes, with all-over black being the most resistant, followed by bays carrying all-over black (Aa) and finally by the pure bays (AA).

This explains why "flashy white" is so often associated with chestnut. It is statistically far more likely to find extensive white markings paired with that base color than with black. This is also why, in breeds better known for their conservatively-marked dark colors, unexpected chestnuts are often carry more white than either parent.

This has relevance to appaloosa patterns because preliminary research has shown that the suppressing action of black works there as well.

A good example is the pattern often referred to as "near-leopard". Some of the appaloosas classified as near-leopard may be, genetically at least, true leopards. Their black base color has effectively





The bottom image has the exact same pattern of body spotting as the top image, reduced to fit onto the hindquarters. The small, dense spotting gives the impression of a pattern too small for the horse, and is typical of a suppressed leopard.

The Mechanics Behind White Markings and Patterns

(with some help from my refridgerator magnet)

To better understand white markings and patterns on horses, it can help to know a little bit about how pigment develops in utero.

Usually my mare Sprinkles (or her color-changing cartoon alter-ego) illustrates concepts for us. But since she drew the line at lying spread-eagled on the ground, I'm going to use this guy.

Normally this horse magnet holds a list of important phone numbers on my fridge, but today he's going to help illustrate how horses get markings.

In mammals pigment in the skin and hair (melanin) is produced by cells known as melanocytes. These cells form along the dorsal line, and then spread from there to each side of the animal as the embryo develops.

Pigment-producing cells form along the blue line and spread outward in the direction of the arrows. Until those cells migrate to those areas, they are without color. The skin and hair are still there, of course, because their development is controlled separately. But without pigment-producing cells, that skin will remain pink and the hair white.

Since the last areas to "fill" are the ends of the limbs, disrupting the process stops the pigment-producing cells from reaching the bottom of the leg. If the migration of these cells stops before the hoof is reached, then that leg will remain its original unpigmented white. Just how much white depends on how far the cells were able to travel down the leg.

Face markings work much the same way, although the spread of the pigment is obviously not lateral like the legs. The face doesn't fill with color in a straight line from the ears down to the nose, but rather spreads toward the front of the face. That is what remains white if the pigment cells do not complete their migration.

Here the pigment migrating down the legs made it all the way to the end on two legs, but stopped short on both the left front and right hind. One those legs the underlying skin will be pink, rather than black. The hair will be white, instead of red or black. (The two forms of melanin, eumelanin and pheomelanin, give us the the two basic pigments - black and red.) The hoof will be shell-colored, since that's how horny tissue looks without pigment.

This is the basic mechanism at work with any kind of white pattern with underlying pink skin. Other genes can change the appearance melanin pigments, but truly white areas are the result of this kind of disruption in the migration of the pigment cells. Pigment never gets to those places. The different white patterns all have slightly different genetic mechanisms, so the nature of the disruption will vary. The gene for tobiano, for example, causes one kind of disruption, and a fairly reliable set of areas remain unpigmented. Another pattern will have a slightly different mechanism, and therefor slightly different areas of white. But the cause - the absence of pigment-producing cells - is the same.

shrunk the leopard pattern so that it no longer covers the entire body.

Even with the classic nose-to-toes type of leopard patterning, though, black leopards tend to have somewhat less white than a chestnut horse with the same level of patterning. They are more likely to retain black on their lower legs and black areas where their legs meet the body. They tend to have dark hairs in their mane and tail.

It appears that this same dynamic is at work with the "characteristics" that go along with the initial leopard complex (Lp) gene, particularly when it comes to skin mottling. Base color is not the only factor here, since appaloosas that are homozygous for leopard complex often have pronounced mottling. But it does appear that chestnut horses develop more extensive mottling (often more rapidly) than black or bay horses with similar patterns.

Implications for Artists

The fact that base color can change the expression of a pattern gene is important to artists because these altered patterns can give the impression that patterns are possible when they are not. In breeds like the Appaloosa, with large populations and a wide range of patterns, this is not likely to be an issue unless an artist is painting a family group. But for breeds where appaloosa patterning is found, but is rare or limited to certain families, it is possible that some patterns are not present.

Because chestnut horses are the least resistant to white markings and patterns, they will often show color even when they don't carry as much in the way of patterning genes. The downside is that chestnut horses often look more colorful, yet have fewer color-producing genes than a similarly marked black or bay horse. When crossed on solid mates, stallions like this one may not produce color as reliably as a black-based horse.

An good example is the Noriker breed. It is often said that appaloosa-patterned Norikers are always leopards and never blanketed. It is quite likely that the smaller pattern genes are not found in the breed. I have, however, seen a handful of near-leopards. All were black, and all had the small, dense spotting seen on suppressed leopard patterns. Knowing that black and bay can change the leopard pattern into something that can look like a blanket pattern - but yet isn't quite the same - prevents the mistake of placing a true blanket pattern on a breed where that might not be possible.

Right now that kind of accuracy isn't necessarily required. Many collectors, and many competition judges, are unaware that spotted breeds can have some patterns and not others. Even fewer can tell a "false blanket" from a true one. But genetic mapping (and the commercial tests that usually result) are progressing at a rapid pace, so that in the future we are likely to see patterns we currently lump into one general group (like "appaloosa" or "sabino") as separate and unique. Twenty years ago, it was enough to paint a horse as a tobiano or an overo. Competent painters no long paint generic overos; they paint frame overos or splash overos or sabinos or combinations of those. Customers know the difference and expect greater accuracy in regards to these patterns, so older pieces that did not take this into account now look dated. One day collectors may view generic appaloosa patterns this way. Staying ahead of the curve is important for finishwork artists who want to create timeless pieces.

In the Next Issue...

There is another way that base colors interact with the appaloosa patterns. In this case it seems the interaction is not with the patterns, but with the leopard complex (varnish) gene itself. That is the "bronzing" effect, and it will be the topic of the next issue's column.