## SO WHY DO FULL-SIBLINGS OFTEN HAVE SO LITTLE IN COMMON?

The answer to this question lies, in part, in the chromosomes. Chromosomes are the structures of DNA that pass genetic information from generation to generation. Horses have 32 pairs of chromosomes, one of each pair is inherited from each parent. But full siblings usually end up with very different combinations of their parents' chromosomes, the reasons for which I will try to explain.

The full-sibling quandary

During sperm development in a stallion, meiotic cell division results in the generation of sperm cells that contain only a single copy of each chromosome (haploid). The same situation arises during egg development in the mare. When fertilisation between a sperm and egg occurs, the chromosomes are paired up again (diploid), as a new individual is formed.

As an example, consider that a Sadlers Wells sperm cell will contain 32 single chromosomes. Some of those chromosomes will have come from Northern Dancer, the others from Fairy Bridge. Whether chromosome #1 in a given sperm cell came from Northern Dancer or Fairy Bridge is completely random. The same is true for the other 31 chromosomes. The distribution of the chromosomes occurs randomly during cell division. This means that most sperm cells are genetically different even though they arise in the same individual.

For example, all of the 32 chromosomes in one individual sperm cell might have come from Northern Dancer, while the next sperm cell might have 30 chromosomes from Northern Dancer but numbers 11 and 18 from Fairy Bridge. The next 5 sperm might all have 16 chromosomes derived from each parent, but completely different sets of 16. What this amounts to is a staggering number of possible combinations of chromosomes, each giving rise to genetically different sperm cells. As there are 32 different chromosomes, each with 2 different possibilities (coming from either Northern Dancer or Fairy Bridge), therefore there are potentially $2^{32}$ [= 4,294,967,296], or approximately 4.3 billion different combinations of Northern Dancer/Fairy Bridge chromosomes in the sperm of Sadlers Wells. As mentioned above, the same number of combinations arise during egg development in the mare to be bred to him, meaning there would be 4.3 billion X 4.3 billion, or 10,844,674,407,000,000,000 possible combinations of chromosomes in the progeny. To further increase genetic diversity, there are countless random

recombination or mutation events that can also occur within individual sperm cells. Simply put, the odds of producing a clone are truly monumental.

The set of chromosomes that each horse receives carries with it the complement of genes that define many aspects of its being. There are estimated to be in the region of 20,000 genes in higher mammals such as horses or humans, and it is unknown how many contribute to the racing ability of a horse. However, while certain simple traits, such as coat colour, are governed by a single, or a very small number of genes, racing ability is dependent on so many factors that it is undoubtedly governed by many genes (hundreds, possibly thousands?). Because these many hypothetical "racing genes" are scattered throughout various chromosomes, with each different combination of chromosomes comes a different mix of "racing genes".

Importantly, genes impact each other by turning each other off or on, a phenomenon known as epistasis. This means that if two individuals differ only by a small number of genes, the differing genes can have a domino effect on other genes, amplifying the difference between closely related individuals. In other words, even when the genotype (genetic information) of 2 horses is similar, the phenotype (racing ability) can, in theory, end up very different.

Therefore, the unique mix of chromosomes, and hence genes, that a horse is endowed with will have its own internal unique epistatic effects, resulting in a unique gene combination value in each individual. Unfortunately, for the frustrated breeder, the gene combination value for racing ability cannot be predicted, nor can it be transmitted from generation to generation because it is unique to the individual, and determined by how the genes interact with one another (epistatic effects) as well as what genes are there in the first place.

Chromosomal distribution and epistasis help explain why closely-related (genetically similar) horses can have greatly different racing abilities. They also shed some light on how horses from very unusual (or weak) genetic backgrounds can unexpectedly end up wearing the roses.

Despite all of these uncertainties when planning a mating, there are steps that can be taken to improve your odds of producing a top racehorse. While approximately 3% of all thoroughbreds become stakes winners, several stallions and successful breeders have much higher strike rates than that. There is no doubt that clever, informed breeding practices can maximise your success. The content of this article merely serves to explain some of the reasons that breeding the best to the best doesn't always yield the best