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# Link between specific genes and racing performance

THOROUGHBRED breeding and racing has a prominent practical side to it where horsemanship, husbandry and management count for a lot.

It is sometimes easy to forget about the breeding of a useful horse and the underlying basis for its success . . . the genetic hand of cards it has been dealt.

The heritability of racing performance has been broadly estimated at anywhere between 35% and 63%. The curious lower figure of 35% has also been popularly distilled from scientific studies to represent the contribution made by genetic factors to racing performance. Where it suits, this is sometimes dismissively quoted as being 'only' 35%. Only?

Putting this into perspective, 35% is a big contribution for a single factor to make. The multitude of other variables including, nutrition, training, jockeyship, veterinary care, soundness and injuries, environmental factors affecting development during growth and let's not forget luck, have to share the other 65% portion between them.

In reality, it is difficult to attach a retrospective, scientific value to the overall contribution of genetics simply because if a foal has not been dealt a decent hand of genes in the first place it will not respond adequately to the other factors in any case.

We all know there are no-hopers that have been dealt such a bum hand that no amount of fine grains, medicines and swimming pools will help them.

Whichever way you look at it, genetics is a big player in influencing the way a horse will perform. If that were not the case then we would not have fine sires and poor sires, good dams and bad dams.

Our latest research, reported in Mitochondrion, is an attempt to provide useful, usable data based on bona fide genetics principles and research, which have practical relevance to breeding and racing and are not part of a merely academic exercise.

The ability to identify key genetic phenomena and metabolic processes involved in performance and to manipulate or manage the underlying sizeable genetic contribution to greater effect could translate to significant improvements on the racetrack.

We focussed our attention on a group of candidate genes, which we suspected of being of importance to racing performance. These genes are normally inherited only from the dam and can therefore be transmitted down the generations solely via the female or dam line.

They can be passed to a colt and function within him, but he is unable to transmit them to his offspring.

These genes are the mitochondrial genes, and are significant as they are involved in energy release in muscle cells through the production of an energy rich chemical called ATP.

In April, 2006, **Dr Stephen Harrison** of **Thoroughbred Genetics Ltd** and **Juan Luis Turrion-Gomez** of the **University of Salamanca** published a peer-reviewed scientific paper in the international journal *Mitochondrion*, which shows, for the first time, an association between specific genes and racing performance. Here, they describe the practical aspects of their findings.

Although there are only 13 of these genes contributing directly to energy release, the mitochondrial DNA (mtDNA) molecule, of which they are part, has an estimated 10,000 copies per muscle cell. That means there are potentially 9998 more copies of mitochondrial genes per cell than there are copies of genes inherited via the 'standard' parental route. This potentially makes them major players in the determination of performance characteristics.

The role of these genes in the athletic performance of humans has already been shown and their importance has been further established via their links with a range of medical conditions in humans including muscular disorders, heart muscle problems and exercise intolerance.

The mitochondrial genes form part of larger respiratory complexes, which also include genes transmitted by both sire and dam via the chromosomal route.

However, as the study of differences in the performance of varying female families is a well known feature of thoroughbred breeding, it was our objective to determine if there was variation in the genes carried on the mtDNA by different female lines and whether this resulted in any obvious performance differences.

We selected DNA samples from a group of 1000 thoroughbreds, and these covered the majority of European, US and Australian female lines in existence.

In the lab, we multiplied the genes of interest in each sample millions of times to provide genetic material for further analysis and applied DNA sequencing and a scoring process called SSCP to check whether different versions of the genes were present in the various female lines.

Eight genes had a number of variants distributed between the different families. Scoring the variant of each of these genes in each horse and combining the information allowed us to assign all horses to one of 17 genetic groups or 'haplotypes'. Using a unique classification system, each group was assigned a Roman numeral. As mtDNA is principally inherited via the maternal route, we would expect all members of the same family to have the same haplotype. This was not the case.

In our analysis of these genes in horses from 33 different female families, we identified 28 'incorrect' sub-branches carrying mtDNA that would not have been expected from pedigree or studbook information. These anomalies were spread over 19 of the 33 lines (58%) examined.

Naturally, to allow observation of performance trends within families and haplotypes, it was important for us to be able to identify those lines, which showed irregularities.

However, the objective of this work was not to correct the thoroughbred studbooks (a futile and impossible task) but to assess the potential role of the mtDNA in performance.

Clearly, it would have been impractical to analyse numerous representative horses (amounting to hundreds) of each haplotype on treadmills, taking biochemical measurements.

To circumvent this problem we employed a study that could essentially link specific gene versions potentially affecting muscular energy release and stamina with actual racetrack performances, as if it were our own physiological trial.

Using the data from the DNA analysis, we were able to determine the probable mtDNA haplotype of members of the current international thoroughbred populations and to make corrections when necessary. We were also able to assign haplotypes to past winners of major horse races.

The complete study took into account 2YO, 3YO and Weight for Age races covering France, Ireland, USA and Australia but the paper reported in the journal covers UK 3YO races run between 1954 and 2003. The shortest race was the 1400m Greenham Stakes and the longest the 2800m St. Leger.

continued on page 44

## Genes and Racing Performance continued from page 42

Dividing the percentage of wins by a haplotype by the percentage of its predicted occurrence in the general population provided a success or Race Index (RI) for the haplotype for each race.

Five haplotypes, accounting for 51.6% of the total 3YO population, showed significant correlations between RI and race distance. Three haplotypes II, XV and XVI leaned towards shorter distances and Type XI and Type IV to longer.

The order of racing merit among these haplotypes changed depending on the distance of race under consideration and the age group looked at. Similarly, some haplotypes showing useful Race Indices or stamina leanings at 3YO did not figure prominently at 2YO and vice versa.

In all, when other racing ages were considered, the number of haplotypes showing stamina bias rose to 11, covering 80% of the population.

The importance in recognising the difference between family performance characteristics and those of the haplotype is clear. The term 'haplotype' is not synonymous with 'family' and the latter is an unreliable quantity.

Nowhere was this more apparent than in the example of past Melbourne Cup winner Jeune, whom we were lucky enough to study. The haplotype associated with a large proportion of his family members has a clear leaning towards 1000m performances at 2YO, but Jeune actually turned out to carry a gene grouping that is associated with greater stamina levels at Weight For Age level and in other diverse family lines.

That is, his particular family sub-branch had been incorrectly recorded at sometime in the past. That may not have been a bad thing and probably supported his general breeding to a greater degree. Had he been of the expected haplotype, it is conceivable he might have achieved nothing.

Horses with errant pedigrees like his have enabled us, through the construction of databases, to determine which gene versions other horses should be carrying and what their basic limitations might be.

The frequency of each haplotype present in the population varied according to the age group looked at, although some were the same. There are also differences in the distribution of the different genetic types between some of the countries, which affects their relative success.

The European percentages seem to have remained relatively constant over 100 years, but it is noticeable in the US and Australian systems that there have been some dramatic shifts.

There is probably a difference in the emphasis of stamina. For instance, in the USA and Australia, there are fewer longer stamina tests and this puts a different emphasis on haplotype requirements.

In the paper, the wider range of running distances available at 3YO in Europe certainly permits greater opportunity for some of the haplotypes to demonstrate clearer stamina leanings.

However, rather than suggesting breeders select mares of specific haplotype, the broader results indicate there are specific niches for a number of haplotypes in racing and that it is possible to manage mares more effectively to make the most of basic stamina attributes affected by the genes studied.

Many thoroughbreds are a mixed genetic bag made up of a range of varying genetic and stamina components. Genetic variability in a horse makes it less of a consistent breeding and racing proposition.

The policy of mating of sires and dams of different stamina potential contributes to this and leads to a mixture of un-co-ordinated genes in the progeny.

There has been no obvious common selection goal in thoroughbred breeding. This is due in part to commercial pressure, but the range of racing distances available also provides people with an excuse not to bother. The result is unpredictability.

Improvement in the co-ordination of the genetic components of stamina is a key to improving success.

The simple mode of inheritance of the mitochondrial genes, down the dam line, means they are easier to manage than chromosomal genes. This, together with their variable stamina preferences, makes them a useful framework around which to build more co-ordinated breeding programs based on stamina.

Using DNA analyses in conjunction with our databases describing haplotype expectations and trends, we are aiming to co-ordinate genes that support common stamina and precocity objectives. For example, through the co-ordination of breeding to complement the stamina specific mtDNA carried by a mare.

Breeders are on a different side of the fence to those in the industry who are presented with a ready-made horse with which to work. There is no question of breeders apportioning an importance value to genetics. The need to supply the raw racing material and to them, the way the genetic cards are dealt, before the other factors come into play, is everything.

Certainly, there are a number of genetic variables at work but if we are able to ensure that some of the basics are in place, then success has a better chance of following. ■

*JEUNE (GB) (Kalaglow-Youthful by Green Dancer), the haplotype associated with a large proportion of his family members has a clear leaning towards 1000m performances at 2YO, but Jeune actually turned out to carry a gene grouping that is associated with greater stamina levels at Weight For Age level and in other diverse family lines.*