PSSM1 and The Purebred Gypsy Cob- A Personal View
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Genetics is a fascinating topic and there are new and exciting developments available to discover every day. Unfortunately, PSSM1 in Gypsy Cobs (GCs) wasn't a discovery that filled many hearts with delight- unless you happened to be in that fortunate group of cob owners who had PSSM1 clear horses, and who then fell into a marketing bonanza.

Some of us watched the unfolding of the spread of information about GC's and this recently discovered gene within the breed with a bit of amazement, as super horses with fabulous genetics were ruthlessly and rapidly culled from the GC gene pool. The cries of 'irresponsible breeding' echoed around the cob world- well at least in the USA.

Fortunately, in NZ there was a more measured response and people took some time to look at the research and the context. However, PSSM1 is a topic that continues to bedevil the GC breed both here and overseas. *This is my personal take on it, informed by both observation, experience, and the reading of available research.*

Firstly–Quarter Horses. Most of the research about PSSM1 started and continues with Quarter Horses. There is no doubt that PSSM1 in QHs is a horrible, debilitating condition. Quite rightly, Quarter Horse associations have attempted to breed it out of the QH population. Many of the expressions of horror from people regarding breeding PSSM1 positive horses comes from those with experience of PSSM1 in Quarter horses and associated breeds.

Secondly – QHs are not GC's. A very good article by Jess Morton (2022) clearly outlines the differences in the muscle structure of horses developed over generations almost exclusively to sprint fast over short distances compared with horses who have been bred to work hard for long hours at steady speeds. The physiology is different. This is also true in human athletes with differences in muscle physiology and types between sprinters and endurance athletes. A horse is a horse is a horse but actually- when we breed selectively over decades they aren't. When you select for specific traits, you also breed in or out other characteristics, and the response to genetic mutations will not necessarily be the same across breeds developed for different purposes.

Even before genetic testing, GC's were selected for hardiness. Genetic selection was occurring by the Gypsy breeders without the use of a lab. Did this mean if horses were symptomatic with PSSM1 they contributed less to the gene pool? Very likely. So, we start with a group of horses with physiology very different to the breed which have major PSSM1 problems and bred by people who selected for hard work and soundness.

We know that a high proportion of GCs carry a PSSM1marker - it's said to be up to 40-60 % depending on the location. Do we see a lot of GCs that are tying up, unable to work and suffering as the QHs do? No, we don't. Valberg, (2020) states many draft horses with a PSSM1 marker are asymptomatic and 'Clinical signs occur most often in horses fed high-grain diets, exercised irregularly, with little turnout'. Horses with two markers (homozygous) are also more likely to show symptoms. If purebred GC's carrying a PSSM1 marker were as devastatingly affected as we might be led to believe, about half of our horses would be dysfunctional. In NZ, four of the most competed GC's have a PSSM1 marker. They are fit, competed relatively hard and successful in their disciplines. I have yet to encounter a purebred GC in NZ with a proven PSSM1 related problem.

Research seems to suggest that other genes and the environment are involved in producing symptoms rather than just the PSSM1 marker. This is the principle underlying the academic field of epigenetics where gene activity is being manipulated by the activity of other genes. RYR1 is one gene that may turn on something that causes horses to be symptomatic and has been found in QHs with severe symptoms (McCue et al, 2008). This idea is further supported by the evidence that only a very few out of many GC's carrying the PSSM1 marker show any clinical symptoms. Until we can identify what causes a horse to be symptomatic (and most purebred GCs are not), there seems to be no good reason to exclude from the gene pool asymptomatic horses who likely lack whatever other gene is present to turn on the PSSM1 symptoms.

Most horses with PSSM1 can generally be managed effectively according to Young (2020) and Valberg (2020). While there do not appear to be any purebred GC's in NZ with symptomatic PSSM1, it is still useful to heed the advice for PSSM1 management as sensible for all cobs. That is keeping them in regular exercise along with a diet of low non-structural carbohydrates with relatively high fat levels. NSC rates can be calculated by adding the % of sugar and starches listed on the feed together. For horses with PSSM1 this should be below 12%. That there are no known purebred gypsy cobs in NZ with PSSM1 symptoms when most of NZ hay is based on feeding cows to produce high quantities of milk and many of our pastures are high octane diary grass, reinforces that carrying a PSSM1 marker in purebred GC's is not an issue. We should also not assume that horses showing symptoms that may be associated with PSSM1 are symptomatic because of a PSSM1 marker. Other conditions can cause similar symptoms, and these should be considered and excluded for the welfare of the horse.

How do we move forward without removing horses from the gene pool that have otherwise fantastic genetics to offer- that have superb temperament and breed characteristics? In my opinion we don't decide not to breed from them based on a genetic marker. We have them under saddle or in harness and if they are then symptomatic, we don't breed from them. IMO we should be doing this with all horses we breed. We should consider were their parents proven to be fit for purpose and we should breed horses with a genetic history of being fit for purpose. Then if the horse itself is asymptomatic, there should be no reason not to breed from it.

A further note of caution is warranted when deciding to eliminate GCs with a PSSM1 marker as contributors to the NZ GC gene pool. In NZ our access to diverse genetics is limited by our geographical location and the prohibitive cost of importing both horses and semen. While we have a reasonable selection of genetics available, by removing what might well be a significant proportion of GCs from the gene pool we risk ending up with significantly inbred horses. This has recently been identified as a future problem in thoroughbred horses. "in-breeding increases the chances of off-spring inheriting recessive traits … this may weaken the biological fitness of the population and hinder its ability to survive-a circumstance known as inbreeding depression' (Morton, 2022). We need to be careful not to breed ourselves into a genetic bottleneck!

Word of warning. We shouldn't breed horses at increased risk of becoming symptomatic. I don't believe in breeding positive GCs for example, to horses that have Quarter Horse in the background. I think this is a recipe for problems. Other horses with a high proportion of fast

twitch fibres may also be more inclined to be symptomatic although there is no evidence to support or refute this idea.

So, breed with care, have your horses under saddle, in harness or doing some form of work to ensure they are sound and suitable for the disciplines you are aiming them and their progeny at. Keep them fit, of an appropriate weight and on an appropriate diet. Plenty of other soundness issues such DCD, can also have genetic influences, and are not found unless the horse does something beyond the paddock gate. Soundness is proven through performance- not just through a genetic marker written on a piece of paper.

Disclaimer: the thoughts put forward in this article are entirely my own and do not necessarily represent the viewpoint of any association.

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