

Training the Untrainable:

EIPH and Horse's Lungs



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So are winners born or created and how important is physical training? In my view the best racehorses are born with or without potential. It's true that a lot can go wrong from the moment a stallion and mares genes mix to produce an embryo that will grow into a foal. Often underestimated is the impact that the environment within the mare has on the development of the foal. For example, the genes may be saying "straight legs" but other factors such as stress on the mare, infections, diet, the condition of the uterus, may well modify how that message is "interpreted" leading to a foal with crooked legs. The impact of the uterine environment was perfectly demonstrated by some ground breaking studies by Professor Twink Allen at the Equine Fertility Unit in Newmarket, where he demonstrated that pony embryos transplanted into Thoroughbred mares resulted in large pony foals and that Thoroughbred embryos implanted into pony mares resulted in small Thoroughbred foals.

Once a foal is born, there is a long and potentially difficult path from birth to racing success, even with the right genes for performance. Diet, disease, trimming, shoeing and even luck all play a role. Then comes training. And here I am focussing on physical training rather than training the horse to run in company, quicken away from a group or go in stalls...what we might consider behavioural training. A recent scientific study from the University of Florida in the USA which looked at horses purchased at yearling sales in the summer for sale at 2-year-olds in training sales the following spring found that 37 out of 40 horses purchased became lame during training. Also interesting was the fact that "the frequency of new cases of lameness increased as the date of the 2-year-olds in training sales approached."

The aim of training should be to maximise the genetic potential of a horse. How much is a horse born with and how much difference does training make? Scientifically that's quite a difficult question to answer. My gut feeling is that training may add perhaps a quarter...so this leaves 75% of performance down to breeding or in other words, the genes. How do I come to this conclusion? Take a horse with a handicap rating of 70lbs with an average trainer and give it to an exceptional trainer, and the latter may be able to improve the horses rating by 15-20lbs. It's not uncommon to see a horse change trainers and increase by 10-20lbs, but to see a horse change trainers and go from a rating of 70 to 130lbs would be exceptional.

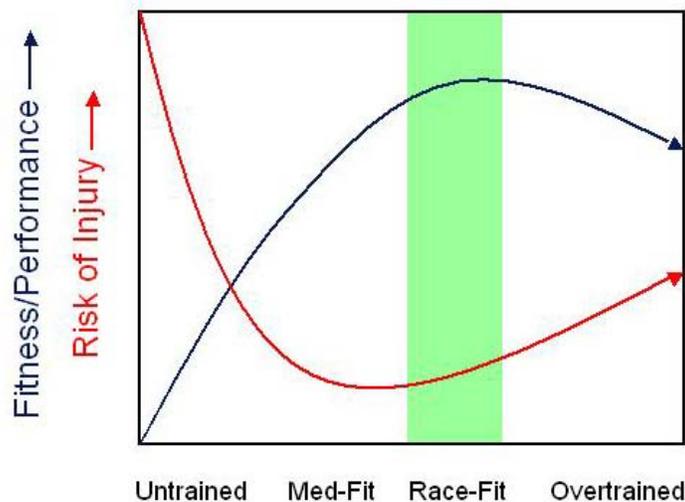
So I believe that elite horses are born, not created through management and training. That's not to downgrade the role of the trainer. Training has to be very important. How many untrained horses win races? But we also know that poor training can take a horse with the potential to win the Derby and turn it into one that never even gets to race and good training could take a horse with an expected rating of 60lbs up to perhaps 80.

Hence, knowing that training can improve poor horses, ruin good horses and vice versa, there can be no doubt that training racehorses is a challenge. Too low of a training load and the horse performs below expectations. Too high and you risk injury; particularly of course

musculoskeletal injury...injury to bone, cartilage, ligament, and tendons and to a lesser extent muscle. Getting it right for each horse is certainly a combination of art, science and skill.

Why is training horses such a challenge? Part of the problem is the way in which different body systems or components respond to training. With appropriate loading or “stress”, the locomotory muscles and the heart (which is of course also a muscle) have a tremendous capacity to adapt to repeated bouts of exercise...or training. However, the intensity and volume (amount) of exercise required to get these systems to adapt is high compared for example to the amount of loading required for healthy bone development. Thus there is a potential imbalance. The heart and locomotory muscles need relatively long durations of exercise at high intensities to cause them to adapt, but this amount of exercise loading is often in excess of what joints, bones and tendons need or are built to cope with.

The graph below illustrates that during training, the period where there is a high risk of injury is also the period when there is the greatest need for “stress” to increase fitness and performance.

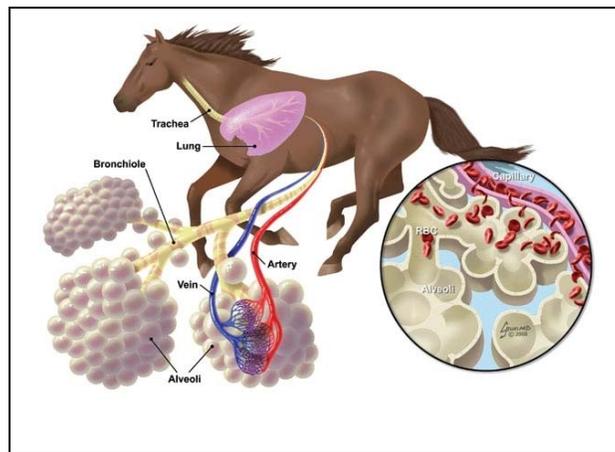


The graph above also indicates that eventually there is some balance achieved between muscle fitness, performance and musculoskeletal injury – the green zone. However, there is one body system – the respiratory system - that never attains this balance and for which exercise almost appears to be contra-indicated. In fact, it may come as a surprise to many to learn that the respiratory system of the horse does not respond to training. The amount of air an unfit/untrained horse moves in and out with each breath with each stride at the walk, trot, canter and gallop does not change when that horse is fit/trained. Many refuse to accept this, but at least three independent scientific studies, including one in my own laboratory, have confirmed this.

Is the lack of adaptation of the respiratory system of the horse to training a problem? Well it is a problem when that system is a limiting factor or weak point in the chain to get oxygen from the outside down to the muscles where it can be used. In unfit/untrained racehorses the heart is probably the limiting factor to performance. But with training the heart adapts, leaving the respiratory system as the “weakest link”, even though it is crucial to racing performance. Unless we want to race over distance of 1 furlong or less, the respiratory system is essential. Even in a 5 furlong sprint race around 70% of the energy to run comes from aerobic metabolism that requires oxygen to be brought into the body by the respiratory

system, to allow the conversion of energy in sugars, stored as glycogen within the muscles cells, into energy for locomotion in the form of ATP.

How do we know the respiratory system is the weakest link? Because if we can give the horse more oxygen to breathe than the normal 21% that is in air, say we increase it from 21% to 30%, we know the heart is able to transport this extra oxygen to the muscles. The muscles are able to use this extra oxygen and as a result performance is improved. (I think at this stage we can of course dismiss oxygen cylinders carried by the jockey with a tube running to the horse's nostrils.) Thus, the limiting point in the chain from nostril to muscle is in the respiratory system and to be more precise, in the deeper parts of the lung where the air containing oxygen passes into the lungs and is separated from the red blood cells in blood vessels on the other side.



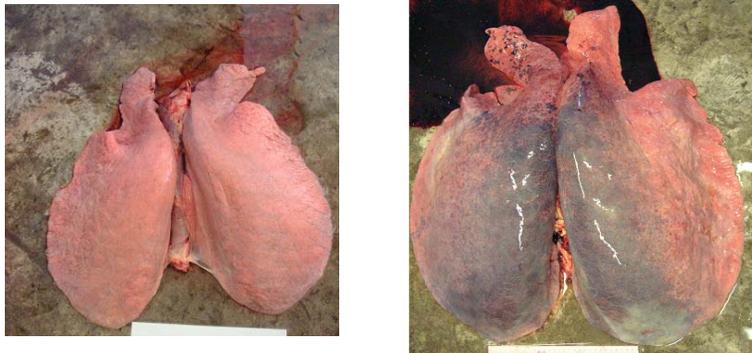
The horse's respiratory system showing that as the trachea passes into the lung it divides like branches of a tree into smaller and smaller airways ending in the "alveoli" where oxygen transfer occurs from the alveoli into the red blood cells (RBCs) passing through the blood vessels (artery). The blow up view on the right shows that at the microscopic level the artery divides into small capillaries that surround the alveoli. Oxygen exchange occurs from the alveoli to the RBCs passing through the capillaries across thin membranes that are 1/100th the thickness of a human hair. The membranes are fragile and can rupture allowing RBCs to pass into the alveoli resulting in exercise induced pulmonary hemorrhage (EIPH or "bleeding").

We also know how fragile and delicate the respiratory system of the horse is. This is usually not apparent from the outside, but only when we consider the microscopic structure of the lung. The horse's windpipe (trachea) is around 5-8cm in diameter, but as the windpipe passes deeper in the lung it begins to divide to produce smaller and smaller airways, much like a tree on its side, with the main trunk representing the windpipe. Each time an airway divides in two, the "daughter" airways are smaller than the "parent" from which they arose. When we get down to the level of the smallest airways, after perhaps 25 divisions, the airways are fractions of a millimetre in size. When the air gets to this point in the chain from nostril to muscle cell, it has to cross from the air space into the blood vessel. This is a passive process. There is nothing that can be done to speed it up as it depends on some fixed factors such as the total surface area available in the lung for oxygen to diffuse (move) across, which does not increase with training. (Incidentally, the total area for oxygen to diffuse across in the horse is equivalent to the area of 10 tennis courts!). It is also dependant on the difference in oxygen level between the air (high) and the blood vessels (lower). Oxygen moves from high to low areas. Finally, it depends on the thickness of the membrane separating the air in the air sacs ("alveoli") and red blood cells in the blood vessels ("capillaries"). So one option is to evolve to make this membrane, sometimes referred to as the blood gas barrier, as thin as possible. And this is exactly what has happened in the Thoroughbred to the point where this

membrane separating blood under pressure in vessels from the air in the airways is around 1/100th of the thickness of a human hair. Perhaps not surprisingly, these small membranes can rupture under the stress of exercise allowing the red blood cells (RBCs) to spill from the capillaries into the alveoli, which we term exercise-induced pulmonary haemorrhage (EIPH). So if the respiratory system does not adapt positively with training, the next best thing we can hope for is that it is not damaged by training. Unfortunately, this is not the case either. Studies from Japan demonstrated that Thoroughbred racehorses that were only trained at the walk, trot and slow canter still experienced rupture of small blood vessels in the lung. It is also true that the harder and more frequently a horse works, the greater the number of vessels that will rupture and therefore that this damage is cumulative. There is individual variation of course, with some horses being minimally affected and some horses affected to the extent that they are effectively untrainable. **And to dispel a myth; this damage (EIPH) is occurring even if you do not see blood at the nostrils or even in the trachea (with a 'scope) after exercise.**

What are the consequences of the rupture of these small vessels? Perhaps the best analogy is to drinking. The bad news is that a bottle of wine may kill off 10 million brain cells. The good news is we start with around 100 billion brain cells. However, after 10 years of heavy drinking the effects can begin to show! In this respect, the lungs are no different. However, the effects are noticeable much sooner.

How many small blood vessels are there in the lung? It's hard to be precise about this, but if we work on the fact that there are 40 generations of airways (divisions or branches) in the horse's lung and if each small airway had a small blood vessel around it, then this would give a figure of around 270 billion. How many break at Canter? At gallop? In a race? Impossible to estimate and again it varies between horses. But what we do know is that after time we can see scarring on the lung surface as a result of previous injury (haemorrhage).



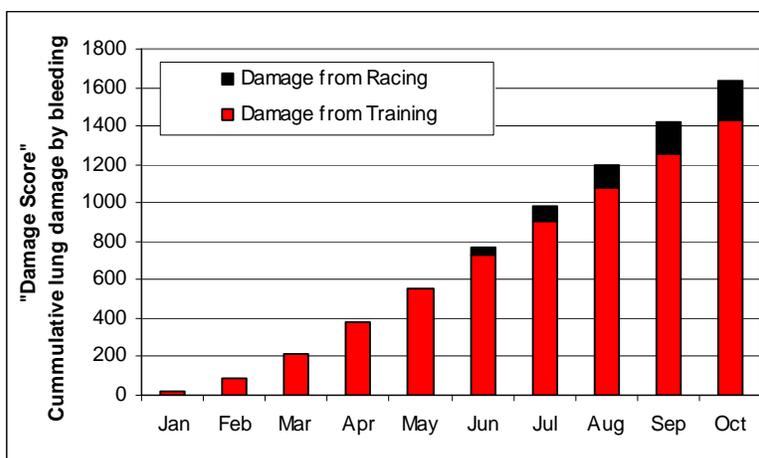
Contrast the relatively undamaged and unstained lungs of an untrained horse on the left with those on the right. Note the deep blue/grey staining showing areas of previous damage on the right, accumulated over many years of training and racing. Blood vessels that are damaged do not regenerate. Scar tissue forms and these areas cease to function normally. The more damage that accumulates, the greater the reduction in respiratory function.

One question that has always intrigued me is how much damage to the lung occurs as a result of broken blood vessels (EIPH) in racing relative to how much occurs in training? One way to try and work this out would be to give a "damage score" to different types of activity and then total up. For example, we could arbitrarily assign a value of 1 (i.e. low) for the damage caused by a slow canter and 3 for a fast canter....i.e. causing more damage. If we then scored a fast canter as 5 and a piece of work at home as 10, then we might put a value of 40 on the damage to the lung caused by a single race. Assuming 6 exercise days per week and therefore 24 exercise days a month from January to October, and starting with 48 days of slow canter in Jan-Feb, 36 days medium canter in Feb-Mar, etc, over this period our horse would have 48

bouts of slow canter, 108 bouts of medium canter, 108 bouts of fast canter and 52 pieces of work.

	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct
Days of Exercise>	24	24	24	24	24	24	24	24	24	24
Slow Canter	24	24								
Medium canter		12	24		12	12	12	12	12	12
Fast Canter			12	24	12	12	12	12	12	12
Gallop				4	8	8	8	8	8	8
Race						1	1	1	1	1

I'm then going to assume our horse ran 5 times in this 10 month period. When we total up the damage caused by training and compare it to that caused by racing, we may get a surprise. Although the damage in racing is more severe, the races are much less frequent and the total estimated damage by racing is only 12% of the total in this example. This leaves 88% of the damage to the lungs occurring during training – less damage per training day, but more training days. This type of approach shows us that perhaps it's training, rather than racing, that we need to be more concerned about as far as EIPH.



So if significant damage is occurring to the lungs as a result of training and racing, what options are there in management? There seem to be an ever increasing number of products marketed for bleeders. However, there are only two treatments that have been scientifically proven to significantly reduce bleeding in horses; Lasix and nasal strips.

Lasix and nasal strips actually both work in a similar way in reducing stress on the blood vessel walls. Lasix works by decreasing the blood pressure in the blood vessels inside the lung and hence decreasing the stress on the walls and reducing the number that rupture. Nasal strips also work by reducing the stress on the wall of the blood vessel walls, but from the air side.

Lasix is a type of drug known as a diuretic. When given to horses it “tricks” the kidneys into producing more urine than normal. This in turn removes water from the blood, reducing the volume of plasma (the watery part of the blood as opposed to the red blood cells) in the circulation. This reduces the blood pressure so that the tiny blood vessels in the lung are less stretched and stressed.

The nasal strip works on the other side of these blood vessels in the lung – the side that is in contact with the air. Nasal strips work by supporting the loose flap of skin behind each nostril. When the horse breathes in this skin is sucked inwards. The more this skin is sucked in the more effort the horse needs to make to move air into the lungs. Horses, unlike us, only breathe through their nostrils, and so any obstruction in this area can have a big effect. This effort in breathing in causes the walls of the tiny blood vessels (known as capillaries) to bulge outwards and in some cases break, resulting in the loss of blood into the air spaces and tubes of the lung. The nasal strip supports this skin over the nose and allows the horse to move the same amount of air in and out with less effort, placing less stress on the lung.

So two treatments. In scientific trials, they showed the same level of effectiveness in reducing bleeding. One is a drug and one is mechanical. Does it matter which one you use? On a one-off gallop probably not. However, with repeated use of drugs tolerance often develops. This may mean that over time you have to use larger and larger doses to get the same effect. Or alternatively, if you keep using the same dose then the effect you get becomes less and less. It is also not uncommon for drugs to have unwanted side effects with repeated use. The degree of dehydration induced by Lasix is also something to consider. Dehydration can have adverse effects on many systems, for example the digestive tract. Whilst to date no-one has looked at the effects of the dehydration resulting from use of Lasix alone on body systems other than the lung, trainers and veterinarians need to be careful to consider other possible factors that will increase dehydration further, such as hot weather, transport and sweating and decreased water and feed intake due to anxiety. The potential advantage of a mechanical device, such as the nasal strip, for treatment of bleeding is that it is almost certainly going to be equally effective each time it is used, tolerance is highly unlikely and there is no possibility of any side effects.

On the basis that each treatment works, is there any advantage to using both? The answer appears to be yes based on a study of horses racing in the USA. Even though both Lasix and nasal strips work on the blood vessel wall, severe bleeders still showed a further reduction in bleeding of 65% when they raced with a nasal strip and were treated with Lasix, compared to being treated with Lasix alone.

On paper, if you listed out the potential problems in training an animal where what one body system needs is what might break another body system, you would have to conclude that training horses is going to be extremely challenging. This is perhaps testament to the high level of skill that any moderately successful trainer clearly must have developed. Training clearly cannot be approached as a pure science and in fact there are some examples of very good scientists who have made poor trainers. But science can potentially help trainers understand more about how the different body systems of the horse respond to training and apply their skills more effectively.