Final summary released of research project

By Mary Durando, DVM, PhD

The objective of this study, conducted at the New Bolton Center at the University of Pennsylvania, was to compare performance with concentrations of a specific blood parameter related to heart damage and electrocardiograms in endurance horses competing in long-distance rides.

Hypothesis

Horses that are unsuccessful during endurance competition and/or have electrocardiographic evidence of heart dysfunction will have values for a selected blood parameter (cardiac troponin I, cTNI) that are significantly different from horses that compete successfully.

Summary

We have finished collecting and analyzing samples from three 100-mile and three 50-mile rides in three different regions of the country. The rides attended were:

-- 2004 Biltmore Estate Challenge FEI/CEI***/**

-- 2004 Fair Hill International FEI/CEI***

-- 2005 Bluebonnet Classic Arabian Horse Association Region 9 Championship.

The participating horses had blood drawn on three occasions: pre-ride, at a mid-point hold, and on completion of the ride. During the ride (at the hold) and at the finish they also had electrocardiograms performed to evaluate their heart rhythm.

The blood was analyzed for cardiac troponin I (cTNI), a heart-specific regulatory protein. This protein can be elevated in people with significant cardiac disease, and has also been found to be elevated in some elite athletes participating in endurance sports such as marathons, ultramarathons, triathlons and endurance bike races. The significance of its elevation in human athletes after competition, or whether it influences performance, is not known, however it does correspond to transient abnormal heart function seen with echocardiography (ultrasound of the heart).

We wished to evaluate this protein marker in competitive endurance horses to determine if there was a relationship to race performance and finish position, as the strenuous nature of their competition resembles elite endurance human athletes, who can have changes in heart function. We have completed assaying all samples for cTNI, evaluating the electrocardiograms (ECG), and gathering ride information (finish status, finish position, ride times, pull data). The data has now been collated and analyzed.

We were able to collect blood from a total of 118 horses from the 50- and 100-mile rides. In all, a total of 299 samples were collected, however we were unable to get complete data sets (three samples) from every horse, because of ride logistics and/or horses being pulled. One hundred of the 118 horses had pre-ride and end-ride samples obtained. If the horse was pulled from the ride, a sample taken at that time was used as the "end of ride" sample.

Seventy-nine of the 118 horses sampled (67%) completed the rides. Horses were eliminated for a variety of reasons (Lame, Metab, RO, OT), but only one horse was eliminated specifically for cardiac reasons (an arrhythmia, atrial fibrillation). This horse had a normal cTnI at baseline, however at the time of elimination it was very elevated over our normal range, and one of the highest measured at all three competitions. No other abnormalities on the three-minute ECGs were seen.

The mean pre-ride cTNI for all horses was within the normal range for horses. Overall, combining rides and distances, cTnI increased significantly over baseline by the end of the ride. This was true of horses that were pulled, as well as those that completed the ride. Therefore, strenuous exercise seems to cause an increase in cTnI. Overall, there was no difference in end cTnI from horses that were pulled, compared with those that completed the rides.

When just the 50-mile rides were evaluated, the horses that were eliminated had higher mean cTnI values than those that finished the ride. Interestingly, the horses that finished in the top 10 for the 50-mile rides had higher mean cTnI values than those that finished behind the first 10 finishers, but this was just a trend, and not statistically significant. Some investigators have seen this change in people, and postulate that the ones who finish faster are more competitive and try harder, thus damaging their heart slightly. It must be emphasized that change was seen in a relatively small number of horses. It would need to be evaluated in a much larger number of competitors before any conclusions can be drawn. Interestingly, the ride sampled had the most influence on the increase in cTnI. The Fair Hill ride had a significantly larger increase in cTnI than the Texas ride, and also had the highest percentage of horses with an abnormal cTnI at the finish. We do not know the reason for this, or its significance at this time. Fair Hill also had a higher percentage of horses eliminated for metabolic reasons, so whether it was a more strenuous ride, or the horses were more competitive and pushed harder is not known.

There was no relationship between the specific horses pulled for metabolic reasons and those with higher cTnI concentrations. This would be interesting to evaluate in other rides considered to be "difficult" rides or very competitive rides, to see if it could be confirmed with larger numbers of horses.

Once again, it is emphasized that these findings are preliminary data that deserve further study, by evaluating more horses at rides of varying difficulty. It does appear that strenuous exercise causes elevations in cTnI. This may be influenced by the terrain and environmental conditions of the ride and the competitive nature of the horses. Whether these horses have other evidence of heart damage, another reason for the increase in cTnI, or how long the elevation lasts is not known. Performing echocardiography (ultrasound of the heart) on these horses may help to determine if they have abnormal function.

Further studies are warranted to determine echocardiographic findings in horses with elevated cTnI to better correlate speed and finish position with cTnI, and to study the effect of fitness, ride severity and weather conditions on cTnI.

This report was submitted by Mary M. Durando, DVM, PhD, DACVIM (internal medicine), research assistant professor in the section of sports medicine at the University of Pennsylvania's New Bolton Center. Co-authors: Eric K. Birks, DVM, PhD; Todd Holbrook, DVM, DACVIM (internal medicine); Meg Sleeper, VMD, DACVIM (cardiology).