

Report on the investigation and review of the seven sudden deaths on the Hollywood Park main track of horses trained by Bob Baffert and stabled in Barn 61

Executive Summary

The investigation into the cluster of seven sudden death fatalities of horses trained by Bob Baffert, stabled in barn 61 at Hollywood Park, and training or racing on Hollywood Park's main track has not identified a definitive explanation for the highly unusual sudden death clustering. There is no factually based explanation as to why these sudden deaths clustered in Baffert's barn at Hollywood Park and not at any other tracks where Baffert stables, races and trains horses with similar training and veterinary practices. There is no evidence whatsoever that CHRB rules or regulations have been violated or any improper activity played a part in the sudden deaths. Except for the clustering of sudden deaths in Baffert's barn at Hollywood Park, which is undeniably exceptional, as will be discussed later, the cases were not atypical sudden deaths associated with racing and training. Throughout the investigation Bob Baffert, his employees and attending veterinarians were cooperative and provided all the information that was requested.

Supervising Investigator Rick Ameva was in charge of the investigation for the CHRB. Racing Safety Steward Jeff Salmon oversaw the environmental toxicology testing under the supervision of Executive Director Kirk Breed. Equine medical director Dr. Rick Arthur advised the CHRB throughout the investigation, liaised with UC Davis School of Veterinary Medicine and CAHFS faculty and staff, wrote portions not attributed to others, and compiled the final report.

Introduction

Equine sudden deaths in racing and training are not uncommon and have been reported to be between 3.5% to 19% of all fatalities depending on country and activity (Lyle). Sudden deaths at CHRB facilities have run between 4-12% of all racing or training fatalities for Thoroughbreds over the last 6 fiscal years in California. The average is 7.8%. The rates have risen in recent years because the numbers of sudden deaths have increased and the numbers of total deaths have fallen. Neither the CHRB nor CAHFs have reported sudden deaths as a stand-alone category even though sudden death fatalities from the CHRB ?CAHFS necropsy program provided the bulk of the cases in a retrospective, international, multi-institutional review of sudden deaths in Thoroughbred racing in 2011(Lyle).

On November 4, 2011, Case #1 died suddenly while galloping at Hollywood Park. There was nothing unusual about the case. CAHFS necropsy lab was notified per protocol, the carcass transported to San Bernardino and the necropsy proceeded routinely. It was the last Baffert sudden death handled in a routine manner. On November 26, 2011, Case #2 died after finishing second in a race at Hollywood Park. Then CHRB official veterinarian contacted the CAHFS and specifically noted on the necropsy submission form that this was the second case for the same owner and trainer. Dr. Bailey contacted the equine medical director. Within a few hours of Case #2's death the equine medical director notified key CHRB personnel including Chairman Keith Brackpool, Vice-chairman Davis Israel, Medication and Track Safety Committee chair Bo Derek, and executive director Kirk Breed, of the unusual circumstance of the same owner/trainer with two sudden deaths in a 3-week period. The sudden death of Case #3, the third horse from the same owner/trainer in 60 days, led to ongoing discussions and review of sudden death necropsy, drug testing and toxicology procedures. Even at this time, the racing community was very much aware of the unusual nature of the sudden deaths in Baffert's stable at Hollywood park. In each case after Case #1, the equine medical director discussed and monitored each case with CAHFS pathologists, toxicologists and Dr. Stanley from the Maddy lab and updated CHRB executive director Kirk Breed. The frustration and interest in sudden deaths in the necropsy program preceded the sudden death Baffert cluster. In a number of sudden death cases, a definitive diagnosis can be elusive. There is an absence of substantial cardiac pathology in a number of sudden deaths presumed to be of cardiac origin. There is speculation that conduction abnormalities or cardiac arrhythmias may be the cause of death without pathological changes (Physick-Sheard). Discussions about sudden death necropsy procedures, especially in regards to toxicology and drug testing, continued throughout the Baffert sudden death cases and are ongoing. Baffert was aware of the strong interest the CHRB had in the sudden deaths in his barn shortly after Case #3's death from a number of discussions with the equine medical director. A large number of horses in Baffert's stables at Santa Anita and Hollywood Park were sampled for out-of-competition testing in the spring of 2012, after Case #3's death. On March 14, 2013, shortly after Case #7 died suddenly at Hollywood Park, the equine medical director advised executive director Kirk Breed that even though the ongoing medical review had not revealed nor suggested any improper activity, the CHRB should conduct an official investigation. The executive director ordered chief investigator Bill Westerman to precede with an investigation into the Baffert sudden deaths. Chief

Westerman assigned the case to supervising investigator Rick Ameva. A review of sudden deaths in Thoroughbreds at CHRB racing and training facilities was conducted as part of the Baffert sudden death investigation.

Sudden Deaths in Racing and Training:

Sudden deaths are most often defined as acute collapse and death in a closely observed and previously apparently healthy racehorse. This definition will include a number of conditions that are musculoskeletal or accident related in addition to medical causes of death. CAHFS has used euthanasia administration to identify sudden deaths. Though reported on the necropsy submission form (CHRB-72) the CHRB does not record in its databases whether euthanasia was administered. Regardless, it is not uncommon for a horse *in extremis* to be administered euthanasia to hasten death for humane reasons. Those cases would not be categorized as sudden deaths. The underlying interest in sudden deaths is horses dying for medical reasons rather than trauma (accidents) or musculoskeletal failure. For that reason, musculoskeletal explanations for sudden deaths, which are well documented in the CHRB/CAHFS necropsy program, such as pelvic fractures and accidents, have been excluded. CHRB records with adequate detail for analysis exist back to July 1, 2007. Those records were reviewed retroactively and sudden deaths identified. After excluding non-Thoroughbred fatalities and a small number of Thoroughbreds that were not racing or training (a few horse die suddenly in their stall or barn area unrelated to exercise) there were 78 thoroughbred sudden deaths after racing or training that are not attributable to musculoskeletal injuries or accidents. None of the excluded horses by breed or non-exercise are related to the Baffert stable.

The rate of sudden death incidents for Thoroughbreds while racing can be calculated by dividing starts by the number of sudden deaths. Starts and starters are well documented for Thoroughbreds. Between July 1, 2007, and June 30, 2013, there were about 202,149 Thoroughbred starts in California and 23 sudden deaths while racing or shortly thereafter resulting in an average of 1 Sudden Death for every 8789 starts racing. This is within internationally reported sudden death fatality rates (Lyle). There are 20 trainers who have had sudden deaths while racing over the 6-year time period reviewed. Three trainers, Doug O'Neill, Keith Cragmyle and Bob Baffert, have two each; the other 17 trainers have one each.

Training sudden deaths are more difficult to quantify. Between July 1, 2007, and June 30, 2013, there have been 55 Thoroughbred sudden deaths while training. The training category is not differentiated by the level of training –working, galloping, jogging, ponying, etc. While the number of recorded works can be retrieved, we really have no reliable information about other training activities. In the Baffert situation 4 of the 5 training sudden deaths under investigation were galloping and only one was working. This is somewhat similar to the ratio of gallops to works in the average stable. Taking a rough estimate of 4,000 Thoroughbred horses training every day at CHRB racetracks, that would mean there are 1,460,000 Thoroughbred training days each year, or, over the 6-years between July 1, 2007, and

June 30, 2013, 1 sudden death for every 158,000 Thoroughbred training days. There are 43 trainers with sudden deaths while training over the 6 year time period from July 1, 2007 to June 30, 2013. Bob Baffert had six including a sudden death at Santa Anita during training in July 2010, Steve Sherman 3, Jeff Bonde, Jorge Gutierrez and John Sadler have two each; the other 38 other trainers have one each.

There is very little data on sudden death training fatalities outside of California. The CAHFS post-mortem examination annual report does not specifically tally sudden deaths nor does the CHRB annual report. The CHRB annual report focuses on track and activity; the CAHFS report provides pathological findings by organ system. A sudden death could be reported under cardiovascular as cardiac failure, respiratory as EIPH, or, as in the case in one of Baffert’s cases, as neurological under equine protozoal myelitis. The CHRB-CAHFS Post-mortem Program Annual Report can be accessed at <http://www.chrb.ca.gov/veterinary.html> under Post-mortem Examination Report by fiscal year.

Eliminating musculoskeletal causes, usually pelvis fractures, the CHRB racing and training sudden deaths for Thoroughbreds from July 1, 2007, to June 30, 2013, are as follows:

Fiscal Year	Racing	Training	Total
07-08	3	5	8
08-09	3	7	10
09-10	3	5	8
10-11	7	12	19
11-12	3	15	18
12-13	4	11	15
Total	23	55	78

The sudden death fatality data was analyzed by Dr. Peta Hitchens, a post-doctoral fellow in epidemiology at the UC Davis School of Veterinary Medicine. The data indicate racing sudden deaths have stayed essentially the same. Rates of racing sudden deaths did not significantly increase over the 6 year period (IRR 1.14; 95% CI 0.90, 1.44; p=0.296). The training sudden deaths did significantly increase over the 6 year period (IRR 1.28; 95% CI 1.10, 1.50; p=0.002). The fiscal years 10-11 and 11-12 had statistically significantly greater incidence of death than 07/08. FY 12-13 was not significantly greater statistically, but trending so (p=0.064). Using race starts as the surrogate denominator for training days, rates of total sudden deaths increased significantly by about 24% (range 9-41%) per year over the 6-year period (IRR 1.24; 95% CI 1.09-1.41; p=0.001). Using the same criteria for sudden deaths (thereby excluding two with pelvic fractures), in the first 4 months of FY 13-14 there have been 3 sudden deaths, 2 racing and one during training. Starts in FY 13-14 are similar to the last few years but running 20-25% below FY 07-08 to FY 09-10 averages.

Intuitively, the eight sudden deaths in Baffert’s stable—the 7 sudden deaths in this review + an earlier sudden death at Santa Anita in 2010-- are obviously significant, but are they significant statistically? Using all sudden deaths for Baffert (8 deaths, 2512 starts) there is an incidence of 3.18 deaths per 1,000 race starts (95% CI 1.37- 6.28). For comparison, all sudden deaths for non-Baffert trained horses (70 deaths, 199,637starts) have an incidence of 0.35 deaths/1,000 race starts (95% CI 0.27-0.44). Baffert

trained horses have a 9.08 (95% CI 4.37, 18.88; $p < 0.001$) times greater incidence of sudden death during racing or training than horses not trained by Baffert. Examining the 7 sudden deaths over 24 months of FY 11-12 & FY 12-13, the results are even more dramatic.

Looking only at racing sudden deaths for Baffert (2 deaths, 2512 starts) there is an incidence of 0.80 deaths/1,000 race starts (95% CI 0.10-2.88). Racing sudden deaths for non-Baffert horses (21 deaths, 199,637 starts) has an incidence of 0.11 deaths/1,000 race starts (95% CI 0.07-0.16). Baffert trained horses that experienced sudden death during a race have a 7.57 (95% CI 1.77-32.28; $p = 0.006$) times greater incidence of sudden death than horses not trained by Baffert.

Summary of Necropsy Findings:

Dr. Uzal's necropsy report and individual necropsy reports are attached.

Between Nov 4, 2011 and March 14, 2013, a 16 month period, Bob Baffert had 7 horses die suddenly while racing or training. All occurred on the Hollywood Park main track. None of the sudden deaths occurred at Santa Anita or Del Mar even though there were as many horses stabled at Santa Anita or Del Mar as at Hollywood Park. The main stable at Santa Anita or Del Mar maintained roughly 40% of horses trained by Baffert at CHR training facilities while the Hollywood Park barn had about 60%. Hollywood Park is Baffert's second string and is where he sends his horses to be started or restarted after layups. The Hollywood Park stable is run by assistant trainer Mike Marlow. Drs. Keith Latson and Ryan Carpenter are the primary veterinarians at Hollywood Park. Dr Latson and Dr. Carpenter are part of the large Equine Medical Center group practice, which also handles the veterinary services at Santa Anita and Del Mar.

On Nov 4, 2011, **Case #1**, a 2YO colt owned by (Owner case #1), died while galloping. The horse was returning from a layup for a SF tendinitis. Necropsy showed eosinophilic and pleocellular, focal, mild to moderate inflammation of atrio-ventricular node of the heart of unknown etiology. The A-V node connects atrial and ventricular electrical conduction. The pathologist listed the cause of death as likely failure of cardiac conduction system. Even though this could easily be a presumptive diagnosis it is considered unexplained by CAHFS pathologists. The horse was reported as uninsured.

On November 26, 2011, **Case #2**, a 5YO horse owned by (Owner Case #2), died while jogging back to be unsaddled after racing at Hollywood Park where he had finished 2nd. Necropsy showed 1.) lymphoplasmacytic and neutrophilic, multifocal to diffuse, mild to moderate valvular endocarditis of the left ventricle; 2.) Very mild lymphoplasmacytic and neutrophilic, focal, interstitial myocarditis; and 3.) Multifocal to locally extensive, with moderate interstitial fibrosis and abundant hemosiderin deposition pulmonary hemorrhage and edema, marked, multifocal to locally extensive, with moderate interstitial fibrosis and abundant hemosiderin deposition with the lesions most prominent on the middle and caudodorsal aspects of the right and left lung (*chronic bleeder*). The presumptive diagnosis was heart failure and/or exercise-induced pulmonary hemorrhage. Case #2 was racing on Lasix. He was routinely treated with adjunct bleeding medications. Attending veterinarian Dr. Keith Latson acknowledged EIPH

was an ongoing issue with Case #2. The veterinary record indicates a number of adjunct bleeder medications were used for both workouts and racing.

On January 6, 2012, **Case #3**, a 4YO colt owned by (Owner Case #3), died after quarter-mile into a scheduled 5F work. The necropsy showed acute to sub-acute, severe encephalomyelitis with a positive immunochemistry for *Sarcocystis neurona*. The definitive diagnosis was equine protozoal myelitis (EPM). EPM is not recognized as a cause of sudden death in race horses even though there was a similar EPM sudden death case several years ago. Assistant trainer Mike Marlow acknowledged in his interview the horse was “weak behind” and had received veterinary treatment on both his back and hocks. Neurological problems in horses often present with subtle hind limb abnormalities which can be confused early on with lameness. Whether that was the case here is only speculation. Neither Dr. Latson nor Dr. Carpenter thought Case #3 was showing any neurological signs before his death. Severe hemorrhage of the mesenteric vessels is an important additional pathological finding in this case in light of subsequent cases of suspected rodenticide intoxication. There was no remaining liver tissue from **Case #3** to analyze when retroactive testing for rodenticides was done on old cases in early 2013. **Case #3** was working on Lasix

On June 12, 2012, **Case #4**, a 4YO colt owned by (Owner Case # 4), died after racing. The necropsy showed peracute, multifocal and locally extensive, moderate myocardial degeneration of the left and right atrium, right ventricular free wall and mild, subacute/chronic, lymphocytic and neutrophilic, multifocal valvular endocarditis of the left atrioventricular valve. Presumptive diagnosis of heart failure. **Case #4** was racing on Lasix. *(Note: This is a presumptive diagnosis of heart failure, an example the conservative approach the pathologists at CAHFS take in determining a definitive diagnosis. This is an observation, not a criticism.)*

On August 2, 2012, **Case #5**, a 2YO colt owned by (Owner Case # 5), died while galloping. Necropsy showed moderate to marked, lymphocytic myocarditis in the right ventricular wall, atrial appendages, and in the semilunar valve region and mild to moderate subendocardial fibroelastosis of the pulmonary artery semilunar valve and other sites in the heart. Clinically, a systolic heart murmur was identified a short time prior to the death as part of examination to renew insurance coverage. On August 18, 2012, two days before Case #5's sudden death, Dr. Latson noted in an e-mail to the insurance underwriter copied to Bob Baffert that an echocardiogram would be necessary to better characterize the murmur but Case #5 was not showing any signs of exercise intolerance or cardiac insufficiency and had worked up to ½ mile. The horse was insured at the time of his death.

On December 21, 2012, **Case #6**, a 3YO gelding owned by (Owner Case #6), died galloping. The necropsy showed massive thoracic and abdominal hemorrhage with no large vessel ruptures being identified. Cause of death was hypovolemic shock from massive hemorrhage. A trace of diaphacinone, an anti-coagulant rodenticide was found in liver samples on toxicology. The diaphacinone found was so low that Dr. Poppenga, the CAHFS toxicologist, was unsure as to the clinical significance of the finding. However, equine internal medicine specialists at UCD-SVM believe any anti-coagulant rodenticide (*rat poison*) in face of unexplained hemorrhage is significant. Rodenticides were not part of the standard toxicology screen before this case. A number of cases, sudden deaths and musculoskeletal fatalities

where liver samples were still available, were tested retroactively for rodenticides. No rodenticides were found in the retroactive testing of any of those cases, which included the first two Baffert sudden death cases where liver was still available (Case #1 and Case #2). Rodenticides are now performed on sudden death toxicology screens where hemorrhage is a finding and was done on the last Baffert case in March, 2013. There have been two sudden deaths in 2013 where trace levels of rodenticide has been found where internal hemorrhage was the presumed cause of death. One was a horse racing at Santa Anita and the other was a pony in the post-parade at Los Alamitos.

On March 14, 2013, **Case #7**, a 5 YO mare owned by (Owner Case #7), died galloping. Necropsy found multifocal severe pulmonary hemorrhage and edema more pronounced in the dorsal segments of the lungs (typical of EIPH). Case #7 was treated with Lasix to work and Lasix and estrone to race. EIPH as a cause of death is most associated with racing or working but is also seen in horses galloping. Even though the pathologist suspected a coagulopathy due to unusual extra-pulmonary hemorrhage, no anti-coagulants were found on toxicology. Otherwise the gross necropsy and histology were consistent with pulmonary hemorrhage, essentially EIPH.

Summary of necropsy findings:

Three horses have what pathologists considered definitive diagnosis:

- Case #6 : Internal hemorrhage (of unknown etiology-presumed rodenticide toxicosis)
- Case #5: Cardiac failure
- Case #3: EPM

Three horses have presumptive diagnosis:

- Case #7 : Pulmonary hemorrhage (EIPH)
- Case #4: Cardiac failure
- Case #2: Pulmonary hemorrhage/cardiac failure

One horse has a suspected diagnosis:

- Case #1: Cardiac failure (mild-moderate A-V node inflammation)

Summary Drug testing at EACL (Maddy):

Dr. Stanley's EACL (Maddy) Report on the drug testing of the seven cases is attached.

Uncoagulated blood needed for analysis at EACL Maddy was not available on any horses for testing. Blood coagulates very quickly after death and except for racing fatalities with Case #4 and Case #2 there would have been no regulatory veterinarian on site. Regardless, urine is the superior testing matrix and these cases were unusual inasmuch as urine was obtained for testing in all 7 cases during the necropsy.

Urine samples are obtained by the pathologist directly from the bladder at the CHAFS necropsy laboratory in San Bernardino. The bladder can be accessed directly after the carcasses are opened up for examination, but urine is not always present in the bladder by the time the carcass reaches the laboratory.

There were no unexpected findings with the exception of diclofenac in the synovial fluid of Case #4. Diclofenac is a NSAID found in the topical cream Surpass which had been properly prescribed to Case #4 nine days prior to the race. Synovial fluid is not a routine testing matrix and this finding is considered incidental.

Due to the incompatibility of the CAHFS's necropsy laboratory information management system (LIMS) and the EACL (Maddy) LIMS system for drug testing results, reports from drug testing at Maddy are not automatically updated and entered into the final necropsy reports as are the toxicology results. The pathologist must manually enter the drug testing results or attach them separately to the file. This has caused confusion even with CHRB official veterinarians over the years as to what drug testing was being done on necropsy cases. The results are generally entered in the text of the report rather than a stand-alone section in the report as with toxicology results.

Baffert's Stable was subjected to out-of-competition testing specifically targeting erythropoiesis-stimulating agent, specifically erythropoietin and darb-erythropoietin, at both his Santa Anita and Hollywood Park stables. Baffert's stables were targeted in response to the 3 sudden deaths that had happened that fall and winter. The testing was primarily conducted in the spring on 2012 and included screens for beta-2 agonists (clenbuterol, ractopamine and zilapterol) and anabolic steroids. Clenbuterol was identified in about 25% of the horses. There are other samples obtained in the normal course of regulatory veterinary activities which are likewise subjected to the CHRB's out-of- competition screen which would include Baffert horses in the normal course of affairs.

CASE #1 (Case #1)

Training: The urine sample was found to contain no foreign substances.

CASE #2 (Case #2)

Racing: The urine sample was found to contain no foreign substances other than properly reported pre-race administration of furosemide and phenylbutazone.

CASE #3 (Case #3)

Training: The urine sample was found to contain furosemide and clenbuterol. The furosemide was reported on the necropsy submission form and there is no regulatory threshold for clenbuterol training.

CASE #4 (Case #4)

Racing: The urine sample was found to contain nandrolone, traces of diclofenac and flunixin as well as properly reported pre-race administration of furosemide and phenylbutazone. Nandrolone is found

naturally in intact males. The synovial fluid sample was found to contain diclofenac and phenylbutazone. The liver sample was found to contain traces of phenylbutazone and traces of clenbuterol. Surpass (diclofenac), Ventipumin (clenbuterol), Banamine (flunixin) and Lasix (furosemide) had all been properly prescribed to Case #4 in the weeks prior to the race and none constituted a violation.

CASE #5 (Case #5)

Training: The urine sample was found to contain nandrolone. Nandrolone is found naturally in intact males.

CASE #6 (Case #6)

Training: The urine sample was found to contain no foreign substances.

CASE #7 (Case #7)

Training: The urine sample was found to contain no foreign substances.

Summary Toxicology

The toxicology investigation is well described in Dr. Bob Poppenga's report and the environmental toxicology report prepared by Safety Steward Jeff Salmon, which are both attached.

The standard CAHFS toxicology protocol included with CHRB cases is a heavy metal screen and for selenium. This screen is included in the CAHFS' routine necropsy protocol and is performed on almost all full necropsy accessions. The screen is run on liver tissue and includes the following metals: arsenic, cadmium, copper, iron, mercury, manganese plus selenium. Selenium is not a heavy metal. To date, there have been no abnormal findings in CHRB cases for heavy metals though there have been cases where selenium deficiencies have been suspected. Other tests are on a case-by-case basis at the direction of the pathologist or in consultation with the equine medical director. Whenever possible in sudden death cases and other cases as requested, drug testing is performed at the Maddy lab when a urine sample is available. The Maddy lab's established protocols using LC/MS techniques have more sensitivity and a broader spectrum of drug compounds. In other circumstances, the testing at Maddy would be considered toxicology. The urine and the plasma or serum portion of blood are relatively "clean" matrices for testing. Until recently, the Maddy lab has not had the necessary equipment to prepare tissue preparations in able to run liver extracts through the Maddy's LC/MS instruments. Aqueous humor (eye fluid) and synovial fluid can be obtained fairly cleanly and have been used for testing at Maddy when other samples are not available. The liver extract testing has been left to the CAHFS toxicology, which has the experience, protocols and equipment to test liver and other tissue extracts.

The most important finding of the toxicology testing has been the identification of the rodenticide diphacinone in Case #6. Case #6 died of massive internal hemorrhage in both his thorax and abdomen

without evidence of a major vessel failure. A rodenticide is rat poison and diphacinone is an anticoagulant rodenticide. Anticoagulant rodenticides inhibit epoxide reductase, causing a loss of vitamin K regeneration. This results in depletion of vitamin K, which leads to the inhibition of coagulation synthesis. Clotting factors are II, VII, IX, and X are dependent on vitamin K. The trace finding of an anticoagulant rodenticide is considered significant. Twenty-eight cases where liver tissue was still available including Case #2 and Case #1 were retroactively tested for rodenticides. No evidence of rodenticides was found in those 28 samples. Liver samples were no longer available for Case #3, Case #4 and Case #5. Subsequently, rodenticides were found in two cases with internal hemorrhage in 2013, one was racing at Santa Anita and the other a pony at Los Alamitos. Diphacinone was the anti-coagulant rodenticide found in Case #6. Per Hollywood Park, the only rodenticide they use is bromodiolone in sealed traps.

The environmental toxicology testing delayed the finalization of the investigation and case review by several months. The three common factors for the 7 sudden deaths being investigated were trainer Baffert, Barn 61 at Hollywood Park, and the Hollywood Park main track. The decision was made to conduct environmental toxicology examining Barn 61 at Hollywood Park and the Hollywood Park main track. Due to agency budgeting issues and state contracting procedures, the contracts were not completed until late July. Two private environmental toxicology firms were selected; rodenticide testing on two samples obtained close to rat traps was referred to CAHFS at UC Davis, which had testing protocols in place for rodenticides that were not available from the two private firms. The sampling was done in August and September with the last report into the CHRB in early October. The environmental toxicology testing results are unremarkable and do not indicate an environmental toxicant of concern. The two samples collected close to rat traps tested negative for rodenticides.

Summary Exercise History

The exercise history for each horse was evaluated using the Exercise History Report developed by Dr. Sue Stover at the Veterinary Orthopedic Research laboratory at UC Davis School of Veterinary Medicine. Exercise history has long been related to catastrophic musculoskeletal injuries (CMI) (Estberg 1996, Estberg 1998). The Exercise History Report was used extensively as part of the CHRB-UCD Racing Safety Program and provides an objective way to compare high intensity exercise between horses.

The Exercise History Report is in 4 parts: Part 1). Graphical Representation of Individual High-Speed Exercise Histories; Part 2). Case and Control Horses Plotted Together; Part 3). Case Horse's Event History (races and works); and Part 4). Comparison of Exercise Variables between Case Horse and all age and breed matched Control Horses in the database. The Exercise History Report is used to relate exercise history to CMI's. For our purposes the graphical representation of the case horse's exercise history in Part 1 and the plotted comparison of exercise variables between case horse and control horses in Part 4 are the most useful. The case exercise history graph in Part 1 allows for recognition of exercise patterns that are unusual. The charts in Part 4 allow for comparison of the case's exercise history with the exercise history of other unaffected racehorses. Dots in the mid (0) position indicate that the case's exercise parameter is average compared to that of a group of other unaffected racehorses of similar

age, sex, and breed. Dots to the left are below average; to the right are above average. The black dots represent parameters within one standard deviation of the average; red dots represent parameters outside of one standard deviation from the average but within two standard deviations and the red X's represent parameters that are over two standard deviations from the average. Several important parameters that will be mentioned include number (#) of career races and works, exercise intensity rates (furlongs/month) at specific times in career (e.g., before death), # events since last layup, and distance (in combined furlongs for works plus races) in the last 6, 8, 10 & 12 months leading up to death. Number of career works is simply the total number of works for the case horse in its lifetime; before death furlongs/month is the rate of high intensity exercise over the last 2-5 events before death; events since last layup is the sum of the number of works and number of races since the last layup; and distance in the last 6, 8, 10 or 12 months is the total high intensity furlongs, races and works, in the last 6, 8, 10 & 12 months before death.

The Exercise History Report does not take into consideration the differences between high intensity events. A five furlong work in 57 flat is recorded the same as 5 furlongs in 1:05 and a horse finishing last beaten by several lengths in a 6 furlong maiden claiming race run in 1:11 is recorded the same the winner as a 6 furlong sprint stakes in 1:09 if they are the same age. Case control horses in Part 2 of the Exercise History Report does compare horses in the same race when a fatal incident occurs in a race. Regardless, the Exercise History Report tends to understate the exercise history differences for the trainer who works his horses faster than average.

Case #5 and Case #1 were both 2YO's early in their careers. Neither had ever raced. Case #5 had just 6 recorded works; Case #1 just 5 recorded works. Both Case #5 and Case #1 were galloping when they died; Case #1 was in his return from a layup. Case #6 had been claimed from another trainer less than three months earlier and had worked just 4 times for Baffert and raced just once since he was claimed. There is nothing out of the ordinary in any of the Exercise History Report for Case #6, Case #5 and Case #1.

Case #3, Case#7, Case #2 and Case #4 have more extensive exercise histories under Baffert's training and have similar exercise profiles. All four have above average values for Distance Last 6, 8, 10 or 12 months and only Case #7 Distance Last 10 months is not at least one standard deviation above normal. Case#7 is the only horse of the seven with a Before Death furlong accumulation rate one standard deviation above normal prior to her death. Both Case #4 and Case #3 had above average time since last layup is time in months since the last layup and events since last layup. Simply, Case #4 and Case #3 had been in training continuously without a layup before their deaths. The Exercise History Report for Case #3, Case#7, Case #2 and Case #4 indicates they had a comparably rigorous racing and training program. The Exercise History Report compares the case horse's exercise history to age, sex and breed matched horses. It does not compare trainers, so we cannot say how these horses compare to horses trained by an equally successful trainer as Bob Baffert.

Summary Veterinary and Medication

Bob Baffert's use of medication and veterinary services, at least in the 7 horses examined, can best be described as moderate and, with the exception prescription medications and supplements dispensed somewhat routinely by his veterinarians, his medication and veterinary services were not out of the ordinary. A table listing the medication usage by horse is attached.

Dispensing routine prescription medications and supplements constitutes a large portion of all veterinary services in the 7 Baffert horses. Those veterinary services will be addressed more below. With the exception of those treatments an overview of the veterinary care can be found in the medication table.

Other than dispensed medications a synopsis of veterinary care is as follows:

Case #1 was returning from a layup for a superficial flexor tendonitis tendinitis. Except for multiple ultrasound examinations for the tendonitis his veterinary care was routine.

Case #2 has a rather extensive veterinary medical record compared to all the other horses going back to March, 2009. He received numerous adjunct bleeder treatments and remedies including Stop 20, a homeopathic anti-bleeder treatment. Attending veterinarian Dr. Keith Latson stated EIPH was an ongoing issue with Case #2 and the veterinary medical records indicates this has been the case since early in 2010. Adjunct bleeder medications were used for both workouts and racing, all within CHRB regulations. Case #2 also received both Adequan and Pentosan which contain polysulfated veterinary products considered chondroprotective medication (anti-arthritis).

Case #3 had a veterinary medical record going back to May, 2010. Assistant trainer Mike Marlow described Case #3 as "weak behind." The horse had received had received veterinary treatment on both his back and hocks. His hocks had been injected with cortisone 7 days before his death and before in November. He had had shockwave treatment on his back and gluteal muscles. Neither Dr. Latson nor Dr. Carpenter thought Case #3 was showing any neurological signs before his death. **Case #3** was working on Lasix when he died.

Case #4 had a veterinary medical record going back to July, 2010. He had various ailments and veterinary treatments over that time that appears to be routine. He did receive a number of pre-race medications including flunixin and methocarbamol injections. Baffert runs almost all of his horses on phenylbutazone administered orally the morning before the race by his barn staff.

Case #5 had one needle inserted in his roughly 7 weeks in Baffert's barn to draw blood for routine laboratory work. All other veterinary treatments are dispensing routine medications and supplements. A systolic heart murmur had been identified on a routine insurance examination. Discussions were underway as to whether an echocardiogram should be pursued when the horse died galloping.

Case #6 had been in Baffert's barn 2 ½ months when he died. The veterinary treatments were routine. He was treated pre-race treatment with flunixin and methocarbamol and phenylbutazone administered

orally the morning before the race by his barn staff. Except for the dispensing routine medications and supplements, his veterinary record is routine and unremarkable.

Case #7 has a veterinary record going back to January, 2012. Somewhere during that time she had “throat surgery” which is not in the record. She has a history of multiple anti-biotic treatments from time to time that don’t appear to be particularly serious. She underwent a number of endoscopic examinations and was treated with estrone and Lasix when she worked.

The routine prescription medications and supplements dispensed by veterinarians in the Baffert barn were:

GastroGard: This is the brand name for the proton pump inhibitor omeprazole. This is the equine equivalent to Prilosec. The medication is very commonly used to treat gastric ulcers that are common in race horses. There have been no serious side effects associated with the use of this medication. (6/7 horses were prescribed GastroGard) See <http://www.gastrogard.com/Pages/index.aspx>

Ventipulmin (clenbuterol): Ventipulmin brand of clenbuterol, the only FDA approved form of clenbuterol for the horse was very commonly used in racing until the CHRB suspended the authorized threshold in the summer of 2012. Clenbuterol has been associated with changes in equine cardiac muscle(Kearns), but has not been definitively associated with sudden death or cardiac arrhythmias. The role of clenbuterol in sudden deaths has been speculated for years but there has been no definitive proof and, at this time can only be considered speculation. http://www.bi-vetmedica.com/main/by_product/ventipulmin.html

Thryo-L (levothyroxine): This is thyroid hormone (T_4) used to treat hypothyroid conditions. It is fairly commonly used at the race track. However, the blanket prescribing of thyroxine to all horses in Baffert’s barn does appear unusual. In a sense, the medication was treated more as a supplement than a medication. Dispensing medications at race tracks can be similar to herd health medication dispensing. Thyroxine is most commonly used by veterinarians to assist weight loss in individual overweight horses especially when they come in from the farm. The goal is to create a relative and artificial hyperthyroid condition thereby facilitating weight loss. Thyroxine must be prescribed by a veterinarian, but as in the Baffert situation, thyroxine is often, if not usually, dispensed at the trainer’s request. Per Dr. Latson, the recommended dose was 12mg which by the label is one level teaspoon. How carefully the dosage was followed was not determined. Per Baffert, barn staff including grooms, were involved in administering the thyroxine in feed. The veterinarians conducted no laboratory tests in Baffert’s stable to determine whether any of the horses were hyperthyroid or hypothyroid before treatment. Accordingly, it is difficult to know whether the supplementation resulted in a hyperthyroid condition. Hyperthyroidism in humans is convincingly associated with cardiac arrhythmias and functional abnormalities(Brandt, Franklyn). Through discussions with a number of veterinarians, prescribing thyroxine without evaluating thyroid levels is consistent with the standard of care for prescribing and dispensing thyroxine at the Thoroughbred race tracks in southern California. Baffert said he had used thyroxine for about over 5 years because he thought the medication helped “build up” his horses. Baffert’s comment is surprising since the drug is most commonly used to assist weight loss and has been shown to cause weight loss in

horses (Nichols). In that same study, the use of thyroxine was considered safe in sedentary horses at much higher dosages, but the authors did identify cardiac alterations, a significant decrease in percent fractional shortening, and cautioned, “further studies are required to determine whether L-T4[thyroxine] can be safely administered to horses with medical problems and whether it remains a safe treatment in horses that are facing physiologic challenges such as exercise or pregnancy.”

Even though the use of thyroxine is concerning in horses with suspected cardiac failure, the medication was used in all of Baffert’s horses at all tracks. Baffert said Thyro-L was used on all his horses. To confirm this, medical records were requested and provided for 7 matched case control horses to each fatality by race or work date for horses at Santa Anita or at Del Mar in the case of Case #5. All 7 had been regularly prescribed Thyro-L. In fact Thyro-L was so routinely prescribed it was dispensed to Case #3 a week after he had died. Since thyroxine was used regularly on all of Baffert’s horses at all tracks, Thyro-L does not explain why all the fatalities occurred at Hollywood Park. Thyroxine was used at the time on all of his horses including Santa Anita and Del Mar. Baffert has said he has discontinued the use of Thyro-L in April after an internal review of his supplement program. (7/7 horses were prescribed Thyro-L.) See http://www.lloydinc.com/media/filer_private/2012/06/15/thyrol.pdf

Muscle Mass was an oral equine vitamin and mineral supplement used for a short time.

Osteon and Platinum Performance: These are popular, widely used supplements in racing stables. They have a reputation for good quality control and are considered a high quality product. See <http://www.platinumperformance.com/equine/> and <http://www.platinumperformance.com/bone/> .

Lixotinic: This is an oral vitamin and mineral supplement produced by Zoetis, the veterinary product company spun-off from Pfizer in recent years. See <http://www.drugs.com/vet/lixotinic.html> .

Visorbin was an oral equine vitamin and mineral supplement from Pfizer that appears to have been discontinued.

Adequan and **Pentosan** are injectable veterinary products purported to be chondroprotective (anti-arthritic).

Methocarbamol is a commonly used centrally acting muscle relaxant that was sold at one time as Robaxin-V. It is a common pre-race medication, which can be administered outside of 48 hours before a race. All methocarbamol is now compounded.

NSAID’s (Non-steroidal anti-inflammatory drug): phenylbutazone, diclofenac (Surpass), ketoprofen (Ketofen), flunixin (banamine) fall into this group. NSAID’s were administered alone or in various combinations on a case by case basis. All are commonly used in race horses and appear to have been administered within CHRB regulations.

Adjunct Bleeder medications: These include estrone (an estrogen), glycopyrrlate(short-acting bronchodilator), Stop 20 (a homeopathic remedy)

Among other efforts Baffert's veterinarians conducted their own clinical investigation that included testing for coagulopathies and heart muscle damage.

- The day following Case #7's fatality, Dr. Latson obtained blood samples from 24 horses at Baffert's barn at Hollywood Park to test for troponin I. Troponin I is used to measure heart muscle damage in humans. Whether troponin I is useful in horses or not is unclear, but all results were below 0.2ng/ml and considered normal.
- Dr. Latson conducted clotting time testing in the spring of 2013 looking at partial thromboplastin time (PTT) and prothrombin time (PT) to identify coagulopathies in a group of 30 horses at Hollywood Park. None of the horses showed prolonged PT or PTT.

Summary of CHRB Investigation

Chief Investigator Bill Westerman assigned the CHRB's investigation to supervising investigator Rick Ameva. Numerous documents and records were compiled for the investigation. The barn was inspected and key personnel interviewed. The equine medical director provided horse specific questions based on necropsy, drug testing, toxicology and veterinary medical records for Drs. Latson and Carpenter and for trainer Bob Baffert and assistant trainer Mike Marlow. Supervising investigator Ameva conducted the interviews. Attorney Steve Schwartz attended interview conducted with Bob Baffert and assistant trainer Mike Marlow. Equine medical director Dr. Arthur was present at Dr. Latson's interview.

Conclusion

The cluster of seven sudden deaths of horses trained by Bob Baffert on the Hollywood Park main and stabled in Barn 61 at Hollywood Park remains unexplained. Three horses had definitive diagnoses; three horses had presumptive diagnoses with specific pathological findings and one death is unexplained. There is no evidence whatsoever CHRB rules or regulations have been violated or any illicit activity played a part in the 7 sudden deaths

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Summary of Drug Test Results for CHRB Necropsy Sample Investigation

The K.L. Maddy Equine Analytical Chemistry Laboratory (Maddy Lab) received seven samples in related cases all from a single Thoroughbred horse trainer in Southern California. All horse died during either racing or training over the period of 18 months (November 2011 – March 2013). The following is a summary description of the analytical testing performed for each of the seven specimens.

Screen and quantify specimens:

The drug testing process aims to separate and identify non-endogenous components or drugs in biological matrixes, such as urine, plasma or necropsy tissues. The analytic process used to accomplish this is divided into three components: extraction of the drug from the biological matrix, isolation of the drug from the other compounds and detection and identification of the drug(s).

All of the Maddy Lab's in-house methods are recognized in the Scope of Accreditation to ISO/IEC 17025:2005 by American Association of Laboratory Accreditation (A2AL - Certificate # 2205.1) and the Racing Medication and Testing Consortium (RMTC - Certificate # 1).

Instrumental Method of Analysis:

The Maddy Lab is designed and equipped to provide innovative and progressive testing methods. The testing scheme utilizes analytical methodologies based on gas and liquid chromatography separation combined with mass spectrometry detection to enables coverage of a wide range of small molecules (~1,500 drugs). Mass spectrometry is the routine tool for our analytical problem solving. In the mass spectrometer, the molecules of the sample are bombarded with electrons producing positive ions, the fragment ions fly through the mass spectrometer separating each fragment by their masses. A mass spectrum of each component is produced which is a unique chemical fingerprint. This technique utilized by the Maddy Lab provides maximum sensitivity for our screening with either gas or liquid chromatography combined with mass spectrometry (GC-MS, LC-MS-MS).

Each of the specimens received from the CHRB's necropsy program for testing were subjected to our instrumental-based drug testing program. The instrumental testing program exceeds the industry target established by the Thoroughbred Owners and Breeders (TOBA) Graded Stakes Committee, better known as the "TOBA Test," meeting the criteria for all North American Graded Stakes races. Analytes detected by the screening tests were identified by validated methods. The Maddy Lab issued a summary report of the chemical identifications and scientific assessment of the findings to the CHRB for its review upon completion of the tests.

These analytical techniques were selected because they have the following characteristics. 1) A wide range of drugs are detected by single extraction procedure with few analytical runs. 2) Interference from other substances is minimal. 3) The limit of detection (LOD) for most drugs is low enough to detect *illicit usage*, currently, the LOD in urine for most substances of concern are below one picogram per milliliter (part-per-billion). 4) A new drug can easily be detected on its first use (e.g. Dermorphin, also known as, Frog Juice). 5) Excretion data for all drugs and metabolites potentially detected by the test are acquired during the validation of the test procedures. 6) The use of these techniques increases the defensibility of chemical identification in administrative hearings on forensic evidence.

Instrumental screening methods involve the coupling of a gas or liquid chromatograph with a mass spectrometer. Separation of the drugs from each other and from other substances in the sample extract occurs in the chromatographic phase. Identification and quantitation occur in the mass spectrometer after chromatographic separation.

Urine analysis: The screening procedure employs solid phase extraction with multi-fraction collection to detect three chemically diverse classes (acidic, basic and neutral drugs). This extraction method makes use of a polymer column, where ion exchange functionalities are sequentially performed in a mixed mode interaction, both ionic and hydrophobic. This isolates acidic/neutral and basic compounds, simultaneously. The Maddy Lab performs this extraction method for all necropsy specimens submitted.

The intended screening methodology has been proven robust, handling more than 150,000 samples from July, 2007 through October, 2013. This screening method has been proven reliable by detecting 99.8% of expected quality control samples.

Basic Fraction: This urine fraction contains several classes of drugs including *β 2-agonist, β -Blockers, narcotic analgesics, local anesthetics, stimulants and sedatives*. Our LC-MS/MS base method is recognized in the Scope of Accreditation to ISO/IEC 17025:2005 by A2LA (Certificate Number 2205.1; Liquid Chromatography/Mass Spectrometry).

The sample extracts are provided as dried residues in 2 mL autosampler vials following solid-phase extraction. The method described here utilizes the Hewlett Packard GC/MS instruments. In brief, this method consists of separates components by gas chromatography and detects them by full-scan electron-ionization mass spectrometry, generates a report which summarizes both mass spectral library and quantitation database search results, reducing the need to manually review excessive amounts of data.

Gas Chromatography-Mass Spectrometry (GC-MS) w/Large Volume Injector and Liquid Handling System (Agilent 5973(5)/6890; Apex Technologies, ProSep 800; LEAP Technologies, Combi Pal). This combination of technologies improves LOD, and allows for a wider spectrum of analyte detection (>1,000 drug compounds).

Acidic and Neutral Drug Fraction: This urine fraction contains several classes of drugs including *anabolic steroids, corticosteroids, diuretics, non-steroidal anti-inflammatory drugs (NSAIDs), and xanthine alkaloids*. Our LC-MS/MS acid/neutral method is recognized in the Scope of Accreditation

to ISO/IEC 17025:2005 by A2LA (Certificate Number 2205.1; Liquid Chromatography/Mass Spectrometry).

This method utilizes a binary liquid chromatograph system equipped with a photodiode -array UV detector and a LTQ linear ion-trap mass spectrometer. The horse urine and blood extracts are provided as dried residues following solid-phase extraction. In brief, the dried extracts are dissolved in the LC mobile phase and aliquots are automatically and sequentially injected onto a LC column where components are separated by reversed-phase gradient liquid chromatography. The separated components are then detected and identified by photodiode -array UV spectra and/or by mass spectrometry detection.

Results Summary for each Specimen:

CASE #1 / EA CL XXXXXX:

Training:

The urine sample was found to contain no foreign substances.

CASE #2 / EA CL XXXXXX:

Racing: This pre-race medication reported legal administration of furosemide and phenylbutazone.

The urine sample was found to contain no foreign substances.

CASE #3 / EA CL XXXXXX:

Training: This pre-exercise medication reported legal administration of furosemide.

The urine sample was found to contain clenbuterol.

CASE #4 / EA CL XXXXXX:

Racing: This pre-race medication reported legal administration of furosemide and phenylbutazone.

The urine sample was found to contain nandrolone, traces of diclofenac and traces of flunixin. The synovial fluid sample was found to contain diclofenac and phenylbutazone. The liver sample was found to contain traces of phenylbutazone and traces of clenbuterol.

CASE #5 / EA CL XXXXXX:

Training:

The urine sample was found to contain nandrolone.

CASE #6 / EACL XXXXXX:

Training:

The urine sample was found to contain no foreign substances.

CASE #7 / EACL XXXXXX:

Training:

The urine sample was found to contain no foreign substances.

Conclusion:

The analyses showed only the presence of therapeutic and authorized medications in the post-mortem submissions. No prohibited substances were detected. At this time no other additional tests or analyses are pending on the seven specimens.

Sincerely,

Scott D. Stanley, Ph.D.
Professor
K.L. Maddy Equine Analytical Chemistry Laboratory

Summary of Toxicology Results for CHRB Investigation of Baffert Sudden Deaths

A number of postmortem toxicology tests have been run on seven horses trained by and which died suddenly during training or racing. The following is a tabular list of the specific toxicology tests performed by the CAHFS Toxicology Section for each accession along with a subsequent discussion of each test.

Accession →	CASE #1	CASE #2	CASE #4	CASE #4	CASE #5	CASE #6	CASE #7
Heavy Metal Screen + Selenium	+	+	+	+	+	+	+
Anticoagulant Rodenticides	+	+				+	+
Mass spectrometry screens by GC- and LC-MS	+	+	+	+	+	+	
Brain cholinesterase	+		+		+		
Alkaloid Screen				+	+		
Ionophore Screen				+	+		
Strophanthidin					+		
Oleandrin				+	+		
4-amino-pyridine (Avitrol®)	+	+	+				
Vitamin E							+
Drugs of Abuse Screen					+		

Heavy Metal Screen and Selenium. This screen is included in the CAHFS' routine necropsy fee and is performed on almost all full necropsy accessions. The screen is run on liver tissue and includes the following metals: arsenic, cadmium, copper, iron, mercury, manganese, molybdenum, lead, zinc, and selenium. The screen was performed on all of the above accessions; no clinically significant findings were noted for any accession.

Anticoagulant Rodenticide Screen. This screen detects the following anticoagulant rodenticides: brodifacoum, bromodiolone, chlorophacinone, coumachlor, difethialone, diphacinone, and warfarin. This screen is not typically run on CHRB cases. Anticoagulant rodenticides (AR) block the action of vitamin K epoxide reductase and enzyme necessary for the normal functioning of clotting factors II, VII, IX, and X. In the absence of these clotting factors, a coagulopathy develops approximately 2 to 4 days following exposure to the AR. There is relatively little known about the sensitivity of horses to AR and few anecdotal or published case reports involving horses.

Due to the presence of substantial postmortem hemorrhage in several of the above accessions, it was decided to rule out the possibility of AR exposure/intoxication by testing liver tissue for their presence in liver samples. No AR were detected at or above our standard reporting limits in three of the four accessions which were tested. Interestingly, the first generation anticoagulant rodenticide, diphacinone, was detected at a “trace” concentration for accession CASE #6. This horse had massive thoracic and abdominal hemorrhage which was consistent with a coagulopathy. A “trace” concentration is defined as confirmation that the analyte of interest was present in a tested sample, but at level that could not be quantitated under the conditions of the test. In this particular case, there was no other postmortem finding that could account for the extensive hemorrhaging noted. In cases in which an AR is detected and no other cause for hemorrhage is found, the results are considered significant and consistent with anticoagulant rodenticide intoxication.

Mass Spectrometry Screens. In situations in which a known toxicant exposure is possible, but there is no history of exposure to a specific toxicant and/or no postmortem lesions suggesting exposure to a particular toxicant, general toxicant “screens” are run utilizing mass spectrometry. In six of the seven cases of interest, screens using both gas chromatography-mass spectrometry (GC/MS) and liquid chromatography-mass spectrometry (LC/MS) were run on one or more samples (typically urine and/or liver). The two screens detect different types of compounds and are complimentary to each other. Any unknown compounds detected by GC/MS are compared to one or more commercial databases of several hundred thousand compounds, thus providing a broad-based screen. Commercial compound databases utilizing LC/MS are unavailable with databases are generated by each laboratory. The CAHFS’ screen includes a chemically diverse group of approximately compounds.

None of the seven accessions contained identifiable compounds of concern.

Brain Cholinesterase Activity. Cholinesterase activity is depressed following exposure to the cholinesterase-inhibiting insecticides in the organophosphorus (OP) and carbamate families. These insecticides inhibit the activity of cholinesterase enzyme in blood and brain. Brain is the sample of choice for postmortem testing. This is considered a “screening” test since it does not

identify a specific cholinesterase-inhibiting compound; if activity is noted to be less than 50% of a “normal” activity range, specific insecticide screens are performed. In the three cases in which brain cholinesterase-activity was determined, activity levels were considered within an acceptable or normal range and no specific OP or carbamate insecticide screens were run.

Alkaloid Screen. The alkaloid screen is designed to detect a number of plant alkaloids; the CAHFS’ screen includes anabasine and nicotine (found in *Nicotiana* spp.), atropine and scopolamine (found in several plant species including *Datura* spp. and *Hyoscyamus* spp.), coniine (found in *Conium maculatum*), deltaline (found in *Delphinium* spp.), sparteine (found in *Lupinus* and *Cytisus* spp.), and taxus alkaloids (found in *Taxus* spp.). This screen was run in two of the seven accessions with negative results. This screen is occasionally run in cases in which myocardial lesions are noted on postmortem exam. Myocardial necrosis was noted for CASE #4 and myocarditis was noted for CASE #5.

Ionophore Screen. The ionophore screen includes the growth promotants and coccidiostats monensin, lasalocid, narasin, and salinomycin. Horses are uniquely sensitive to the toxic effects of these cardiotoxic compounds. In two cases in which myocardial lesions were noted (CASE #4 and CASE #5), colon contents were tested to see if there had been possible exposure. In both cases, none of the ionophores included in the routine screen were detected at or above the indicated reporting limit.

Oleandrin and Strophanthidin. Oleandrin and strophanthidin are the toxic cardiac glycosides found in *Nerium oleander* and *Adonis aestivalis* (summer pheasant’s eye), respectively. These tests are most often run following a history of exposure to either plant or in cases where myocardial lesions are noted. Oleander intoxication of horses occurs on a regular basis whereas intoxication from *Adonis aestivalis* is rare. Thus, the latter test was run on only one case.

4-aminopyridine. 4-aminopyridine is an avicide (bird toxicant) that causes CNS stimulation. While there is little evidence that this compound is used to improve performance in horses, it was felt that a few accessions associated with sudden death should be assessed for the compound. Since the first three of the seven accessions were negative, no further testing was performed.

Vitamin E. Vitamin E is an important antioxidant vitamin that works in combination with selenium to prevent oxidative damage to cells. This test is not often run on CHRB associated accessions. The liver vitamin E concentration was within a “normal” range in the one animal tested (CASE #7).

Drugs-of-Abuse Screen. A more comprehensive drug screen is performed by the EACL. However, a drugs-of-abuse screen was run on a urine sample from accession CASE #5 to investigate this case as thoroughly as possible. The screen is for human drugs of abuse and is

used as a diagnostic tool to confirm either intentional or accidental exposure of companion animals to illicit drugs. The screen included the following drugs: amphetamine, cocaine, benzoylecgonine, norcocaine, ephedrine, lysergic acid diethylamide, methamphetamine, MDMA, nicotine, phentermine, psilocin, delta-9-tetrahydrocannabinol (THC), THC-OH, methadone, morphine, THC-COOH, JWH-018, HU-210, 6-monoacetylmorphine, fentanyl, norfentanyl, JWH-073. No drugs of concern were detected in the one tested urine sample.

Private Laboratory Environmental Testing – E.S. Babcock & Sons, Inc. (Riverside, CA) and Aurora Industrial Hygiene, Inc. (San Diego, CA).

Concern was expressed regarding a possible environmental toxicant associated with the barn where the horses in question were stabled. Since the CAHFS Toxicology Section is not considered an environmental testing laboratory nor does it have analytical protocols for appropriately sampling or testing matrices such as dirt or air, it was decided to contract with specialty environmental laboratories for further testing. Babcock Laboratories collected soil samples and barn debris samples for testing. Soil testing included screens for metals (N = 17), volatile organics, organochlorine insecticides, polychlorinated biphenyls (PCBs), organophosphorus insecticides, and chlorophenoxy herbicides. All testing used EPA-approved methods. Two composite samples (feed room composite and shedrow soil composite) were submitted to CAHFS Toxicology Section to test for anticoagulant rodenticides.

Aurora Industrial Hygiene, Inc. collected air samples within the barn in question, the pathway leading to the race track, and near the synthetic race track surface. NIOSH approved methods were used to test for total particulates, respirable particulates, and total petroleum hydrocarbons.

The environmental test results are reported elsewhere. Results are unremarkable and do not suggest an environmental toxicant of concern. The two samples tested for AR were also negative.

Conclusion. The only toxicology result of note was the trace amount of the AR, diphacinone, which was found in conjunction with extensive postmortem hemorrhaging in CASE #6. Unfortunately, there is no current postmortem test that can confirm the antemortem occurrence of a prolonged clotting time (coagulopathy). If a prolonged clotting time was present antemortem, then a diagnosis of AR (diphacinone) intoxication would be relatively straightforward. However, given the failure to identify another cause for the extensive hemorrhaging noted in the horse, it is certainly possible that exposure to diphacinone contributed to the demise of the horse. The question then becomes one of a possible source for the exposure. Since there is no history of use of diphacinone in the barn in question, it seems unlikely that inadvertent exposure as a result of routine use is possible. It might be that

feed is occasionally contaminated with low concentrations of diphacinone, since it is a widely used AR for rodent control. After the detection of diphacinone in CASE #6, twenty-seven other liver samples from CHRB cases (unrelated to the cases evaluated in this report) were screened for AR and were all found to be negative.

Subsequently, we detected AR in two other CHRB cases (S1301728 and S1302829) as a result of more extensive testing of CHRB cases. Potential sources for exposure were not identified in either of these two cases. Perhaps more routine screening of feed for the presence of AR would be useful to determine whether this is a possible route of exposure.

Respectfully submitted,

Robert H. Poppenga, DVM, PhD, Diplomate, ABVT

Dr. Uzal's Necropsy Report is attached separately

Jeff Salmon's Environmental Toxicology is attached separately