Volume 23 No 1, January 2005

CEH HORSEREPORT

A publication of the Center for Equine Health, UC Davis School of Veterinary Medicine

School of Veterinary Medicine

Genetics and Horses

From Peas to DNA: Genetics in the 21st Century

Prior to the epic studies of Johann Gregor Mendel, many people believed that organisms learned from their surroundings and used these "lessons" to form new traits in their offspring. The transmission of hereditary characteristics from parents to progeny was believed to play a minor role in evolutionary adaptation. After thousands of experiments with peas, Mendel came to realize that characteristics must be carried from one generation to another by some material means which he referred to as an "element"-- possibly a fluid, gas, or some solid particle, but not by some mystic environmental factor.

Mendel's work was not truly appreciated until some time after his death, when other scientists independently repeated his work and discovered similar rules. By the 1920s, the concepts of genetics were becoming clear, and scientists knew that chromosomes were the structures inside cells that contained the heritable "elements" that Mendel had identified much earlier. These basic heritable elements were called *genes*, and each gene existed in several slightly different forms known as *alleles*.

Since Mendel's time, our knowledge of the mechanisms of genetic inheritance has grown immensely. We now understand that inheriting one allele (i.e., trait) can sometimes increase the chance of inheriting another, or affect how and when a trait is expressed in an individual's physical, biochemical, and physiological makeup. Likewise, there are degrees of dominance and recessiveness with some traits. Modern genetic studies have shown that genes that follow simple rules of dominance increasingly seem to be rare, and that the rules of inheritance according to Mendel do not always apply. The science has expanded to include the combined effect of more than one pair of genes — how genes form or work together to create more complex traits.

In 1990, the International Human Genome Sequencing Consortium, involving researchers from six countries, was formed to launch the Human Genome Project in an effort to obtain a highly accurate

— Continued on page 3

INSIDE THIS ISSUE...

UCDAVIS School of Veterinary Medicine

DIRECTOR'S MESSAGE

The Age of Genomics Is Here

Dr. Gregory L. Ferraro

While this space has traditionally been reserved as a platform for my own thoughts and opinions, a wise man knows when it is time to relinquish the podium. For this issue of the Horse Report, I have asked my esteemed colleague Dr. Niels Pedersen, Director of the UC Davis Veterinary Genetics Laboratory, to serve in my stead as the harbinger of new knowledge. While some may not consider Dr. Pedersen as handsome and charming as yours truly, all would ascend to his superiority in the intellectual realm.

As Director of the UC Davis Veterinary Genetics Laboratory, it is my privilege to make a few comments about the state of genetics today. The Veterinary Genetics Laboratory has been one of the foremost horse genetics research units in the world. Although we are perhaps best known for providing parentage, coat color and genetic disease testing for horse owners, these tests have required a considerable expertise acquired through an equally considerable expense in research and development. The funding for this research has come mainly from service income, which is reinvested almost entirely in genetics research. We also have benefited from the funding support of several animal registries, foundations, private donors, and organizations such as the Center for Equine Health.

Finally, we have reached the true age of genomics. The genetic causes of almost all pre-existing genetic disorders of the horse are known. We now have the tools to rapidly identify the genetic basis of new genetic diseases within months of their recognition. This capability has come about by the sequencing of entire human and animal genomes and with the recognition that the structure of all animal and human genomes is to a large extent shared. Although the horse genome has not been completely sequenced at this time, the Veterinary Genetics Laboratory and other laboratories have collaborated in creating a complex comparative map of the horse genome. This map is already powerful enough to allow us to pinpoint the general location of most genetic defects to a specific horse chromosome and a specific region on that chromosome. We can search the analogous sequenced regions of human, mouse or dog chromosomes for genes that may cause a similar disorder in other species, or for genes whose known function or structure may explain the defect in question. The next step is to sequence DNA from the suspected region of horses known to carry the defect, to have the defect, or to be free of the defect.

The greatest resource of the Veterinary Genetics Laboratory besides its staff is its huge DNA bank and database. Pedigrees that have been DNA-verified are available for many generations of horses, depending on the breed. The American Quarter Horse is one breed that has well-managed pedigrees for many generations. DNA for most of the later generations has been stored in the form of hair. If a new disease were to appear in the Quarter Horse, we would work with this registry and use pedigrees to identify affected families and access stored DNA to identify the genetic defect.

The greatest resource of the Veterinary Genetics Laboratory besides its staff is its huge DNA bank and database. Pedigrees that have been DNA-verified are available for many generations of horses, depending on the breed.

We are grateful for all of the horse registries that have been part of our service over the past decades and are glad that the time has finally come when genetics can be applied to its full potential. Hopefully, genetic diseases of horses will continue to be uncommon. However, just as we have seen HYPP, JEB, GBE,

SCIDS and other disorders appear in the past, new diseases will appear in the future. However, we can take solace in the fact that these disorders will be recognized sooner, and that extremely accurate tests for each will be developed with great haste.

Although genetic diseases are important, remember the normal traits that make up the horse or a breed of horse: coat color, color patterning, coat character, size, performance, soundness, longevity, behavior, trainability for specific activities, and so forth. These are all traits of varying complexity that are being genetically defined bit by bit. The genomic age is truly an exciting period to witness and in which to participate.

Remember also that even though some of us did not inherit good looks or charm, we did inherit traits that might even be better!

Niels C. Pedersen, DVM, PhD
Director, Veterinary Genetics Laboratory
University of California, Davis

Genetics — Continued from page 1

sequence of the human genome. The major benefit from this effort is to allow scientists to study the causes of disease by finding all key heritable factors predisposing to diseases like diabetes, or by finding genetic mutations that underlie various types of cancers "with confidence that little can escape detection" (*Nature* **431**, 931-945, 2004). The Human Genome Project, as well as that of the mouse, dog and other species of higher and lower animals, has demonstrated an amazing conservation of numbers and types of genes in mammals. Therefore, other animal species such as the horse can greatly benefit from the experience and information gained from the Human Genome Project.

Genetic Diseases Shared by Humans and Horses

Researchers have identified over 13,000 genetically inherited traits in humans. More than 5,000 of them are diseases or other abnormalities. Some of these diseases also occur in animals. **Hyperkalemic periodic paralysis disease (HYPP)** is a muscular disease that affects both humans and horses. HYPP is caused by a hereditary genetic defect that disrupts the sodium ion channel and results in hyperkalemia, an excessive amount of potassium in the blood that causes the muscles to contract more readily than normal. Horses with HYPP can experience unpredictable attacks of paralysis, which in severe cases can lead to collapse and sudden death due to cardiac arrest or respiratory failure. Because this disease is shared by humans, early advances made in sequencing the human genome made it possible to identify the gene in horses sooner than would have been possible without this connection.

According to Dr. Sharon Spier of the Department of Medicine and Epidemiology, School of Veterinary Medicine, the original genetic defect causing HYPP was a natural mutation that occurred as part of the evolutionary process. The majority of such mutations (which are constantly occurring) are not compatible with survival. HYPP has been associated with horses of heavy musculature, though not necessarily all horses with well-developed musculature are afflicted. The mutant gene causing HYPP has been identified in the descendents of the American Quarter Horse sire "Impressive." Research has not yet been performed on other bloodlines to determine whether the same or similar genetic mutation existing in other bloodlines also may cause HYPP. Since "Impressive" descendents are so numerous, the genetic mutation in the bloodline is widespread. Moreover, the trait is inherited from generation to generation with equal frequency; it does not become "diluted." Breeding an affected heterozygous horse (N/H) to a normal horse (N/N) will result in approximately 50% normal offspring, while 50% will carry the defective gene (N/H). Breeding an affected homozygote (H/H) will result in all offspring carrying the gene mutation, regardless of the status of the other parent. Normal (N/N) horses can be bred safely without fear of HYPP being inherited. Selective breeding of only normal (N/N) horses could entirely eliminate HYPP disease. A DNA test is currently available to identify horses carrying the defective gene that causes HYPP.

A newly identified genetic disease in horses that also has a human counterpart is **glycogen branching enzyme disease (GBED).** A similar disease in humans has been described as a rare, heritable disorder

— Continued on page 4

All their markings are described on their registration papers, and all have numbers and files. Their live births have been noted, their blood has been taken and tested to be sure they descend from whom their owners say they descend, their births have been recorded under the names of their sires in a number of documents, and published in the Thoroughbred Times. Already they are successful, having gotten conceived, gestated, born, nursed, weaned, halter-broken, shod, transported, and taught some basic manners with some misadventure but nothing fatal.

They are all related to one another. Every one of them carries the blood of the Darley Arabian, and Eclipse. You could hardly have a Thoroughbred who did not. Every

Genetics — Continued from page 3

that has a range of manifestations, from mild adult-onset muscle weakness to neonatal death from liver failure. Recently, a fatal neonatal disease closely resembling the human disease was reported in the American Quarter Horse. The initial clinical cases consisted of late-term abortion or dysfunction of skeletal muscle, cardiac muscle, or liver by 8 weeks of age.

This disease was described as an autosomal recessive trait in some Quarter Horse families. Examination of pedigrees containing affected foals revealed that some popular Quarter Horse sires with many descendants are carriers of the responsible gene, and that this gene is also present in related bloodlines of both cutting and pleasure horses. A DNA test has just become available for identifying horses that carry the defective gene. This test will provide critical information to horse breeders to avoid producing affected foals.

Junctional epidermolysis bullosa (JEB) is another inherited disease that causes moderate to severe blistering of the skin and oral membranes and sloughing of hooves in newborn foals. This condition is also known as red foot disease. Affected foals are typically born alive but soon develop skin lesions at pressure points. The condition worsens with time and the foal eventually succumbs from severe infection or must be euthanized. JEB has been shown to be the result of a specific mutation in a gene that affects the production of normal and healthy skin. To date, this mutation has been found only in Belgian Draft horses and derivatives of that breed.

JEB is inherited as a recessive trait. Animals that carry two copies of the mutated gene (J/J, or homozygous recessive) will develop the disease. Animals that carry one copy of the mutated gene and one copy of the normal gene (N/J, or heterozygous for the disease) are carriers of JEB. Carriers do not develop the disease and have normal epithelium, but they have a 50% chance of passing on the mutation to their offspring. Affected animals do not survive to breeding age.

A DNA test is currently available that detects the mutation that has been associated with JEB in Belgian Draft horses and in other breeds derived from Belgian Draft stock. Breeders can reliably use test results to enhance breeding strategies to avoid producing affected foals. Carriers do not need to be removed from the breeding pool. A successful breeding program can use matings of carriers (N/J) to normal horses (N/N) without the worry of producing an affected foal.

Hereditary equine regional dermal asthenia (HERDA) is an inherited skin disease characterized by skin lesions that develop under the saddle area in affected horses. It affects Quarter Horses and other breeds with Quarter Horse lineage, such as Paints and Appaloosas. The disease causes the skin along the horse's back and neck to stretch and tear easily, making the horse unsuitable for riding. Horses are therefore not normally diagnosed with the disease until they are 2 years old and being broke to ride.

Currently, there is no treatment for HERDA, and horses that are diagnosed are usually euthanized or become pasture pets. The disease incidence seems to be increasing. Currently, approximately 5 cases per year are seen at UC Davis, and generally referring veterinarians inform UC Davis of at least another 15 that are seen in other areas of the country. The disease is most likely inherited as an autosomal recessive

disorder (both parents are carriers), since many of the parents of affected horses are completely normal. Breeders who produce foals with the disease may never know that their horses are carriers. If animals that are popular sires are carriers of HERDA, the frequency of the disease will increase over time. A DNA test to identify the mutation that causes this disease is needed to allow breeders to avoid producing affected animals and thereby reduce its incidence. Researchers at UC Davis in Dr. Danika Bannasch's laboratory have been working to identify the location of the gene that causes HERDA so that such a test can eventually be developed.

The UC Davis Veterinary Genetics Laboratory

Testing for Genetic Diseases

Genetic testing is available for the diseases described above, and researchers continue to work on identifying genes for others.

The UC Davis Veterinary Genetics Laboratory (VGL) is a self-supporting unit of the School of Veterinary Medicine operating under the direction of Dr. Niels Pedersen, DVM, PhD. It is the largest horse parentage testing facility in the world and is renowned for genetic testing in horses. The Laboratory was originally established in the 1950s under the direction of Dr. Clyde Stormont for verifying parentage for cattle registries.

The VGL has developed and currently offers diagnostic tests for a number of equine genetic diseases and coat colors and has an active research and development program in this area. Tests for diseases include hyperkalemic periodic paralysis (HYPP), junctional epidermolysis bullosa (JEB), glycogen branching enzyme disease (GBED), and overo lethal white syndrome.

Parentage Testing

Historically, heritage has been an important component for horses, whether they were used for war, for riding pleasure, for working, or for breeding. It was a source of pride to know that one's horse descended from a respected bloodline. Thoroughbred horses have been traced to extraordinarily great runners. Similarly, other animals such as sheep and cattle have been bred for different qualities in a bloodline, such as for wool or for meat.

The Veterinary Genetics Laboratory performs approximately 150,000 tests annually for parentage verification, the bulk of which is to validate pedigree records for more than 30 breed registries. Testing is also performed to match a sire and dam with an offspring having unusual markings or colorings or to identify foals with similar markings, which a breeder suspects may have been misidentified after weaning. There are many instances in which the identity or parentage of animals comes into question. Genetic testing provides answers to problems such as these that are frequently encountered by horse owners and breeders.

In the early years of operation, all identity and parentage testing was carried out with blood typing analysis of serum proteins and red blood cell surface proteins. Today, all genetic testing is based on DNA assays. The Laboratory not only continues to offer animal parentage verification for many livestock and companion animals, but also expanded the work to include research and service for laboratory animals, wildlife species, animal forensics, genetic diagnostics, and genetic disease.

As a pioneer in the development of DNA-based animal parentage verification, in the early 1990s the Laboratory switched from doing primarily blood typing to using DNA

— Continued on page 6

one of them, too, carries the blood of Stockwell and of Nearco. Three of them carry the blood of Rock Sand. Two descend from the great female progenitor Pocahontas. Two are more American than English, going back to Lexington. The lucky ones carry St. Simon. Hyperion appears here and there, a dot of sunlight in any pedigree. The four great broodmare sires—War Admiral, Princequillo, Mahmoud, Blue Larkspur—appear, too, even though no one around any of these foals is old enough to have actually seen them race.

Excerpted with permission from ***Horse Heaven***, by Jane Smiley (2000).

Genetics — Continued from page 5

as the principal tool for parentage testing. It was the first laboratory to offer DNA testing to the horse, cattle and camelid industries and has since introduced DNA-based tests for elk, deer, alpaca, llama, dogs, cats, sheep, goats, camelids and primates. Using this technology, the Laboratory has generated nearly one million DNA profiles.

Equine Genome Research

For several decades, the UC Davis Veterinary Genetics Laboratory has been a leader in equine genetic research. More recently, researchers have been involved in the International Equine Gene Mapping Project. Gene mapping involves using known animal genomes (human, dog or mouse) to map the position of analogous chromosomes in the horse and plays a critical role in the study of genetic traits. One of its primary values is to allow scientists to identify genes that may be responsible for diseases for which the only cure is to avoid breeding. In horses, genes have been identified for hyperkalemic periodic paralysis (HYPP), severe combined immunodeficiency (SCID), overo lethal white foal disease (OLWFD), junctional epidermolysis bullosa (JEB), and glycogen branching enzyme disease (GBED). As a result, genetic tests are now available to screen horses for these diseases and to assist breeders in their breeding programs. Research on other diseases and performance traits continues at UC Davis and other universities around the world, and it can be expected that in the near future additional tests will become available to the horse industry.

While parentage testing and diagnostic tests are the main services, the Veterinary Genetics Laboratory has increasingly been involved in research on genetic mapping in collaboration with researchers in England, Australia, Europe, Japan, South Africa, Scandinavia, and other parts of the United States. Both the human and dog genomes have been sequenced. While it is unlikely that the horse genome will be fully sequenced in the near future, a highly developed and dense gene map of the horse genome could be completed within the next three years.

Genetics in the Average Horse Owner's Life

It is to everyone's benefit to become more informed about genetic defects in the horse and to take the necessary steps to selectively avoid production of affected foals. In the short term, it may not be possible or wise to completely eliminate a genetic disease from a breed, because doing so may cause the loss of other beneficial traits and diminish rather than enhance the genetic pool. It is, however, possible to use the information gained from genetic tests to select breeding stock and plan matings so that affected foals are never produced.

The Veterinary Genetics Laboratory offers tests not only for the genetic diseases described, but also for coat colors such as red factor, agouti (bay/black), cream dilution, and tobiano homozygosity. The popularity of coat color tests reflects the interest of horse breeders to produce animals with desirable or popular colors and patterns. Researchers are currently working on identifying the genes for other colors, including dun dilution, sabino white spotting, and silver dilution. Research on the genetics of coat color in horses has been a hallmark of the Laboratory.

The VGL also offers genetic counseling to horse owners and breeders, often in connection with the coat color tests. In addition to providing a detailed written report on the findings of tests, genetic experts at the Laboratory provide additional explanation of the test results and assist in determining probable outcomes from a specific breeding.

The Future of Genetics

Modern genetics is much like detective work. The challenge is to accurately identify affected vs. unaffected animals and then to search for the gene. Which chromosome is the gene on? Which gene is it? Routine diagnostic tests in horses will eventually involve collection of tissues to investigate gene expression or genotypes associated with various conditions. Breeders and veterinarians will not change the way they evaluate horses, but they will have more tools at their disposal. If a human, dog, cat or mouse genetic disorder has been genetically defined, it is likely that an identical genetic disease syndrome in a horse will have an identical or closely related gene

abnormality. This was the case for HYPP, JBE and GBED — a fact that made identification of the abnormal gene in horses that much easier.

And some day in the not-too-distant future, researchers will be trying to identify genes related to highly complex traits such as performance and behavior. Perhaps then, instead of asking *Which chromosome is the gene on?*, the question would be: *Could this horse be a winner?* ✤

PROFILE

Dr. Cecilia Penedo, Geneticist

Dr. Cecilia Penedo

Dr. Cecilia Penedo is one of the key geneticists in the UC Davis Veterinary Genetics Laboratory, where she has worked since 1982. For many years, she has supervised the genotyping service and has worked to develop genetic tests for livestock. She received her PhD in genetics from the University of California, Davis, in 1999 and was a graduate student and colleague of renowned equine geneticist Dr. Ann Bowling. Under Dr. Bowling's mentorship, Dr. Penedo conducted research on the genetics of coat color determination in horses and was intrigued by its complexity. After Dr. Bowling's sudden death in 2000, Dr. Penedo continued this line of research as well as branched out into other areas of horse genomics.

As Associate Director of the Veterinary Genetics Laboratory, Dr. Penedo also heads the Genomic Research and Development Unit there. She has focused her research on development of a genetic map of the horse and on gene mapping with a view to identifying the genes/mutations associated with traits and diseases. The end goal of this research is to develop diagnostic assays that can be used to screen horses for traits and diseases. Currently, she is working on gene mapping for the dun coat color, white spotting patterns tobiano and overo, and the genetic neurological defect known as cerebellar abiotrophy. In collaboration with researchers at the University of Minnesota, she has developed a newly released diagnostic assay for glycogen branching enzyme deficiency (GBE), which is a fatal neuromuscular genetic disease that affects horses with certain Quarter Horse bloodlines.

COMING EVENTS

You are cordially invited to attend the
Nineteenth Annual Charles Heumphreus Memorial Lecture on

Improving Management of Equine Laminitis

Saturday, January 22, 2005 – 9:00 am to 12:00 pm
170 Schalm Hall – Health Sciences Complex, UC Davis Campus

Presented by Dr. James Belknap, Professor, Department of Equine Internal Medicine and Surgery, College of Veterinary Medicine, The Ohio State University. This lecture series honors the memory of Charles Heumphreus, the School's farrier from 1967 to 1985, and is free of charge. For more information, contact the Office of Public Programs at (866)426-5693 (toll free) or visit their Web site at:
www.vetmed.ucdavis.edu/ce

HELPFUL TIPS

A Guide to Vaccinating Your Horses

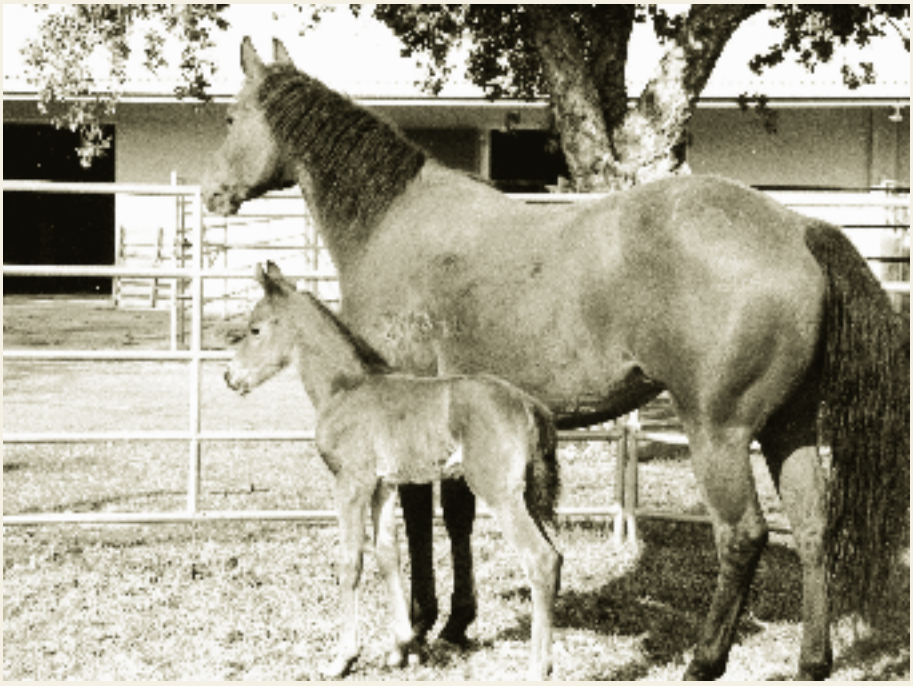
By W. David Wilson, BVMS, MS

Vaccination is important to an overall management program for controlling infectious diseases in horses. The decision to use a particular vaccine depends on the risk of acquiring infection and on the medical and economic consequences of infection balanced against the anticipated efficacy, cost, and potential adverse effects of the vaccination program.

Factors to consider when evaluating the risk of exposure to specific diseases include the age, type, gender, number, use, and stocking density of horses; season; environmental conditions; and the operation's facilities, management practices, and geographic location. For these reasons, there is no "standard" vaccination program that can be recommended for all horses. Each situation must be evaluated individually. Estimates of cost should include expenses incurred and money lost during the time horses are out of competition, labor and medication expenses for horses that develop clinical disease and require treatment, and the expenses in time, labor and vaccines required for proper immunization. Expectations should be realistic and should take into account the following realities:

• Vaccination reduces the risk of infection but does not prevent disease in all circumstances. No vaccine gives a 100% guarantee of protection.

• The horses in a population are not all protected to an equal extent or for an equal duration after vaccination.



• It takes several weeks or months after administration of the first dose of vaccine to induce protection. Optimal protection generally is not achieved until two to three weeks after completion of the primary series or for one or more weeks after administration of a booster dose to a previously vaccinated horse.

• The effectiveness of vaccines directed against different diseases varies considerably, as does the effectiveness of vaccines from different manufactureres directed against a particular disease.

• Whenever possible, all horses in a herd should be vaccinated on the same schedule. This simplifies record-keeping, minimizes replication and transmission of infectious agents in the herd, and optimizes herd immunity.

All horses should be vaccinated against tetanus, encephalomyelitis, and West Nile Virus. The use of other vaccinations depends on the individual risk of infection, as delineated in the following table, ***Vaccination Guidelines for Horses in the Western United States***.

VACCINATION GUIDELINES FOR HORSES IN THE WESTERN UNITED STATES*

W. David Wilson, BVMS, MS
School of Veterinary Medicine, University of California, Davis
November 2004

Disease/Vaccine	Foals/Weanlings	Yearlings	Performance Horses	Pleasure Horses	Broodmares†	Comments
Tetanus (inactivated toxoid) *Core vaccine - all horses*	***Foal of vaccinated mare:*** First dose: 6 months Second dose: 7 months Third dose**: 9 to 10 months ***Foal of nonvaccinated mare:*** First dose: 3 to 4 months Second dose: 4 to 5 months Third dose**: 6 to 8 months	Annual	Annual	Annual	Annual, 4 to 6 weeks before foaling	Booster at time of a penetrating injury or surgery if last dose of tetanus toxoid was not administered within the past 6 months.
Encephalomyelitis (EEE, WEE inactivated vaccine) *Core vaccine - all horses*	**WEE, EEE (in low-risk areas):** ***Foal of vaccinated mare:*** First dose: 6 months Second dose: 7 months Third dose**: 9 to 10 months ***Foal of nonvaccinated mare:*** First dose: 3 to 4 months Second dose: 4 to 5 months Third dose**: 6 to 8 months	Annual, spring	Annual, spring	Annual, spring	Annual, 4 to 6 weeks before foaling	For VEE, follow same protocol as for WEE/EEE if indicated by threat of exposure or requirements for interstate or international transportation. VEE may be available only as a combination vaccine with EEE and WEE.
	EEE: (in high-risk areas) First dose: 3 to 4 months Second dose: 4 to 5 months Third dose**: 6 to 8 months	Annual, spring	Annual, spring	Annual, spring	Annual, 4 to 6 weeks before foaling	In high-risk areas for EEE, booster EEE and WEE every 6 months. A series of at least three doses is recommended for primary immunization of foals.
West Nile Virus (WNV inactivated vaccine) *Core vaccine - all horses*	***Foal of vaccinated mare:*** First dose: 3 to 4 months Second dose: 4 to 5 months Third dose**: 6 to 8 months ***Foal of nonvaccinated mare:*** First dose: 2 to 4 months Second dose: 3 to 5 months Third dose**: 5 to 8 months	Semiannual (twice annually)	Semiannual	Semiannual	Semiannual; time one booster 4 to 6 weeks before foaling. Avoid administration to mares during the first 60 days of gestation if possible.	Peak seasonal exposure to WNV is in summer and fall. Time one booster in early to late spring to precede local seasonal mosquito activity and a second booster in mid- to late summer to precede expected peak local incidence of disease. Mosquito control is important for effective WNV prevention in both horses and humans.

* Appropriate application of these guidelines depends on specific assessment of risk on your particular premises by your veterinarian. As with the administration of all medications, the label and product insert should be read before administration of all vaccines.

** When □

□ ed according to risk.

Continued on pages 10 and 11

VACCINATION GUIDELINES — *Continued*

Disease/Vaccine	Foals/Weanlings	Yearlings	Performance Horses	Pleasure Horses	Broodmares†	Comments
Influenza *Add to core for horses experiencing contact with other horses at shows, events, competitions, and on breeding farms*	**Intranasal modified live virus:** First dose: 11 months Optional second dose 3 months later	Semiannual	Semiannual	Semiannual	Annual before breeding (see comments). Use an inactivated injectable influenza vaccine for pre-partum booster	Modified live intranasal vaccine is recommended as core of influenza vaccination programs because of demonstrated efficacy. If the first dose is administered to horses less than 11 months of age, administer a second dose at or after 11 months of age.
	Inactivated injectable: ***Foal of vaccinated mare:*** First dose: 9 months Second dose: 10 months Third dose**: 12 to 13 months Then at 4-month intervals ***Foal of nonvaccinated mare:*** First dose: 6 months Second dose: 7 months Third dose**: 9 to 10 months Then at 4-month intervals	Every 4 months	Every 4 months	Semiannual, or annual with added boosters prior to likely exposure	At least semiannual, with one booster 4 to 6 weeks pre-partum	For injectable influenza vaccines, a series of at least three doses is recommended for primary vaccination of foals, regardless of vaccination status of the dam.
Rhinopneumonitis (EHV-1 and EHV-4) *Add to core as outlined for influenza. All pregnant mares should be vaccinated against EHV-1*	First dose: 4 to 6 months Second dose: 5 to 7 months Third dose**: 7 to 10 months Then at 4-month intervals	Every 4 months if elected	Every 4 months if elected	Semiannual if elected	At 5, 7, and 9 months of gestation (inactivated EHV-1 vaccine); optional dose during third month of gestation.	Vaccination of mares with an EHV-1/EHV-4 combination vaccine prior to breeding is recommended. Vaccinate breeding stallions semiannually, with one of the doses timed before the start of breeding season.
Strangles *Add to core when risk of exposure is high, particularly on breeding farms*	**Intranasal live vaccine:** First dose: 4 to 6 months Second dose: 2 to 3 wks later Third dose**: 7 to 10 months	Semiannual	Optional: semiannual if risk is high	Optional: semiannual if risk is high	Semiannual, but use M-protein injectable vaccine for pre-foaling booster, 4 to 6 weeks before foaling	Use when endemic conditions exist or risk is high. Foals as young as 6 weeks-of-age have been vaccinated with the intranasal product but a 3rd dose should be administered before weaning.
	Injectable inactivated vaccine: First dose: 4 to 6 months Second dose: 5 to 7 months Third dose**: 7 to 9 months (depending on product used) Fourth dose: 12 months	Semiannual	Optional: semiannual if risk is high	Optional: semiannual if risk is high	Semiannual, with one dose of inactivated M-protein vaccine 4 to 6 weeks before foaling	Use when endemic conditions exist or risk is high.

* Appropriate application of these guidelines depends on specific assessment of risk on your particular premises by your veterinarian. As with the administration of all medications, the label and product insert should be read before administration of all vaccines.

** When □

□ ed according to risk.

VACCINATION GUIDELINES — *Concluded*

Disease/Vaccine	Foals/Weanlings	Yearlings	Performance Horses	Pleasure Horses	Broodmares†	Comments
Rabies (inactivated vaccine) *Add to core when significant risk of exposure to wildlife vectors of rabies exists*	***Foals of vaccinated mares:*** First dose: 6 months Second dose: 7 months Third dose: 12 months ***Foals of nonvaccinated mares:*** First dose: 3 to 4 months Second dose: 12 months	Annual	Annual	Annual	Annual, before breeding	Vaccination is recommended in endemic areas where potential exists for contact with wildlife vectors such as skunks, raccoons, foxes, badgers, and bats.
Potomac Horse Fever (inactivated vaccine) *Special circumstances only in endemic areas*	First dose: 5 to 6 months Second dose: 6 to 7 months A third dose in the primary series should be given if the first dose was given before 5 months.	4 to 6 month interval	4 to 6 month interval	4 to 6 month interval	4 to 6 month interval with one dose 4 to 6 weeks before foaling	Efficacy of vaccination protocols for prevention of Potomac Horse Fever is questionable. Booster during May to June in endemic areas if elected.
Botulism (Shaker Foal; inactivated type B toxoid) *Special circumstances only in endemic areas to protect foals*	***Foal of vaccinated mare:*** Three-dose series at 30-day intervals is best delayed until foals are 6 months old but can be started as early as 2 months of age	Not applicable	Not applicable	Not applicable	Initial three-dose series at 30-day intervals, with the last dose 4 to 6 weeks before foaling. Annually thereafter, 4 to 6 weeks before foaling	Only in endemic areas on breeding farms on which risk of infection is high. Protection of the foal is best accomplished by vaccinating the mare. Vaccination of young foals from nonvaccinated mares is commonly practiced but may not protect them during the first few months of life when they are most susceptible.
Equine Viral Arteritis (modified live vaccine) *Special circumstances only*	Intact colts intended for future use as breeding stallions: One dose at 6 to 12 months of age	Annual for colts intended to be breeding stallions	Annual for colts intended to be breeding stallions	Annual for colts intended to be breeding stallions	Annual for seronegative, open mares before breeding to carrier stallions; isolate mares for 21 days after breeding to carrier stallion	Use only under special circumstances. Annual for breeding stallions and teasers, 28 days before start of breeding season. Vaccinated mares do not develop clinical signs after breeding to carrier stallions even though they become transiently infected and may shed virus for a short time.
Rotavirus A (inactivated vaccine) *Special circumstances only on breeding farms*	Little value to vaccinate foal because there is insufficient time to develop antibodies to protect during susceptible age	Not applicable	Not applicable	Not applicable	Vaccinate mares at 8, 9, and 10 months of gestation, each pregnancy. Passive transfer of colostral antibodies aid in prevention of rotaviral diarrhea in foals.	Use on endemic farms or when risk of infection is high. Check concentrations of immunoglobulins at 24 hours of age to verify adequate passive transfer.

* Appropriate application of these guidelines depends on specific assessment of risk on your particular premises by your veterinarian. As with the administration of all medications, the label and product insert should be read before administration of all vaccines.

** When a third dose is recommended in the primary immunization series, this should be administered 8 to 12 weeks after the second dose.

† Schedules for stallions should be consistent with the vaccination program of the adult horse population on the farm and modified according to risk.

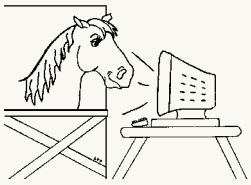
WEST NILE UPDATE

Summary of WNV Outbreak in California During 2004

As of November 4, 2004, there were 552 confirmed cases of West Nile Virus (WNV) in horses from 32 of 58 counties in California. Of these cases, 231 horses were reportedly dead or euthanized, and 321 horses survived. Although the vaccination status of these horses is not completely known, cases occurred in 239 nonvaccinated horses, 86 improperly vaccinated horses (that is, the vaccination series lapsed or was not completed), and 8 properly vaccinated horses. Note that many equine cases of WNV are neither confirmed nor reported.

The case definition for clinical WNV infection in horses requires compatible clinical signs, residency in California at the time of exposure, and a positive laboratory test result. For 2004, the California Department of Food and Agriculture is collecting information from horse owners and their veterinarians about the nature of the disease in all positive confirmed California horses. A standardized questionnaire is used to document relevant data from the owner and/or attending veterinarian, either in person, via telephone, or by mail.

There are currently two fully approved West Nile Virus vaccines available for horses. Each requires an initial series of at least two vaccinations, followed by periodic "booster" injections. The Center for Equine Health recommends that horse owners keep their horses properly vaccinated at all times against this disease. We suggest that you consult with your personal veterinarian regarding which vaccine is most appropriate for your particular horses and how often you should administer booster vaccinations.



Visit our Web site at
www.vetmed.ucdavis.edu/ceh
If you are accessing the *Horse Report* from our Web site and no longer want a paper copy, just let us know...save us the postage; the horses will benefit!
Send an e-mail request to **ljchristison@ucdavis.edu**

CEH
HORSEREPORT

Center for Equine Health
(530) 752-6433
www.vetmed.ucdavis.edu/ceh

Director:
Dr. Gregory L. Ferraro
e-mail: glferraro@ucdavis.edu

Writer/Editor:
Barbara Meierhenry
e-mail: cehwriter@ucdavis.edu

Management Services Officer:
Katie Castelli
e-mail: kacastelli@ucdavis.edu

Dean, School of Veterinary Medicine:
Dr. Bennie I. Osburn

The Center for Equine Health is supported with funds provided by the Oak Tree Racing Association, the State of California Pari-Mutuel Fund and contributions by private donors.

CEH
HORSEREPORT
Mail ID#1415
Center for Equine Health
School of Veterinary Medicine
University of California
One Shields Avenue
Davis, CA 95616-8589

RETURN SERVICE REQUESTED

Nonprofit Org.
U.S. POSTAGE
PAID
UC Davis