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Epidemiologic and Economic Study of Hyperelastosis Cutis/ HERDA in the Quarter Horse Cutting Industry

Sally Grant Tipton

EPIDEMIOLOGIC AND ECONOMIC STUDY OF HYPERELASTOSIS CUTIS/HERDA IN THE QUARTER HORSE CUTTING INDUSTRY

By

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Hyperelastosis Cutis (HC) is an autosomal recessive disorder in Quarter Horses. Homozygous (Hr/Hr) horses are affected with fragile, hyperextensible skin that sloughs and scars easily following minor trauma. Heterozygous (N/Hr) horses appear normal, but carry one copy of the gene. Objectives were to determine inbreeding coefficients of affected (Hr/Hr), carrier (N/Hr), and normal (N/N) Quarter Horses, compared to Thoroughbreds, and evaluate economic effects of HC within the cutting horse industry. Of the top cutting horses from 1985 through 2006, 35 were confirmed carriers by either DNA analysis or production of affected offspring. Although 23% of the earnings from the leading 100 lifetime earning sires ($388 million) were attributed to 12 carrier sires, average offspring earnings of carrier sires were not significantly different from normal sires. The increase in dollars is due to the number of earning offspring

produced by carriers, highlighting the need for DNA testing and appropriate breeding selections.

DEDICATION

I would like to dedicate this research to my husband, Harry B. (Tres) Tipton, III, and daughter, Nina for their patience, help, and encouragement; as well as to Buco Little Bit and 50% of her offspring.

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CHAPTER 1

INTRODUCTION

Significance

Hyperelastosis cutis (HC), also known as hereditary equine regional dermal asthenia (HERDA), is an autosomal recessive skin disorder in Quarter Horses (Rashmir et al., 2004, White et al., 2004, and Tryon et al., 2005). Heterozygous (N/Hr) horses are carriers of the recessive Hr gene, but appear normal, without physical signs of the disease. Horses that are homozygous recessive (Hr/Hr) are physically affected, and exhibit hyperextensible skin, extensive cutaneous lesions, skin sloughing, and characteristic scarring. Linebreeding, which is common in cutting horse bloodlines, can increase homozygosity of recessive genes. There is no known treatment for the disease.

Thesis Statement and Objectives of the Study

The hypothesis for this study was that selection pressure for performance traits associated with the Hr gene has resulted in higher inbreeding coefficients of affected (Hr/Hr) and carrier (N/Hr) horses, and may have impacted earnings in the cutting horse industry. The specific objectives of this study were to obtain information about the level of inbreeding within distinct groups of Quarter Horses, compared to Thoroughbreds as a control group, and evaluate the economic

effects of carrier (N/Hr) horses within the cutting horse industry compared to normal (N/N) horses. Information about the numbers of carriers (N/Hr) and their impact on the cutting horse industry is important in the development of breeding strategies. Selective breeding could reduce the number of animals that suffer from this disease and decrease financial losses incurred by owners.

CHAPTER 2

LITERATURE REVIEW

Related Diseases

Hyperextensible, fragile skin is a feature of many inherited skin disorders in animals and man. Ehlers-Danlos Syndrome (EDS) is the term used for the human disease and is a group of diverse, inherited connective tissue disorders that result from mutations in collagen genes. In other animals, including cattle, sheep, dogs, cats, mink, and rabbit, the disease has been identified as dermatosporaxis and cutaneous asthenia (Radostits et al, 2000 and Sequeira et al., 1999). In horses, the disease is referred to as hyperelastosis cutis (HC) or hereditary equine regional dermal asthenia (HERDA), (White et al., 2004).

Description and Diagnosis of the Disease

Hyperelastosis cutis in horses was first reported in 1978, (Lerner and McCracken, 1978). Genetic studies based on the clinical diagnosis of the HC phenotype in large numbers of progeny demonstrate that the skin disorder is inherited as an autosomal recessive mutation. If a carrier (N/Hr) is bred to a carrier (N/Hr), a 1:2:1 genetic ratio is observed with affected (Hr/Hr) male progeny occurring at the same rate as affected (Hr/Hr) female progeny.

Recessive genes are generally expressed phenotypically when two copies are inherited, one from the sire and one from the dam. The recessive Hr gene must be homozygous (Hr/Hr) or present in duplicate to be physically apparent in an affected horse. Carrier horses are heterozygous (N/Hr), with one copy of a normal gene and one copy of the Hr gene. However, carriers (N/Hr) do not appear to exhibit phenotypical manifestations of the disease, as the single recessive gene remains silent.

Skin consists primarily of collagen. When collagen attaches with elastin, the combination forms a strong connective material, which binds the skin layers together. Normal collagen fibers are uniform and tightly packed. However, collagen fibers in an affected (Hr/Hr) horse are damaged and disorganized. This weak connective material cannot stand up to external stress and the dermal layers separate. Therefore, affected (Hr/Hr) horses have hyperextensible skin that does not adhere properly to the body. Recently linkage analysis suggests that the HC mutation is linked to ECA1 in a region containing *PPIB* genes. These genes function in collagen metabolism (Tryon et al., 2007). However, much information is yet to be learned about the development of the disease.

Initial clinical diagnosis of HC in an affected (Hr/Hr) horse is based on the presentation of persistent lesions and characteristic scarring due to delayed, impaired healing (Pascoe and Knottenbelt, 1999 and Stannard, 2000). Seromas and hematomas, which are reported commonly, can be solitary or multiple and are located predominantly on the dorsal body surface. The DNA test can be used to confirm the clinical diagnosis and will identify the disease in pre-lesion

animals. The test utilizes hair or blood samples and detects affected (Hr/Hr), carrier (N/Hr), and normal (N/N) status (Lee, 2008). DNA analysis is available at Cornell University and the University of California at Davis.

An Equine Urinary Pyridinium Crosslink Assay screens for an elevated ratio of deoxypyridinoline:pyridinoline, two byproducts of collagen degradation, similar to human Ehlers-Danlos syndrome (Swiderski et al., 2006) . The urine test is diagnostic in the presence or absence of the disease, but does not identify the carrier (N/Hr) state.

Treatment and Prognosis of the Disease

No treatment exists except for minimization of environmental exposure and trauma, coupled with diligent wound care. Since affected horses' skin is fragile, they generally cannot be ridden because of subsequent wounds caused by the saddle. Due to the heritability of the disease, affected (Hr/Hr) horses are generally not retained for breeding, and are frequently euthanized. Nevertheless, the oldest affected (Hr/Hr) horse on record lived to an age of 17. Additionally there is a wide variation in the expression of the disease, as each horse brings its own unique genetic composition to bear on the expression of the disease. Outside factors such as climate, diet, stress level, and sex also appear to influence the severity of a horse's condition.

Proliferation of the Disease

Affected (Hr/Hr) animals are most commonly produced by carrier (N/Hr-to-N/Hr) matings. Statistically, 25% of the offspring from such matings will be homozygous (Hr/Hr) for the gene and will be affected with the skin defect; 50% of all offspring will inherit one copy of the Hr gene and become carriers (N/Hr); and 25% will be normal (N/N) both genotypically and phenotypically. The expected results of various breeding choices using Punnett squares are demonstrated in Table 2.1.

Table 2.1 Genotype and Phenotype Comparisons.

BREEDING CHOICE		GENOTYPES			PHENOTYPES	
		Carrier N/Hr	Affected Hr/Hr	Normal N/N	Normal Appearance	Physically Affected
Carrier x Carrier	(N/Hr x N/Hr)	50%	25%	25%	75%	25%
Carrier x Affected	(N/Hr x Hr/Hr)	50%	50%	0%	50%	50%
Carrier x Normal	(N/Hr x N/N)	50%	0%	50%	100%	0%
Affected x Affected	(Hr/Hr x Hr/Hr)	0%	100%	0%	0%	100%
Affected x Normal	(Hr/Hr) x N/N)	100%	0%	0%	100%	0%
Normal x Normal	(N/N x N/N)	0%	0%	100%	100%	0%

Punnett Square Examples:

Carrier (N/Hr)	Carrier (N/Hr)	
	N	**Hr**
N	N/N	N/Hr
Hr	Hr/N	Hr/Hr

Carrier (N/Hr)	Affected (Hr/Hr)	
	Hr	**Hr**
N	N/Hr	N/Hr
Hr	Hr/Hr	Hr/Hr

Carrier (N/Hr)	Normal (N/N)	
	N	**N**
N	N/N	N/N
Hr	Hr/N	Hr/N

Affected (Hr/Hr)	Normal (N/N)	
	N	**N**
Hr	Hr/N	Hr/N
Hr	Hr/N	Hr/N

For example:

- When a carrier (N/Hr) is bred to an affected (Hr/Hr) horse, half of the foals will be carriers (N/Hr) genotypically, although normal in appearance and half will be affected (Hr/Hr), both genotypically and phenotypically.

- When a carrier (N/Hr) is bred to a normal (N/N) horse, half of the offspring will be normal (N/N) genotypically and phenotypically, and half will be carriers (N/Hr) genotypically, but appear normal phenotypically.

- When an affected (Hr/Hr) horse is bred to another affected (Hr/Hr) horse, all of the offspring will be affected (Hr/Hr), since a homozygous animal can produce only one gene combination.

- When an affected (Hr/Hr) horse is bred to a normal (N/N) horse, none will be physically affected (Hr/Hr), but all of the offspring will be carriers (N/Hr) by genotype, although normal by phenotype.

As with any recessive trait, in any species, removing the carrier state from the population is difficult.

Pedigree Evaluations and Confirmation of an Autosomal Recessive Mode of Inheritance

The pedigrees of the affected (Hr/Hr) horses seen at MSU implicated a famous cutting horse bloodline, when a common link to Poco Bueno became apparent during pedigree searches. The evaluation of 75 pedigrees indicated that 96% trace back to Poco Bueno bloodlines within 6 generations and 4% trace back to Poco Bueno within the 7^{th} generation; this study also confirmed an autosomal recessive mode of inheritance. (Rashmir et al., 2004).

In 2005, a study based on 58 affected HERDA horses assessed the heritability and mode of inheritance of the disease. (Tryon et al., 2005). Heritability was estimated to be 0.38 ± 0.13, which is consistent with a heritable

locus. The study excluded an X-linked mode of inheritance for the disease, indicating that sex does not play a role in the heritability of the disease. Full pedigrees were available for 52 of 58 affected horses. Analyses revealed that all 52 affected horses were related to the same undisclosed stallion on both maternal and paternal sides of the pedigree, consistent with a single Mendelian autosomal recessive mode of inheritance. (Tryon et al., 2005).

Use of Linebreeding/Inbreeding Mating Strategies

Inbreeding is defined as the mating of individuals more closely related than the average for the population, or that results in the sire and the dam having common ancestors. (Bourdon, 2000). If parents are completely unrelated, there is no inbreeding, which is unlikely among animals of the same breed. Therefore, for practical purposes in this study, if two mated individuals had no common ancestor within the last five generations, their progeny were not considered inbred (Vogt et al., 2005).

The primary consequence of inbreeding is an increase in homozygosity, the probability that both genes of a pair are identical by descent (copies of a single ancestral gene). Within a breed, some proportion of all the genes will be homozygous. The inbreeding coefficient may be regarded as indicating what proportion of the remaining genes has been made homozygous by inbreeding. Calculation of inbreeding coefficients is an attempt to measure the probable percentage reduction in heterozygosity (increase in homozygosity), relative to a

base population. However, it is not a direct measure of homozygosity because the two alleles may be alike for other reasons.

Mean inbreeding coefficient for 52 affected horses with full pedigrees was 0.0261; four fold higher than the mean of 0.0066 for control horses (Tryon et al., 2005). Thirty of these horses had inbreeding loops that led to a calculated inbreeding coefficient greater than zero, ranging from 0.25 to 0.008. However, these numbers are representative of a subset of the population, as Quarter Horses represent a diverse breed, and selection has been used to produce numerous performance disciplines, such as cutting, reining, halter, pleasure events, and racing. Many of the assumptions associated with genetic analysis, such as random mating of an outbred population and Hardy-Weinberg Equilibrium, cannot be relied on when determining the genetic basis of the disease or easily extrapolated to the entire population. Additionally, stallions that have sired thousands of foals contribute disproportionately to the gamete pool, which can alter the gene frequency over time (Tryon et al., 2005).

Poco Bueno's genetic contribution has been multiplied through many generations, so the opportunity for carrier (N/Hr-to-N/Hr) matings has increased. Poco Bueno sired 405 AQHA registered first generation foals, 50% of which should be carriers (N/Hr). His second generation included 537 foals, 25% of which should be carriers (N/Hr), and his third generation included 1,352 foals, 12.5% of which should be carriers (N/Hr). These three generations alone total approximately 500 carriers (N/Hr) from Poco Bueno. According to AQHA, of the 3.2 million registered Quarter Horses that are still alive, 1.7 million trace back to

Poco Bueno, which is considered the fastest growing segment of the Quarter Horse population (Winand, 2008, personal communications).

Similarities to HYPP

An autosomal codominant disease in Quarter Horses, known as Hyperkalemic Periodic Paralysis (HYPP), causes uncontrolled muscle twitching and profound muscle weakness. HYPP is inherited as a codominant trait, which means a heterozygous (N/H) or affected stallion or mare bred to a homozygous (N/N) normal horse, will result in approximately half of the offspring being affected and half being normal.

Along with superior muscling desired by halter horse breeders, the sire Impressive was also the source of the gene for this disease. A genetic defect disrupts the sodium ion channel and results in an excessive amount of potassium in the blood, causing the muscles to contract more readily, potentially resulting in paralysis, cardiac arrest, or respiratory failure. However, in contrast to HC, HYPP is treatable through diet and medication in many cases.

HYPP is listed as a genetic defect in AQHA's regulations. Recommendations for DNA testing began in 1998. Foals of 2007 and later tracing to Impressive are required to be parentage verified and tested for HYPP, unless their Impressive-bred parent already has N/N results on file. Foals testing homozygous (H/H) for HYPP are no longer eligible for AQHA registration (2007 55th edition of the Official Handbook of AQHA's rule and regulations).

CHAPTER 3

MATERIALS AND METHODS

Phase I - Epidemiology

Identification of Carrier (N/Hr) and Affected (Hr/Hr) Horses

Carrier status (N/Hr) for the disease was determined by either DNA analysis or production of an affected (Hr/Hr) offspring by a phenotypically normal sire or a dam. Due to the autosomal recessive nature of the disease, production of an affected (Hr/Hr) offspring confirms that both sire and dam are carriers (N/Hr).

Selection of Groups

The top money winners and lifetime earning sires were evaluated to determine which carry the Hr gene. The inbreeding coefficients and relationship coefficients were calculated for four distinct groups of horses to address and compare differences in inbreeding levels between the groups, including:

- five randomly-selected DNA-confirmed (Hr/Hr) horses that were identified in this study as affected with the disease and known to be bred from cutting horse lines,
- the top 5 five-year leading cutting horse sires for years 2001 through 2005 that were DNA-confirmed carrier (N/Hr) horses,

- the top 5 five-year leading cutting horse sires for years 2001 through 2005 that were DNA confirmed or mathematically confirmed normal (N/N) horses, and
- the top 5 earning Thoroughbreds for years 2001 through 2005.

HC status of animals was confirmed by DNA analysis (patent pending) by Dr. Nena Winand at Cornell University's College of Veterinary Medicine Molecular Genetics Laboratory. In the absence of a DNA test, the normal (N/N) status of an animal was determined mathematically by examining the status of the progeny resulting from at risk matings using the AQHA database. Normal (N/N) status was considered to be confirmed when 100 or more matings to at risk pedigrees failed to produce an affected (Hr/Hr) individual. If the carrier status (N/Hr) rate among at risk pedigrees approaches 20%, lack of an affected (Hr/Hr) offspring following 100 matings to at risk pedigrees assures with greater than 99% accuracy that the animal in question lacks the Hr allele (Bourdon, 2000). The carrier status (N/Hr) rate among horses tested to date is 28% (Bannasch, 2008).

Calculation of Inbreeding Coefficients

SAS PROC INBREED was selected as the statistical software to calculate the inbreeding coefficients of the selected individuals within the four selected groups. The coefficient of relationship between individuals was also calculated in a covariance coefficient matrix. For individual X, the INBREEDING COEFFICIENT, which indicates the probability that the two alleles for any gene are identical by descent, is denoted by F_x and is equal to the coancestry between

its parents, or $F_x = f_{AB}$. The covariance coefficient between individuals X and Y, or COEFFICIENT OF RELATIONSHIP, is defined by $Cov(X,Y) = 2f_{XY}$, where f_{XY} is the coancestry between X and Y. (Falconer and Mackay, 1996 and SAS, 2004). Only a full DNA analysis can identify exactly how inbred these horses are; however, inbreeding probabilities or coefficients estimated through pedigree analyses can be used to predict the probable percentage of identical genes that were inherited as a function of the number and location of the common ancestors in the pedigree.

A five-generation pedigree of each horse (n=5), within each of the four groups, was formatted in a spreadsheet for using SAS PROC INBREED to calculate the inbreeding coefficient of each horse within each group. Coancestors (parents appearing on both the sire and the dam side of the pedigree) were identified and utilized in the computation of the individual's inbreeding coefficients. (SAS/Genetics® version 9.1 statistical software, SAS Institute Inc., Cary, NC).

Mean inbreeding coefficients within each group were assessed to determine if they were significantly different from zero (non-inbred) using the Student's t-test. A One-Way Analysis of Variance (ANOVA) with a Tukey's Studentized Range (HSD) Test was used to compare mean inbreeding coefficients among groups, where the independent variable was the group affected (Hr/Hr), carrier (N/Hr), and normal (N/N) Quarter Horse, or Thoroughbred) and the dependent variable was the mean inbreeding coefficient. Values were considered significant at $P \leq 0.05$. Means and descriptive statistics

were also calculated for each group using SAS® Enterprise© Guide 4.1 statistical software, (SAS Institute Inc., Cary, NC).

Phase II - Economics

Performance records for all ages and all divisions were obtained for the top 100 horses and top 100 lifetime earning (LTE) sires within the cutting horse industry for years 1985 through 2006, (EquiStat, 1986 – 2007), and are included in Appendix A. Economic data were obtained from Quarter Horse News and Equi-Stat, as well as the American Quarter Horse Association. Top cutting horses, reining horses, and reining cow horses were compared to the list of confirmed carrier (N/Hr) and affected (Hr/Hr) horses that was previously assembled. Carriers (N/Hr) were identified in several of the performance disciplines. However, due to the prevalence of the disease within the top cutting sires, this evaluation focused on the cutting horse industry only.

Annual Trends

Data were collected and analyzed for annual trends for top horses of all ages and all divisions, including:

- annual horses' winnings, all horses (stallions, geldings, and mares), all ages, all divisions, no information on offspring;
- leading sires' earnings, all ages, all divisions, including total offspring and average offspring earnings for each of the top sires in a given year; and
- leading lifetime earnings (LTE) of sires, all ages, all divisions, including total offspring earnings and average offspring earnings over each top sire's lifetime through 2006.

A linear model using a GLM Procedure (SAS® Enterprise© Guide 4.1 statistical software, SAS Institute Inc., Cary, NC) was used to identify significant differences at $P \leq 0.05$, where the dependent variable was averaged annual dollars of offspring earnings and the classification variables were year and Hr group.

Offspring Information

A One-Way Analysis of Variance (ANOVA), with a Tukey's Studentized Range (HSD) Test was used to compare the offspring numbers of carrier (N/Hr) sires to normal (N/N) sires for the leading 100 lifetime earning sires as of 2006, (SAS® Enterprise© Guide 4.1 statistical software, SAS Institute Inc., Cary, NC). In the first analysis, the dependent variables were the number of earning, performing, or total offspring and the independent variable was Hr group. In a different analysis, the dependent variable was average offspring earnings and the independent variable was Hr group. Values were considered significant at $P \leq 0.05$. Analysis of variance was additionally used to compare offspring and average earnings within the following groups:

- Top Five Carrier (N/Hr) sires to Top Five Normal (N/N) sires
- Top Ten Carrier (N/Hr) sires to Top Ten Normal (N/N) sires
- Top 25 Carrier (N/Hr) horses to Top 25 Normal (N/N) horses
- Top 50 Carrier (N/Hr) horses to Top 50 Normal (N/N) horses
- Top 12 Carrier (N/Hr) horses to Top 12 Normal (N/N) horses
- Top 12 Carrier (N/Hr) horses to Top 25 Normal (N/N) horses
- Top 12 Carrier (N/Hr) horses to Top 50 Normal (N/N) horses

Top 250 Five-Year Sires

Offspring earnings of the top five leading carrier (N/Hr) sires (DNA confirmed) were compared to that of the top five leading normal (N/N) sires (DNA or mathematically confirmed) for the five-year period from 2001 through 2005. The average offspring earnings per sire was calculated as the total earnings of all the sire's offspring during the five-year period divided by the number of offspring earning money during the period. A One-Way Analysis of Variance (ANOVA) with a Tukey's Studentized Range (HSD) Test was used to compare the mean values for each group of sires (carrier N/Hr vs. normal N/N), where the dependent variable was the average offspring earnings and the independent variable was the group, (SAS® Enterprise© Guide 4.1 Statistical Software, SAS Institute Inc., Cary, NC). Average offspring earnings were also correlated to inbreeding coefficients for the top five carriers (N/Hr) and top five normal (N/N) horses using a linear regression, with the inbreeding coefficient as the independent variable.

CHAPTER 4

RESULTS

Phase I - Epidemiology

Identification of Carrier (N/Hr) and Affected (Hr/Hr) Horses

A total 194 affected (Hr/Hr) horses were identified through DNA analysis and urine testing, or clinical presentation, 99 of which were mares and 63 of which were stallions, (Table 4.1). As an affected (Hr/Hr) horse was identified, pedigrees were obtained to determine the sire and dam, and thus establish them as carriers (N/Hr). Additionally, 283 offspring of known affected (Hr/Hr) horses carry at least one copy of the Hr gene. From this information, carrier (N/Hr) status was determined for 606 horses, 265 of which were mares and 285 of which were stallions.

Table 4.1 Horses Identified in Phase I by Sex and Status.

STATUS	Total	Geldings	Mares	Stallions	Unknown
Affected (Hr/Hr)	194	22	99	63	10
Carrier (NHr)	606	56	265	285	0
Total	800	78	364	348	10

As shown in Table 4.2, 2.1% of the total 1,634 horses identified in the annual top horses and leading sires for years 1985 through 2006, carry the Hr gene. Eight percent of the top 250 five-year sires for years 2001 through 2005 are carriers (N/Hr). Twelve percent of the top 100 leading lifetime earning (LTE) sires are carriers (N/Hr), as well as two percent of the dams in the top 100 LTE dams. Ten of the 35 carrier (N/Hr) horses were a top performer and producer, or in other words, 29% were both a superior horse and an outstanding sire. However, 12 of the 35 carrier (N/Hr) horses (or 34%) were top producers of great offspring, but have not been a top performer themselves.

Table 4.2 Summary of Performance Records from 1985 through 2006, indicating Horses identified as Carriers (N/Hr) vs. Normal (N/N).

Category Summary	Total Horses	Carrier Horses	% Carriers	Normal Horses	% Normal Horses
Total Performance Horses 1985-2006	1,634	35	2.1%	1,599	97.9%
Top 5-Year Sires 2001-2005	250	20	8.0%	230	92.0%
Leading Sires 2006	100	12	12.0%	88	88.0%
Leading Dams 2006	100	2	2.0%	98	98.0%

Comparison of Inbreeding Coefficients to Zero (Non-Inbred)

When horses have no common ancestors (no inbreeding), the inbreeding coefficient is zero. The mean inbreeding coefficients were calculated for groups of affected (Hr/Hr), carrier (N/Hr), and normal (N/N) Quarter Horses, and Thoroughbreds, as a control group. Table 4.3 displays the mean inbreeding coefficients for each group of five horses studied.

Table 4.3 Mean Inbreeding Coefficients for Each Group of Horses (n=5).

GROUP	Mean Inbreeding Coefficient	Std Error (SEM)	Std Dev	Student's *t*	*P*-Value
AFFECTED (Hr/Hr)	0.0514	0.0113	0.0252	4.5536	0.0104
CARRIER (N/Hr)	0.0238*	0.0102	0.0229	2.3278	0.0804
NORMAL (N/N)	0.0024*	0.0016	0.0037	1.4804	0.2129
THOROUGHBRED	0.0102	0.0035	0.0077	2.9398	0.0424

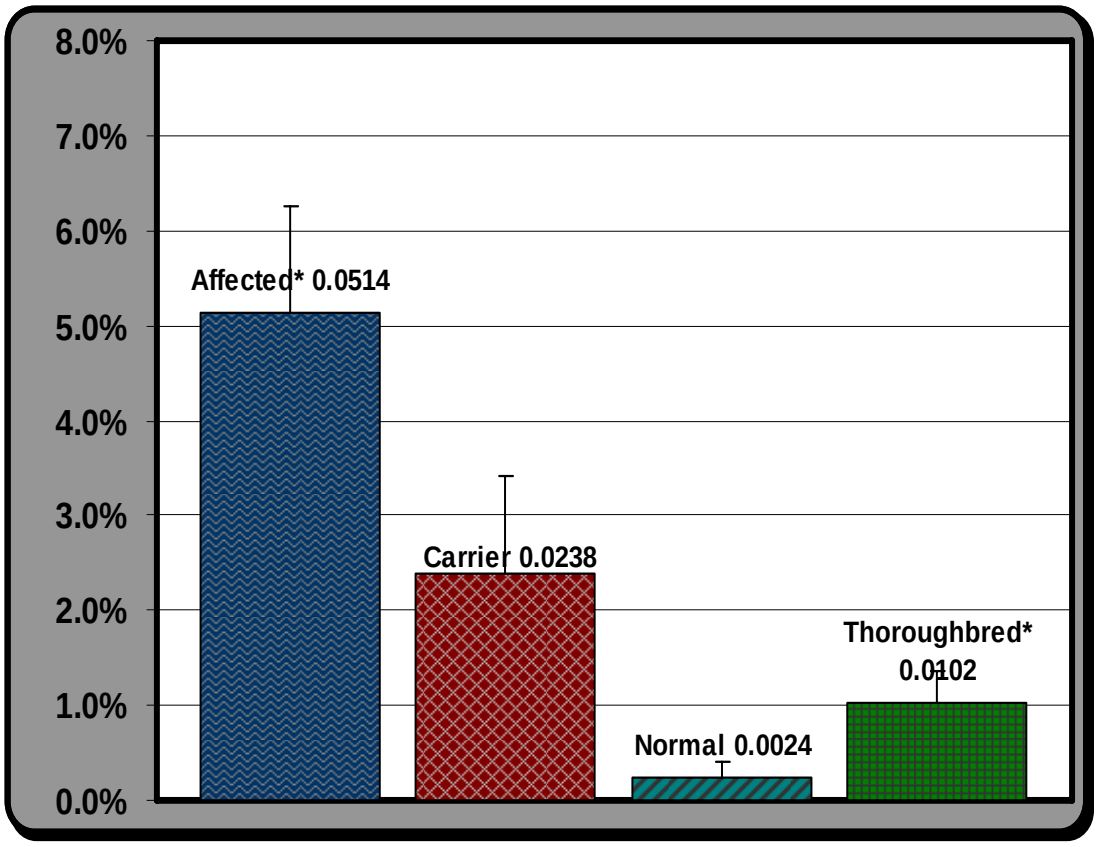
Within a column, means without * (a common superscript letter) differ ($P < 0.05$).

The mean inbreeding coefficient of horses in the affected (Hr/Hr) group was 0.0514, indicating they were significantly more inbred than non-inbred animals ($t = 4.5536$, $P = 0.0104$). The mean inbreeding coefficient for the carrier group (N/Hr) was 0.0238, which trended toward being significantly different from that of non-inbred horses ($t = 2.3278$, $P = 0.0804$). The inbreeding coefficient of horses in the normal (N/N) group was 0.0024, indicating that they were not significantly more inbred than non-inbred horses ($t = 1.4804$, $P = 0.2129$). Within

the four groups of horses studied, normal (N/N) horses were the least inbred and had the least variation in inbreeding coefficients among the groups. The group of Thoroughbreds had an inbreeding coefficient of 0.0102, which was significantly inbred compared to non-inbred animals (t = 2.9398, P = 0.0424).

Comparison of Inbreeding Coefficients among Groups

Inbreeding coefficients among the four groups are shown in Figure 4.1 and Table 4.4 (F = 7.50, P = 0.0024). Means with the same letter in Table 4.4 are not significantly different.



*Significantly different from zero (non-inbred) at $P \leq 0.05$.

Figure 4.1 Comparison of Mean Inbreeding Coefficients (y-axis) among Four Groups of Horses (x-axis), with Standard Error Bars.

Table 4.4 ANOVA Model and Comparison of Mean Inbreeding Coefficients among Four Groups of Horses (n=5).

Source	DF	Sum of Squares	Mean Square	F value	Pr>F
Model	3	0.0069	0.0023	7.50	0.0024
Error	16	0.0049	0.0003		
Corrected Total	19	0.0119			

STATUS	Mean Inbreeding Coefficient for group	Std Error	Tukey's Grouping*	
AFFECTED (Hr/Hr)	0.0514	0.0113	A	
CARRIER (N/Hr)	0.0238	0.0102	A	B
NORMAL (N/N)	0.0024	0.0016		B
THOROUGHBRED	0.0102	0.0035		B

*Means with the same letter are not significantly different at Alpha 0.05.

Horses in the affected (Hr/Hr) group were significantly more inbred than the horses in the groups of normal (N/N) horses and Thoroughbreds. The group of affected (Hr/Hr) horses was twice as inbred as the group of carriers (N/Hr); however, there was no significant between these two groups. Carriers (N/Hr) were ten times more inbred than normal (N/N) horses and twice as inbred as Thoroughbreds; however, the differences were not statistically significant. The

groups of normal (N/N) horses and Thoroughbreds were not significantly different from each other or from the group of carriers (N/Hr), but these two groups were significantly less inbred than the group of affected (Hr/Hr) horses. Although, the group of carriers (N/Hr) was not significantly different than any of the other three groups of horses, the inbreeding coefficient for carriers (N/Hr) was intermediate between the affected (Hr/Hr) horses and the normal (N/N) Quarter Horses and Thoroughbreds.

Phase II - Economics

A total 1,634 horses were identified in the published lists of the top money winning horses and leading lifetime earning sires from 1985 through 2006. As presented previously in Table 4.2, 35 of these horses were confirmed carriers (N/Hr), with 20 in the top 250 five-year sires for years 2001 through 2005, 12 in the leading lifetime earning (LTE) sires, and two in the top 100 lifetime earning dams.

Trends – Annual Winnings of Top Horses

Total annual winnings of carriers (N/Hr) and normal (N/N) horses were divided by the number of horses in each group to depict the averaged annual winnings for each group from 1990 through 2005, illustrated in Figure 4.2. Carrier (N/Hr) winnings increased an average of $4,575 per year and normal (N/N) horses increased an average of $2,112 per year. The differences in averaged annual winnings between carriers (N/Hr) and normal (N/N) horses and between years were statistically significant ($F = 131.67$, $P < 0.0001$, Table 4.5).

The greater increases in carrier (N/Hr) winnings compared to normal (N/N) horse winnings are most likely due to a greater number of earning offspring for carrier (N/Hr) sires compared to normal (N/N) sires. With carrier (N/Hr) winnings increasing at twice the rate of normal (N/N) horses, carriers (N/Hr) exceeded normal (N/N) horses as of 2001.

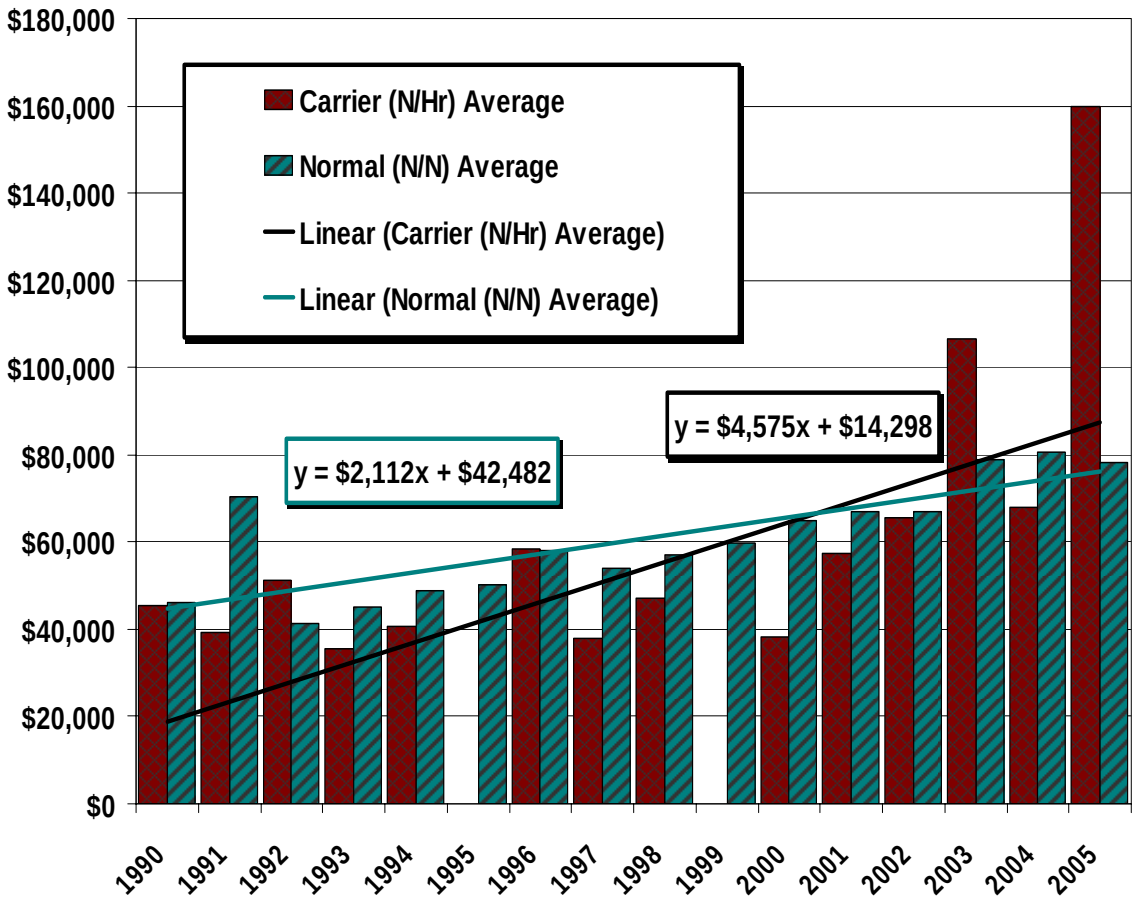


Figure 4.2 Averaged Annual Winnings for Carrier (N/Hr) and Normal (N/N) Horses for 1990 through 2005 including Stallions, Geldings, and Mares, (No Offspring Information).

Table 4.5 ANOVA GLM Procedure Model for Averaged Annual Winnings per Group of Horse per Year.

Source	DF	Sum of Squares	Mean Square	F value	Pr>F
Model	1	103,275,072,784	103,275,072,784	131.67	<0.0001
Error	31	24,314,456,986	784,337,322		
Corrected Total	32	127,589,529,770			

Trends – Annual Earnings of Leading Sires

A similar evaluation was conducted for leading sires from 1991 through 2006. Total annual earnings of carrier (N/Hr) and normal (N/N) sires were divided by the number of sires in each group. The averaged annual earnings for carrier (N/Hr) and normal (N/N) sires are illustrated in Figure 4.3. Carrier (N/Hr) sire earnings increased by an average of $63,207 per year compared to normal (N/N) sire earnings, which increased by an average of $24,525 per year. The differences between the two groups and between years were statistically significant ($F = 165.25$, $P < 0.0001$, Table 4.6). Again, earnings of carriers (N/Hr) increased more than 61% per year than normal (N/N) sires.

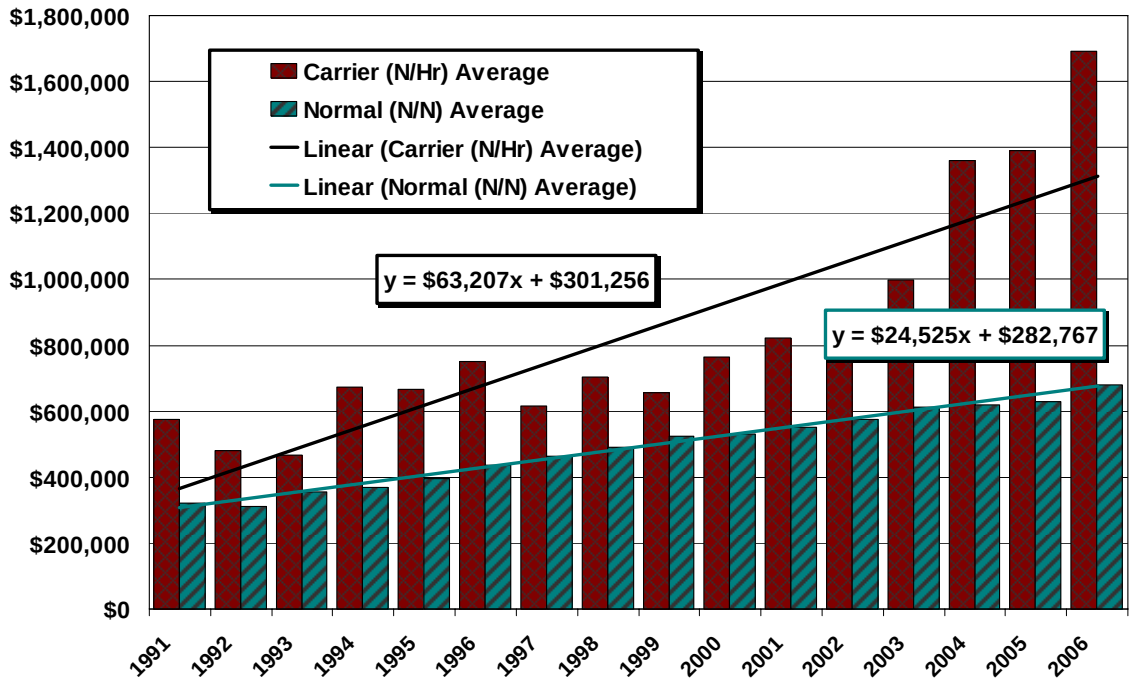


Figure 4.3 Averaged Annual Earnings of Carrier (N/Hr) and Normal (N/N) Sires for Years 1991 through 2006.

Table 4.6 ANOVA GLM Procedure Model for Comparing Average Offspring Earnings of Leading Annual Sires per Year.

Source	DF	Sum of Squares	Mean Square	F value	Pr>F
Model	1	1.5185855E13	1.5185855E13	165.25	<0.0001
Error	35	3.2164441E12	91,898,403,145		
Corrected Total	36	1.8402299E13			

Trends – Lifetime Earnings of Leading Sires

Total lifetime earnings (LTE) of the top 100 leading sires also were reported annually and included offspring earnings over the sire's lifetime. The total earnings of carrier (N/Hr) and normal (N/N) sires were divided by the

number of sires in each group to depict the averaged earnings per group from 1998 through 2006, illustrated in Figure 4.4. Although carrier (N/Hr) sires earned more money than normal (N/N) sires on average per year, their increase of $182,277 average per year was slightly less than the average increase of $187,532 per year for normal (N/N) sires. The difference between the two groups and between years were statistically significant (F = 83.27, P < 0.0001, Table 4.7).

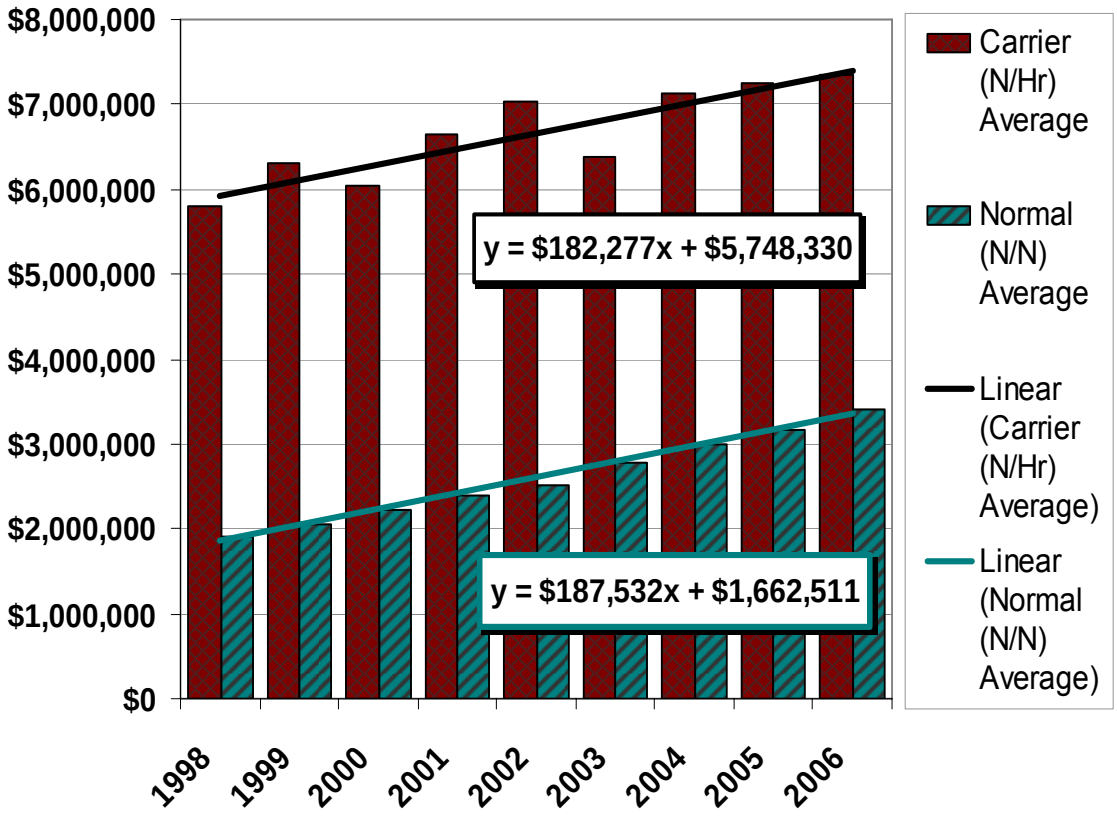


Figure 4.4 Averaged Total Lifetime Earnings of Offspring per Sire for Leading Lifetime Earning Carrier (N/Hr) and Normal (N/N) Sires.

Table 4.7 ANOVA GLM Procedure Model for Comparing Average Offspring Earnings of Lifetime Earning Carrier (N/Hr) and Normal (N/N) Sires per Year.

Source	DF	Sum of Squares	Mean Square	F value	Pr>F
Model	1	3.858546E14	3.858546E14	83.27	<0.0001
Error	17	7.8773874E13	4.6337573E12		
Corrected Total	18	4.6462848E14			

Figure 4.5 displays the percentage of carrier (N/Hr) earnings relative to the total earnings of both carrier (N/Hr) and normal (N/N) sires from 1998 through 2006. Earnings of carriers (N/Hr) increased an average of one-half percent annually.

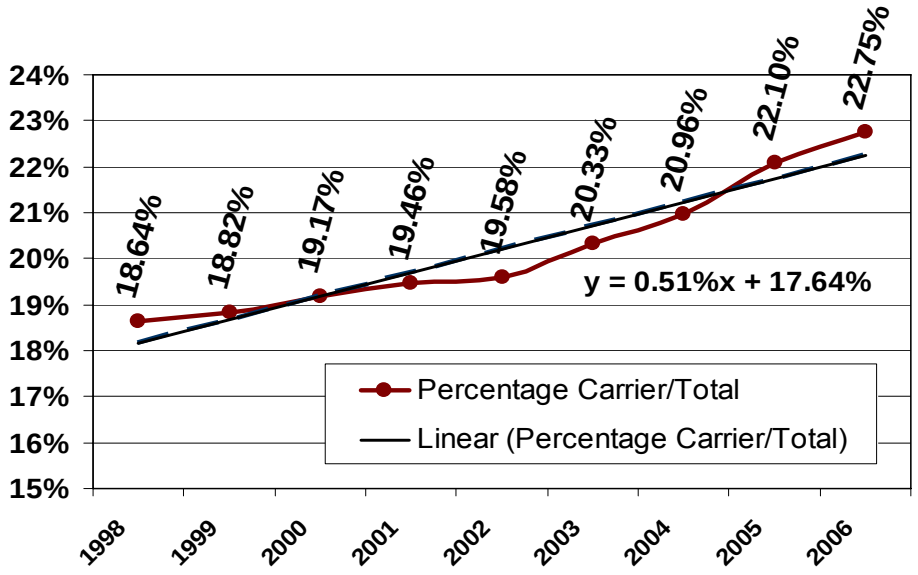


Figure 4.5 Percentage of Carrier (N/Hr) Sire Lifetime Earnings Relative to Total 100 Sire Lifetime Earnings (1998 through 2006).

As of 2006, earnings of the leading 100 lifetime earning sires were $388 million, of which 22.7% ($88 million) was attributed to 12% of the sires, which have been DNA confirmed as carriers (N/Hr).

Offspring Information

As of 2006, 30% of the progeny produced by carriers (N/Hr) were earning offspring, whereas 25% of the progeny produced by normal (N/N) sires were earning offspring (Table 4.8). In addition, 40% of the offspring produced by carriers (N/Hr) have performed, while only 36% of the offspring produced by normal (N/N) sires have performed.

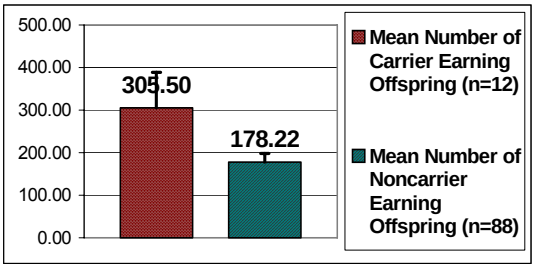
Table 4.8 Comparison of the Leading 100 Lifetime Earning Carrier (N/Hr) and Normal (N/N) Sires' Offspring Information including Average Earnings per Offspring and Numbers of Offspring (2006).

TOTAL OFFSPRING EARNINGS	Average Offspring Earnings/ 2007 SR	Total Number of Earning Offspring/ 2007 SR	Average Number of Earning Offspring/ 2007 SR	Average % Earning Offspring/ Total Offspring	Total Number of Total Offspring/ AQHA*
Carriers (N/Hr) n=12	$19,165	3,666	306	**30%**	11,168
Normal (N/N) n=88	$18,472	15,683	178	**25%**	57,850
Total		19,349	193		69,018
TOTAL OFFSPRING EARNINGS		Total Number of Performing Offspring/ AQHA*	Average Number of Performing Offspring/ AQHA*	Average % Performing Offspring/ Total Offspring	Average Number of Total Offspring/ AQHA*
Carriers (N/Hr) n=12		4,815	401	**40%**	931
Normal (N/N) n=88		22,387	257	**36%**	665
Total		27,202	275		697

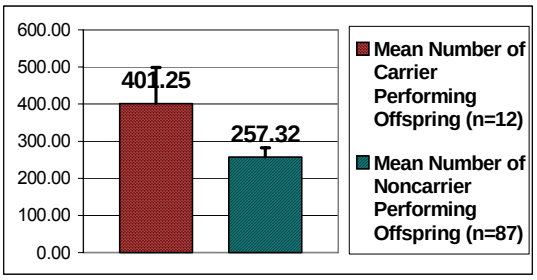
*Based on 99 horses

There was a significant difference in the number of earning offspring produced by carriers (N/Hr) compared to the number of earning offspring produced by normal (N/N) sires (F = 4.21, P = 0.0428). However, there was no

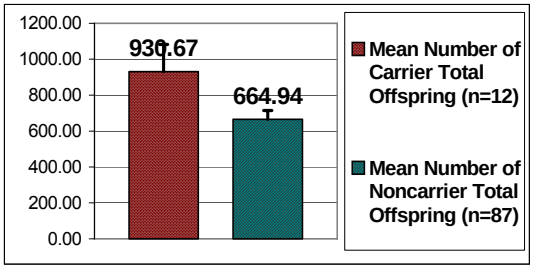
significant difference in the number of performing (F = 3.62, P = 0.0600) or total offspring (F = 3.46, P = 0.0658) between the two groups, Figure 4.6.



STATUS	Mean Number of Earning Offspring	n=	Std Error	Tukeys Grouping*
CARRIER (N/Hr)	305.50	12	82.82	A
NORMAL (N/N)	178.22	88	20.04	B
TOTAL	193.49	100	20.48	



STATUS	Mean Number of Performing Offspring	n=	Std Error	Tukeys Grouping*
CARRIER (N/Hr)	401.25	12	97.17	A
NORMAL (N/N)	257.32	87	24.80	A
TOTAL	274.77	99	25.01	



STATUS	Mean Number of Total Offspring	n=	Std Error	Tukeys Grouping*
CARRIER (N/Hr)	930.67	12	152.85	A
NORMAL (N/N)	664.94	87	48.73	A
TOTAL	697.15	99	47.18	

*Means with the same letter in the Tukey's Grouping are not significantly different at an Alpha 0.05.

Figure 4.6 Comparisons of Number of Earning, Performing, and Total Offspring of Carrier (N/Hr) and Normal (N/N) Sires as of 2006.

For comparison purposes, the same analyses were conducted on various combinations of leading sires and the number of offspring produced by carriers (N/Hr) vs. normal (N/N) sires. In each of the following cases, there was no significant difference in any of the number of offspring between the groups at a $P \leq 0.05$, not even in the number of earning offspring between the groups.

- Top Five Carrier (N/Hr) sires to Top Five Normal (N/N) sires
- Top Ten Carrier (N/Hr) sires to Top Ten Normal (N/N) sires
- Top 25 Carrier (N/Hr) horses to Top 25 Normal (N/N) horses
- Top 50 Carrier (N/Hr) horses to Top 50 Normal (N/N) horses
- Top 12 Carrier (N/Hr) horses to Top 12 Normal (N/N) horses
- Top 12 Carrier (N/Hr) horses to Top 25 Normal (N/N) horses
- Top 12 Carrier (N/Hr) horses to Top 50 Normal (N/N) horses

Average offspring earnings of the leading 100 lifetime earning sires as of 2006 averaged $19,165 for carrier (N/Hr) sires (n=12), which was greater than the average of $18,472 for normal (N/N) sires (n=88), illustrated in Figure 4.7. However, there was no significant difference between the two groups (F = 0.08, P = 0.7801, Table 4.9).

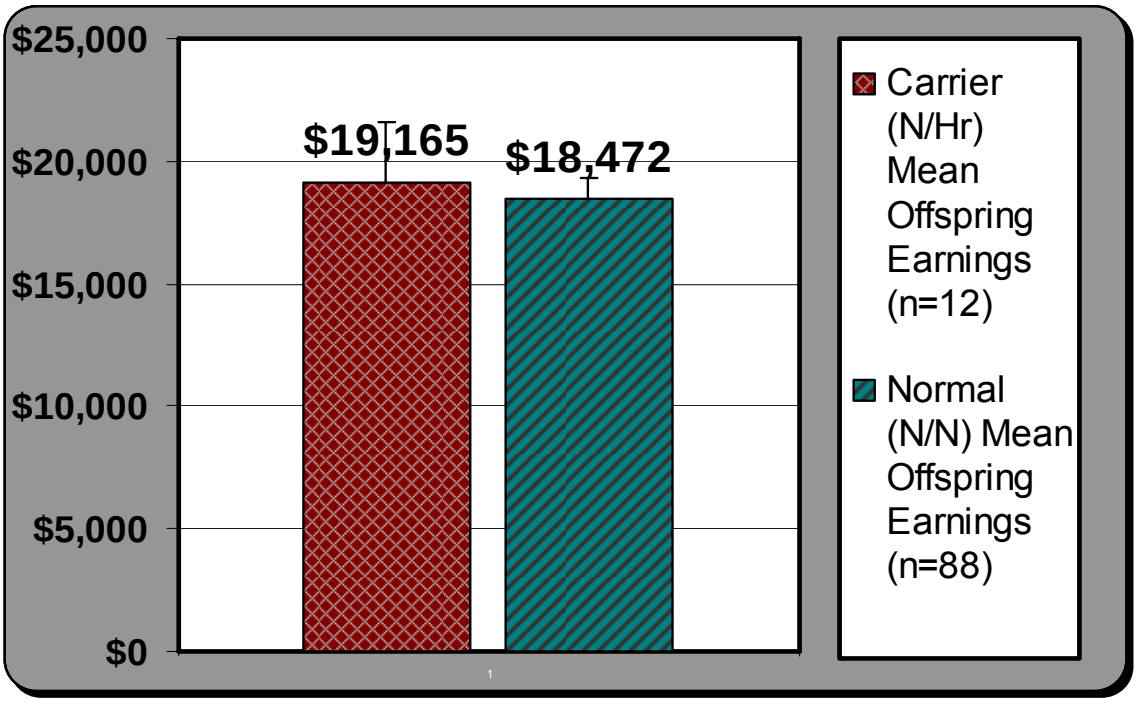


Figure 4.7 Comparison of Average Offspring Earnings of the Leading 100 Lifetime Earning Carrier (N/Hr) Sires and Normal (N/N) Sires as of 2006.

Table 4.9 ANOVA Model and Comparison of Average Offspring Earnings of the Leading 100 Lifetime Earning Carrier (N/Hr) Sires and Normal (N/N) Sires as of 2006.

Source	DF	Sum of Squares	Mean Square	F value	Pr>F
Model	1	5076753	5076753	0.08	0.7801
Error	98	6344972278	64744615		
Corrected Total	99	6350049031			

STATUS	n=	Mean Dollars $ of Earning Offspring	Standard Error	Tukeys Grouping*
CARRIER (N/Hr)	12	$19,165	$2,423	A
NORMAL (N/N)	88	$18,472	$853	A
	100	$18,555	$801	

*Means with the same letter are not significantly different at an Alpha 0.05.

Top 250 Five-Year Sires

Average offspring earnings of the top five carrier (N/Hr) sires (n=5) for the five-year period of 2001 through 2005, averaged $17,044, which was less than the average of $18,887 for normal (N/N) sires (n=5), illustrated in Figure 4.8. However, there was no significant difference between the two groups (F = 0.18, P = 0.6858), Table 4.10

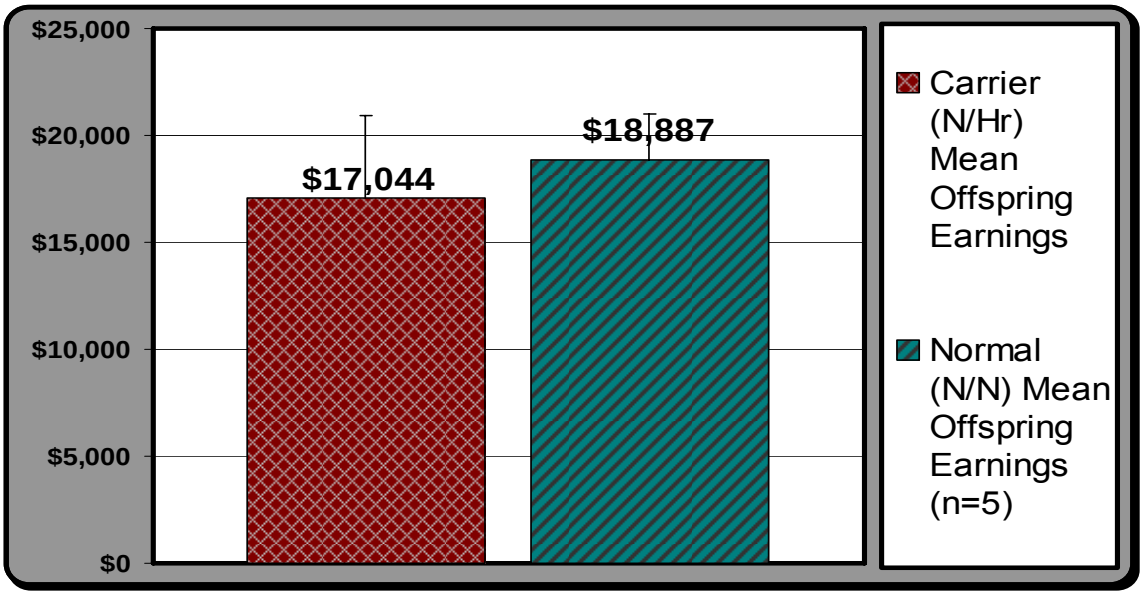


Figure 4.8 Comparison of Average Offspring Earnings of the Top Five-Year Carrier (N/Hr) Sires and Normal (N/N) Sires (2001 through 2005).

Table 4.10 ANOVA Model and Comparison of Average Offspring Earnings of the Top Five-Year Carrier (N/Hr) Sires and Normal (N/N) Sires (2001 through 2005).

Source	DF	Sum of Squares	Mean Square	F value	Pr>F
Model	1	8484252.1	8484252.1	0.018	0.6858
Error	8	385448358.4	48181044.8		
Corrected Total	9	393932610.5			

STATUS	n=	Mean Offspring Earnings $	Std Error	Tukey's Grouping*
CARRIER (N/HR)	5	$17,044	$3,854	A
NORMAL (N/N)	5	$18,887	$2,102	A
	10	$17,965	$2,092	

*Means with the same letter are not significantly different at an Alpha 0.05.

Comparison of Inbreeding Coefficients and Offspring Earnings

Inbreeding coefficients and average offspring earnings of the top five carriers (N/Hr) and top five normal (N/N) sires for 2001 through 2005 are compared in Figure 4.9.

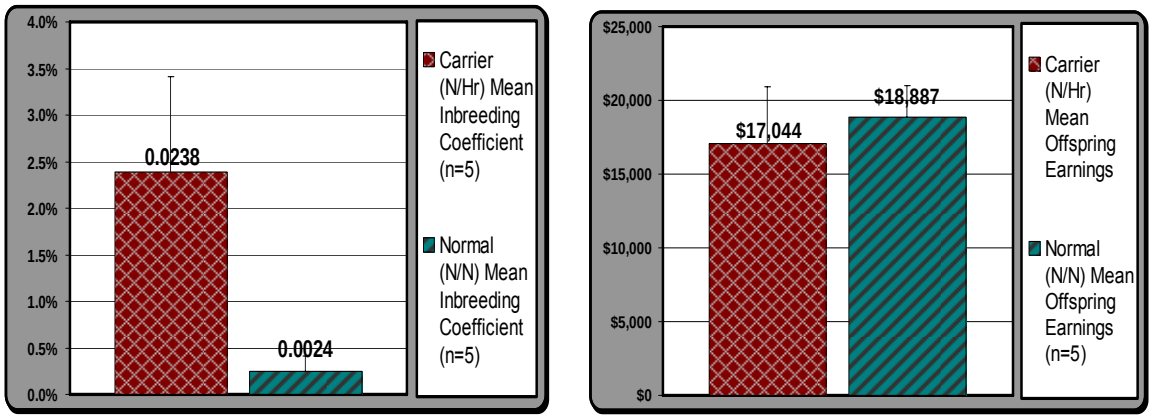


Figure 4.9 Comparison of Mean Inbreeding Coefficients of Top Five-Year Carrier (N/Hr) Sires and Normal (N/N) Sires and Comparison of Average Offspring Earnings of Top Five-Year Carrier (N/Hr) Sires and Normal (N/N) Sires (n=5).

Carriers (N/Hr) had a mean inbreeding coefficient of 0.0238, which was ten times greater than the inbreeding coefficient for normal (N/N) sires of 0.0024. Average offspring earnings of carrier (N/Hr) sires were $17,044 and average offspring earnings of normal (N/N) sires were $18,887. Neither inbreeding coefficients (Table 4.4) nor average offspring earnings (Table 4.10) were different between carriers (N/Hr) and normal (N/N) sires. Figure 4.10 displays the correlation of the effect of inbreeding coefficients on average offspring earnings of carrier (N/Hr) vs. normal (N/N) sires. A correlation value (r) for the relationship of inbreeding coefficients and average offspring earnings was 0.0070 for normal

(N/N) sires, indicating that there was almost no correlation between inbreeding coefficients and average offspring earnings of normal (N/N) horses. However, there was a positive correlation value between the two parameters for carrier (N/Hr) sires, which was 0.6578, indicating that as the level of inbreeding increases, the average offspring earnings increase.

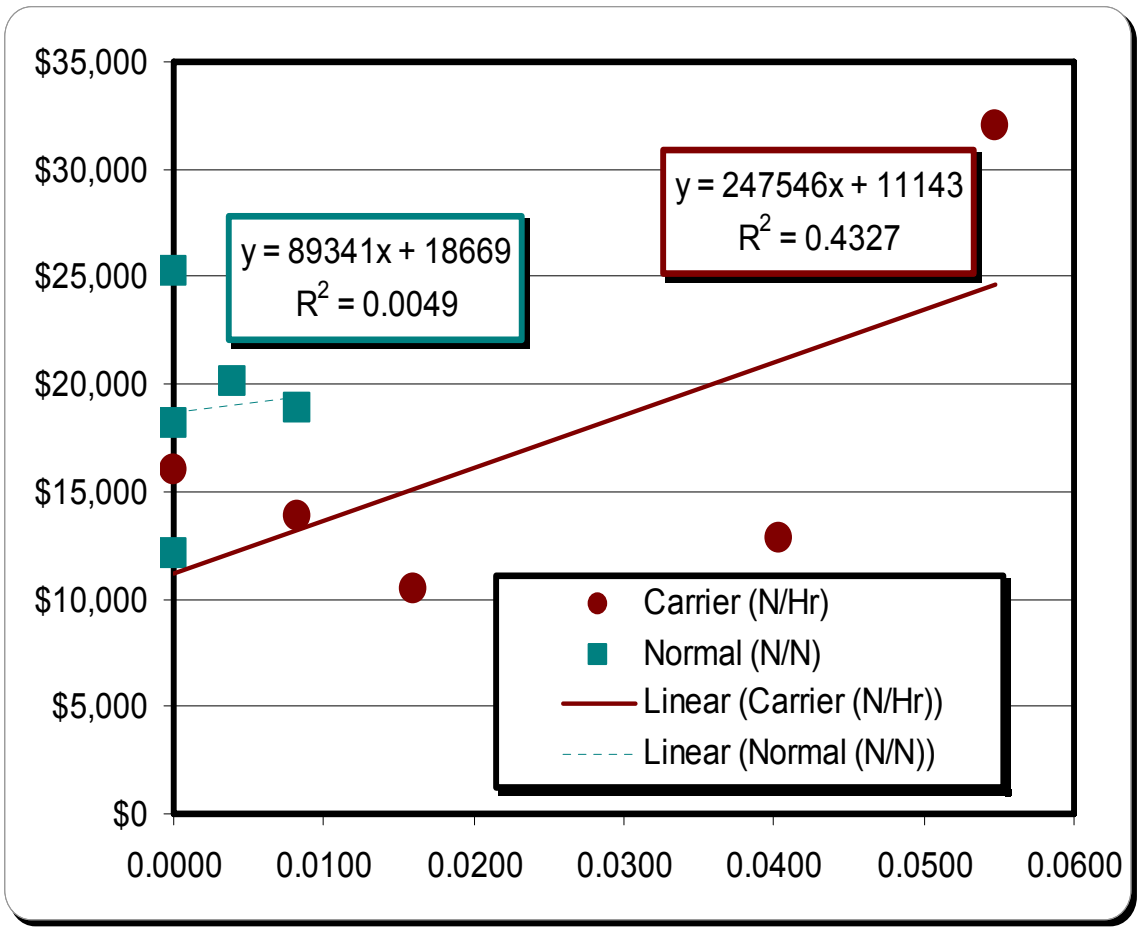


Figure 4.10 Correlation of Top Carriers (n=5) and Top Noncarriers (n=5) Inbreeding Coefficients vs. Average Offspring Earnings.

However, when the one outlier horse (a pre-eminent sire) for carriers (N/Hr) was removed in Figure 4.11, a correlation value of 0.4925 was calculated, indicating that as the level of inbreeding increases, the average offspring earnings decrease.

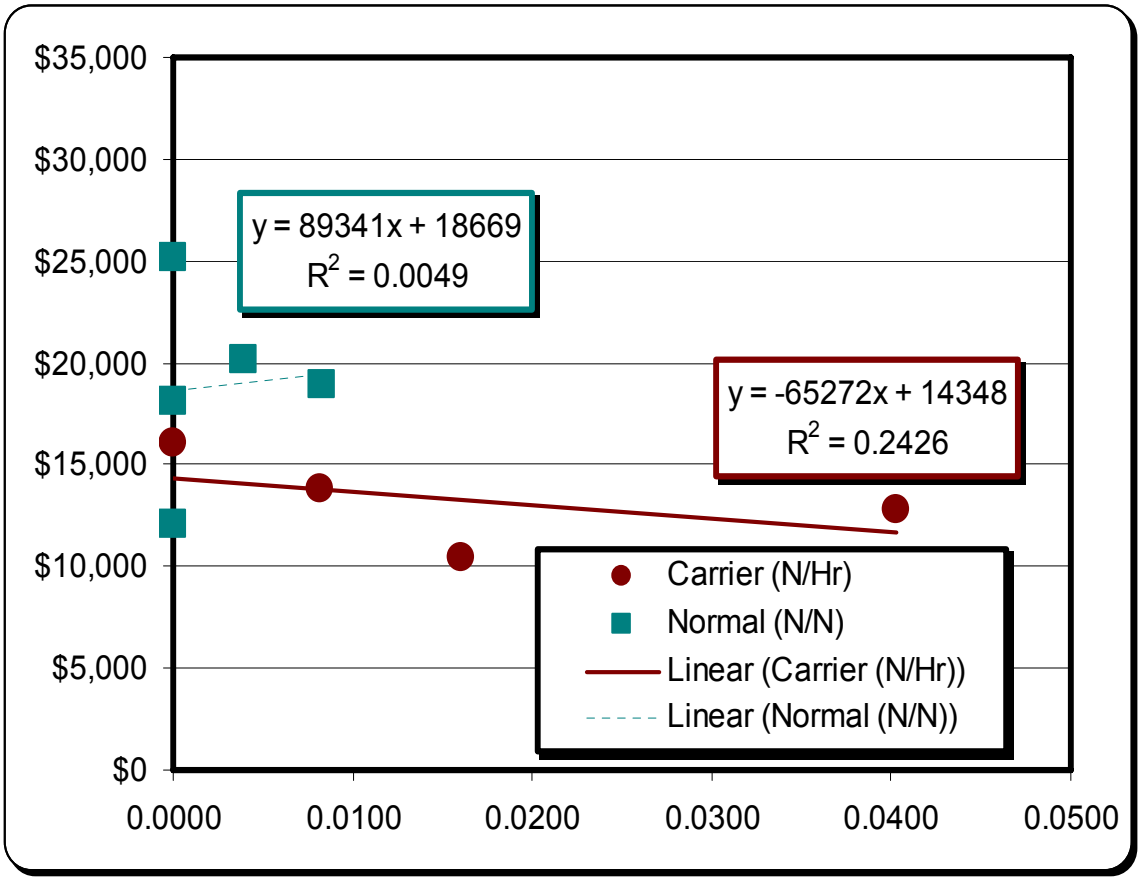


Figure 4.11 Correlation of Top Carriers (n=4 without outlier) and Top Noncarriers (n=5) Inbreeding Coefficients vs. Average Offspring Earnings.

A summary of the results presented in the tables and figures is provided in Appendix B.

CHAPTER 5

DISCUSSION

Phase I - Epidemiology

Linebreeding/inbreeding programs are used to maintain a degree of genetic relationship to an outstanding ancestor. Although animals with performance advantages can be produced, increased inbreeding can result in a higher frequency of hereditary abnormalities, such as HC in Quarter Horses, and eventually an overall reduction in performance due to inbreeding depression.

Compared to a non-inbred animal (zero inbreeding), the normal (N/N) horses in the group studied also were not inbred. In addition, normal (N/N) horses had the lowest inbreeding coefficient of the four groups. Although carriers (N/Hr) had an inbreeding coefficient of 2% and demonstrated a trend toward inbreeding, this was not statistically significant. The inbreeding coefficient of carrier (N/Hr) horses was considerably higher than normal (N/N) Quarter Horses and Thoroughbreds, although it was not statistically different from the inbreeding coefficient of any of the other groups. Affected Quarter Horses and Thoroughbreds were significantly inbred, with the group of affected (Hr/Hr) Quarter Horses being the most inbred of the groups studied. Affected (Hr/Hr) horses, which manifest the disease physically, were twice as inbred as carriers (N/Hr), 21 times more inbred than normal (N/N) horses, and five times more

inbred than Thoroughbreds, further demonstrating the negative effects of inbreeding. This finding is consistent with an increase in inbreeding levels and an increase in hereditary abnormalities due to increased homozygosity of harmful recessive alleles. Despite the harmful effects, inbreeding has been useful in the development of prepotent animals that uniformly stamp desirable characteristics upon their offspring by replicating genes for good conformation, athleticism, and attitude. However, it also exposes certain weaknesses within a breed such as HC.

Quarter Horses represent a diverse breed, and selection has been used to produce numerous performance disciplines, such as cutting, reining, halter, pleasure events, and racing. The inbreeding coefficients calculated for this study were representative of extremely small groups (n=5) of affected (Hr/Hr), carrier (N/Hr), and normal (N/N) Quarter Horses within the cutting industry and may not reflect true inbreeding levels. The same would be true for the Thoroughbred population, as the five Thoroughbreds chosen for the study were the top five money winners for the period 2001 through 2005. However, the relatively high inbreeding coefficient for this group of Thoroughbreds compared to normal (N/N) Quarter Horses in this study does indicate that elite Thoroughbreds are more inbred than the elite normal (N/N) Quarter Horses. This finding is not surprising in light of the closed studbook and the tremendous selection pressure within the Thoroughbred population. When a Thoroughbred does not win races, prospects for breeding are limited, often through gelding. However, Quarter Horses are

used in a number of disciplines, and if not successful in one, may be sold for use in another.

Phase II – Economics

Thirty five out of 1,634 of the top performers and leading producers (1985 through 2006) were confirmed carriers (N/Hr). Although many of the elite horses carry the Hr allele, including 12% of the leading lifetime earning sires and 2% of the leading lifetime earning dams, the majority of top performers and leading producers do not. The number of elite carriers (N/Hr) will most likely increase because additional horses will be DNA analyzed which test positive for the Hr allele. When elite carriers (N/Hr) are bred, more top money earners are produced, half of which will be carriers.

Annual winnings of carriers (N/Hr) and normal (N/N) horses indicate that carrier (N/Hr) winnings ($4,575) increased at a rate of twice that of normal (N/N) horses ($2,112) per year. Annual earnings of carrier (N/Hr) and normal (N/N) sires indicated that carrier (N/Hr) sires earnings increased an average of $63,207, which is also more than twice the rate of normal (N/N) sires ($24,525) per year. Averaged annual lifetime earnings (LTE) of the top 100 leading sires indicate that carrier (N/Hr) sires earned significantly more money than normal (N/N) sires on average per year, however, the increase was slightly less per year than that of normal (N/N) sires. Earnings of carrier (N/Hr) sires relative to the total earnings of all sires increased by an average of one-half percent

annually and, by 2006, 12% of the top 100 lifetime earning sires, which were DNA confirmed carriers (N/Hr), earned almost 23% of the money.

These increases in annual trends are attributed to the significantly higher ($P \leq 0.05$) average number of earning offspring produced by carrier (N/Hr) sires (30%) compared to the average number of earning offspring produced by normal (N/N) sires (25%). The average numbers of performing and total offspring produced by carrier (N/Hr) sires were also greater than the averages for normal (N/N) sires, although not statistically different. These findings indicate that numbers of carriers (N/Hr) are increasing, which is further evidence of the increase in affected (Hr/Hr) horses that are being reported, despite the fact the allele has been present since 1944 due to Poco Bueno.

Average offspring earnings between carrier (N/Hr) sires and normal (N/N) sires in either the leading 100 lifetime earning sires or the top five sires for 2001 through 2005 were not statistically different. This is also an indication that the number of earning offspring is a greater factor in annual dollar increases than the average amount of earnings won by offspring.

Finally, there was no correlation between inbreeding coefficients and average offspring earnings of normal (N/N) sires. Although, a positive correlation was initially observed for carrier (N/Hr) sires (n=5), when the one outlier carrier (N/Hr), a pre-eminent sire, was removed (n=4), average offspring earnings actually decreased as inbreeding increased, indicating that there actually may be no economic advantages to be gained by inbreeding in the cutting horse industry.

Conclusion

Heritability is the measure of consistency and reliability of the relationship between performance (phenotypic values) and breeding values (genotypic values) for a particular trait within a population. On average, when heritability is high (> 0.04), performance will be a good indicator of breeding value. Heritability of the Hr gene was estimated to be 0.38 (Tryon et al., 2005), between 0.2 and 0.4, which generally is considered moderate, although closer to the higher end of moderate. In contrast, cutting ability has a heritability of 0.12, or less than 0.2, which is considered a low heritable trait (Bourdon, 2000). When a trait is not very heritable, as with cutting ability, performance records of parents are not good predictors for progeny performance, which will be determined more by other factors, such as environment or training.

Linebreeding can increase homozygosity of desired traits of outstanding performance horses, as most likely the case with both normal (N/N) and carrier (N/Hr) Quarter Horses. However, it also can express negative traits, as the case with affected (Hr/Hr) Quarter Horses. Within the general Quarter Horse population, the Hr allele frequency is 0.02. However, cutting horses have a significantly higher allele frequency of 0.14, indicating that the disease is much more prevalent within cutting horse industry (Bannasch, 2008). DNA analysis is required to identify the difference between a phenotypically and genotypically normal (N/N) horse, and a phenotypically “normal” (N/Hr) horse that genotypically carries the recessive gene. However, the carrier (N/Hr) horse will

transmit the undesirable gene to half of its progeny or foals, thus perpetuating the transmission of the disease.

Genetic protocols provide the means to increase the incidence of this disease through super ovulation, artificial insemination, and embryo transfer. One stallion may breed as many as 200 mares in one season, which could amount to the production of several thousand foals in his lifetime, and mares can potentially produce three or even four viable embryos within the same year. These techniques are altering the gene frequencies over time, as owners and breeders seek out the top performing horses and leading offspring producers for breeding choices.

Stud fees on some of these leading sires are in excess of $20,000 and embryo transfer fees may exceed $3,500. Thousands of dollars are invested in a foal before parturition. When the disease goes unrecognized until the colt or filly enters training, at two or more years of age, substantial dollars may have been expended. When a carrier (N/Hr) is bred to another carrier (N/Hr), the breeder or owner is at risk of losing 100% of this investment, 25% of the time when an affected (Hr/Hr) foal is produced. This loss can be prevented by a DNA test, which is a highly reliable but relatively inexpensive tool. The DNA analysis will confirm the absence of a carrier (N/Hr) to carrier (N/Hr) mating, and eliminate the possibility of producing an affected (Hr/Hr) offspring.

Additionally, an affected horse is more likely to be euthanized at a young age, because of high maintenance for wound management and lack of productivity potential due to breeding concerns.

The American Quarter Horse Association (AQHA) ultimately decides the outcome of animals with genetic defects within the United States registry, as in the cases of HYPP, parrot mouth, and cryptorchidism. However, the Australian Quarter Horse Association has already implemented requirements for HERDA testing in Australia. As of January 1, 2008, all new Foundation Stock recording applications will require HERDA testing on top of the already required DNA, OLWS, and HYPP testing. A negative test must be achieved before they will be accepted into the Foundation. Additionally, all stallions registered in Australia as approved breeding sires will be required to have their HERDA status on record with the Association and printed on their Certificate of Registration. (Appendix C).

In addition to the financial concerns and registration requirements facing breeders and owners, there are ethical questions regarding an owner's obligation to disclose carrier (N/Hr) status, as well as an owner's legal recourse if the resulting foal is affected (Hr/Hr) with the disease (Appendix D). These issues will be resolved by the horse market or in the court. However, DNA analysis and appropriate breeding selections can prevent the production of affected horses that suffer from this disease, reduce economic losses incurred by owners and breeders, and moderate the perpetuation of the disease within the Quarter Horse population. As with any recessive trait, in any species, removing the carrier state from the population is difficult.

The results of this epidemiologic and economic survey of HC emphasize the importance of DNA testing, particularly when pedigree evaluations indicate

Poco Bueno bloodlines. According to AQHA, 1.7 million Quarter Horses trace back to the great sire, Poco Bueno. Thus, pedigree evaluations will continue to provide insight as to the horse's lineage and can be an effective screening tool in determining whether to utilize a DNA test. Additional information would also be gained by the evaluation of sale price of carrier (N/Hr) offspring compared to normal (N/N) offspring.

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APPENDIX A

LIST OF ECONOMIC DATA

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Table A.1. Summary List of Economic Data and Number of Horses Included in Each Year

		Annual Earnings		Lifetime Earnings		
		QHN	QHN	QHN	QHN	SR
YEAR	Date of Publication	TOP HORSES/ All Ages/All Divisions ANNUAL	EADUNG SIRES/All Ages/All Divisions ANNUAL	LTE HORSES/ All Ages/All Divisions	LTE SIRES/All Ages/All Divisions	LTE SIRES/All Ages/All Divisions
1985	14-Mar-86	1-25				
1986	27-Feb-87	1-25				
1987	26-Feb-88	1-25				
1988	24-Feb-89	1-100	1-25			
1989	9-Mar-90	1-100	1-30			
1990	8-Mar-91	1-100		1-100		
1991	13-Mar-92	1-100	1-30	1-100	1-40	
1992	12-Mar-93	1-100	1-30	1-100	1-30	
1993	11-Mar-94	1-100	1-30	1-100	1-25	
1994	10-Mar-95	1-100	1-30	1-100	1-25	
1995	8-Mar-96	1-100	1-30			
1996	14-Mar-97	1-100	1-30	1-100	1-30	
1997	13-Mar-98	1-100	1-30	1-100	1-30	
1998	15-Mar-99	1-100	1-30			1-100
1999	15-Mar-00	1-100	1-30			1-100
2000	15-Mar-01	1-100	1-30			1-100
2001	15-Mar-02	1-100	1-30			1-100
2002	15-Mar-03	1-100	1-30			1-100
2003	15-Mar-04	1-100	1-30			1-100
2004	15-Mar-05	1-100	1-30			1-100
2005	15-Mar-06	1-100	1-30			1-100
2006	15-Mar-07	1-100	1-30			1-100

Table A.1 (continued)

		Annual Earnings		Lifetime Earnings		
		QHN	QHN	QHN	QHN	SR
YEAR	Date of Publication	TOP HORSES/All Ages/All Divisions ANNUAL	LEADING SIRES/All Ages/All Divisions ANNUAL	LTE HORSES/ All Ages/All Divisions	LTE SIRES/All Ages/All Divisions	LTE SIRES/All Ages/All Divisions
5-YEAR SIRES/ All Ages/All Divisions 2001 - 2005	15-Jan-07				1-250	
LTE DAMS/ All Ages/All Divisions	15-Feb-07				1-100	
LTE SIRES/ All Ages/All Divissions	1-Jul-07				1-100	
Top Horses/ All Ages/All Divisions Money won thru 2006	1-Jul-07			1-100		

APPENDIX B

SUMMARY OF RESULTS

APPENDIX B

SUMMARY OF RESULTS

Phase I – Epidemiology

Identification of Carrier (N/Hr) and Affected (Hr/Hr) Individuals

Table 4.1 Horses Identified in Phase I by Sex and Status

- **800 Carriers, 45% female 53% male**

Table 4.2 Summary of Performance Records from 1985 – 2006 indicating Horses identified as Carriers (N/Hr) vs. Normal (N/N) Horses

- **98% are Normal, including 92% of top 250, 98% of top dams, 88% of top LTE sires**

Comparison of Inbreeding Coefficients to Zero (Non-Inbred)

Table 4.3 Mean Inbreeding Coefficients for Each Group of Horses

- **Affected and Thoroughbreds were inbred**
- **Carriers tended towards inbreeding**
- **Normal were not inbred**

Comparison of Inbreeding Coefficients among Groups

Figure 4.1 Comparisons of Mean Inbreeding Coefficients of Four Groups of Horses

Table 4.4 ANOVA Model and Comparison of Mean Inbreeding Coefficients among Groups

- **Affected and Carriers were not different from each other**
- **Carriers, Normals, and Thoroughbreds were not different from each other**
- **Only Affected were different than Normals and Thoroughbreds**
- **Affected had highest inbreeding coefficient; Normals had lowest inbreeding coefficient**

Phase II - Economics

Annual Trends – Annual Winnings for Horses

Figure 4.2 Mean Annual Winnings for Carrier (N/Hr) and Normal (N/N) Horses

Table 4.5 ANOVA GLM Procedure Model only

- **Carrier winnings are increasing twice as fast as Normals**
- **With more Carriers winning, so more are bred, producing 50% more Carriers that are winning**
- **As of 2001 Carriers were winning more than Normals**

Annual Trends – Annual Earnings for Sires

Figure 4.3 Mean Annual Earnings for Carrier (N/Hr) and Normal (N/N) Sires

Table 4.6 ANOVA GLM Procedure Model only

- **Carriers earn more than Normals (as early as 1991, when these data were first reported)**

- **Carrier earnings increase at a more rapid rate than Normals by 61%**

Annual Trends – Lifetime Earnings for Sires

Figure 4.4 Mean Lifetime Earnings of offspring per sire for leading LTE Carrier (N/Hr) and Normal (N/N) Horses

Table 4.7 ANOVA GLM Procedure Model only

- **Carriers earn more than Normals (1998-2006)**
- **Carrier rate of increase is slightly less than Normals**

Figure 4.5 Percentage of Carrier (N/Hr) Lifetime Earning Sires to Top 100 Lifetime Earning Sires per Year

- **Earnings of Carriers increase by ½% every year**
- **23% of earnings attributed to 12% of sires**

Numbers of Offspring - Lifetime Earning Sires

Table 4.8 Comparison of Leading 100 Lifetime Earning Carrier (N/Hr) and Normal (N/N) Offspring Information including Average Earnings per Offspring and Numbers of Offspring (2006)

- **Greater average percentage of earning offspring produced by Carriers compared to Normal sires**
- **True for average percentage of performing offspring**

Figure 4.6Comparisons of Number of Earning, Performing, and Total Offspring of Carrier and Normal Sires as of 2006

- **Average numbers of earning, performing, and total offspring are greater for carriers than normals**
- **The only difference that was statistically significant was earning offspring**

Average Offspring Earnings - Lifetime Earning Sires

Figure 4.7 Comparison of Average offspring earnings of the leading 100 LTE earning carrier and normal sires 2006

- **Average offspring earnings were greater for carriers than normals**

Table 4.9 ANOVA Model **and** Comparison of Average Offspring Earnings of the Leading 100 Lifetime Earning Carrier (N/Hr) Sires and Normal (N/N) Sires as of 2006

- **Differences were not statistically significant**

Average Offspring Earnings - Top 250 Five-Year Sires

Figure 4.8 Comparison of average offspring earnings of the top five-year Carrier (N/Hr) sires and Normal (N/N) sires from 2001 through 2005

- **Average offspring earnings were less for carriers than normals**

Table 4.10 ANOVA Model **and** Comparison of average offspring earnings of the top five-year Carrier (N/Hr) sires and Normal (N/N) sires from 2001 through 2005

- **Differences were not statistically significant**

Comparison of Inbreeding Coefficients and Offspring Earnings

Figure 4.9Comparison of Mean Inbreeding Coefficients of leading five-year Carrier (N/Hr) and Normal (N/N) sires and Average Offspring Earnings of leading five-year Carrier (N/Hr) sires and Normal (N/N) sires

- **Carrier inbreeding coefficient were higher than Normals**
- **Average offspring earnings were greater for Normals compared to Carriers**
- **The differences in inbreeding coefficient and earnings were not statistically significant**

Figure 4.10Linear Regression of Top Five Carriers (N/Hr) and Top Five Normal (N/N) Leading Five-Year Sires Inbreeding Coefficients vs. Average Offspring Earnings

- **No correlation between inbreeding coefficients and average offspring earnings for normals (n=5)**
- **Carrier (n=5) average offspring earnings increase as inbreeding coefficients increase**

Figure 4.11Linear Regression of Top Four Carriers (N/Hr) (without outlier) and Top Five Normal (N/N) Leading Five-Year Sires Inbreeding Coefficients vs. Average Offspring Earnings

- **Outlier in carriers is removed (n=4) and average offspring earnings decrease as inbreeding coefficients increase**

APPENDIX C

AUSTRALIAN QUARTER HORSE ASSOCIATION HERDA REQUIREMENTS

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AUSTRALIAN QUARTER HORSE ASSOCIATION HERDA REQUIREMENTS

Registry Services Update - HERDA
22 January 2008
Anna Stevenson

AQHA have been successfully facilitating the HERDA testing procedure through Cornell University in the USA for the last 2 months. We have received a wonderful response from members, who have been ordering HERDA testing kits through our office for the special discounted price of $55 which AQHA negotiated with Cornell for our members. The most encouraging thing has been members ordering kits for their breeding mares, even though AQHA have not requested it at this time. This shows us that members are keen to support our steps to control the progression of HERDA and ultimately eradicate this crippling disease in Australia.

AQHA have implemented the new requirements for HERDA testing, which have come into effect since 1 January 2008. We hope that members will continue their support by adhering to the below requirements. It is important to note that horses already registered with AQHA that test positive (N/Hr or Hr/Hr) to this disease will not be de-registered. At this point all new registrations coming through for horses born in Australia that test positive (N/Hr or Hr/Hr) will be accepted for registration. Only imported horses testing positive (N/Hr or Hr/Hr) will be affected by the new HERDA requirements for 2008. Please see points below.

1.Effective 1 September 2007, any horses purchased or embryos harvested overseas **prior to** 31 December 2007 will be accepted for Registration with the Association but will require HERDA testing not later than 31 March 2008 and have the result marked on their Certificate of Registration.

2.Effective 1 January 2008, any horses purchased or embryos harvested overseas **after** 31 December 2007 must be tested for HERDA by 31 March 2008. Any horse testing positive to HERDA (N/Hr or Hr/Hr) will not be eligible for AQHA registration.

3.Effective 1 January 2008, AQHA will only accept the results of HERDA testing facilitated by AQHA through Cornell University. Results received from testing done by members direct through Davis, Cornell or any other testing facility will no longer be accepted. If you would like to test your horse for HERDA and have the results accepted by AQH A, you must have been issued an AQHA

HERDA testing kit. These are available for $55 through the office.

4.Effective 1 January 2008 all stallions registered with AQHA as approved breeding sires (IBF endorsed or AQHA classified) will be required to have their HERDA status on record with the Association and printed on their Certificate of Registration by **31 July 2008**. Any breeding sire without HERDA testing having been completed by this date may have Stallions Returns held or have their registration suspended form 1 August 2008 until testing has been carried out. AQHA is in the process of putting HERDA results up on the online stud book for all Classified or IBF approved breeding sires. Results for colts, fillies, geldings or breeding mares will not be advertised at this point, however members can chose to have the result printed on their horses Certificate of Registration if they wish.

5.Effective 1 January 2008 all new colts and stallions going through the IBF process will be required to have HERDA testing done before they will be accepted as a breeding sire with the Association.

6.Effective 1 January 2008 all new Foundation Stock recording applications will require HERDA testing on top of the already required DNA, OLWS and HYPP testing. A negative test must be achieved before they will be accepted into the Foundation recording system.

Members with breeding mares that have linage to the stallion POCO BUENO (USA 3044) are encouraged to test for HERDA so they can make educated decisions when choosing a stallion to send their mares to for the 08/09 season. AQHA would like to stress that this disease is active within **all** Quarter Horse disciplines and warn that HERDA testing of all breeding stock with lineage to POCO BUENO will become mandatory within the next 3 years to help put a stop to the progression of this debilitating disease in Australia.

If you have any queries in regard to the new regulations or would like to discuss HERDA or our method of testing, please contact the AQHA Registry Department on (02) 6762 6444.

Please note that AQHA has extended the lodgement date for 07/08 Stallion Returns and Applications for Registrations for foals born during the 07/08 season due to the Equine Influenza outbreak that has affected the entire equine community.

The lodgement date for Stallion Returns in the 07/08 breeding season has been extended to:
Mares served between 1 August 2007 and 28 February 2008 – lodge paperwork by **30 June 2008.**
Mares served between 1 March 2008 and 31 July 2008 – lodge paperwork by 31

October 2008, as normal.

The lodgement date for Applications for Registration for foals born during the 07/08 season has been extended to:
Foals born between 1 August 2007 and 28 February 2008 – lodge paperwork by **30 June 2008**.
Foals born between 1 March 2008 and 31 July 2008 – lodge paperwork by 31 October 2008, as normal.

Australian Quarter Horse Association
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Tamworth, NSW 2340
Phone: (02) 6762 6444 Fax: (02) 6762 6422
ABN: 41 000 964 643

APPENDIX D

LIST OF JOURNAL ARTICLES

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Birchacek, Andra. "HERDA: New Breakthroughs Offer Hope." Performance Horse.

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