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A genetic analysis of epistaxis as associated with EIPH in the Southern African Thoroughbred

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Abstract

Pedigree and race run data from Thoroughbreds racing in Southern Africa, covering the period 1986-2002 (63 146 horses in pedigree data-set and 778 532 race runs), were analysed in order to study genetic and environmental factors affecting the incidence of epistaxis as associated with "exercise-induced pulmonary haemorrhage" (EIPH). Variables that were tested as significant in preliminary data analyses were included as fixed effects for the model. Various combinations of such variables namely age, weight, altitude, sex, month and going were tested. Fixed effects that were included in the final model were gender, going and altitude. The heritability estimates from a logit transformed analysis for epistaxis fitting both the animal and sire models were 0.23 and 0.40, respectively, which indicated that epistaxis as associated with EIPH in Southern African Thoroughbred sires has a strong genetic basis. Genetic trends indicating an increase in epistaxis were also found. Affected stallions and those racing whilst being treated with furosemide should be barred from breeding and not be considered as future sires. Estimated breeding values for epistaxis should be used as a tool for selecting against it and be considered in breeding programmes to decrease the incidence thereof.

Keywords: Estimated breeding value, genetic factors, heritability, racehorse, Thoroughbred
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Introduction

Epistaxis (post-race bleeding from the nostrils) is a common disorder world-wide amongst racehorses and has been reported for more than 300 years. Originally the blood was thought to be of nasal origin. However, Pascoe *et al.* (1981), examining horses with the fibre-optic endoscope, showed that the lung was indeed the site of haemorrhage. He coined the term "exercise-induced pulmonary haemorrhage" (EIPH), linking the readily identifiable features, exercise and lung haemorrhage. This disorder is of great concern because of the financial implications resulting from a decrease in performance potential, suspension of "bleeders" from racing, lost training days and the necessity for pre-race medication of affected horses. This could run into millions in most racing countries world-wide.

Pfaff (1950) published the first report on the incidence of epistaxis in a group of racing Thoroughbreds in South Africa. He found that at least 1.2% of horses bled from the nose after racing. In a later similar South African study undertaken by Weideman *et al.* (2003), an incidence of 2.1% was recorded for the period 1986-2002 and a sharp increase, especially during the last five years, was noted. These figures are in line with most research done world-wide, though an incidence as high as 13.5% was recorded by Kim *et al.* (1998) for Thoroughbreds racing in Korea. Although the incidence is low, it has serious financial implications. Of more concern is the already mentioned increasing trend in the incidence of epistaxis.

A number of hypotheses has been proposed to explain the pathophysiology of epistaxis, notably those of West *et al*. (1994) and Schroter *et al*. (1998). An appreciable number of investigations into non-genetic factors as cause of epistaxis has been undertaken over the last three decades. However, according to Takahashi *et al*. (2001) the primary factors responsible for EIPH have not been identified. A variety of findings has been reported. These include a positive relationship between epistaxis as associated with EIPH and age (Takahashi *et al*., 2001), epistaxis as associated with EIPH and month of the year (Johnson *et al*., 1973), epistaxis as associated with EIPH and sex (Pfaff, 1976) and epistaxis as associated with EIPH and altitude (Weideman *et al*., 2003). On the other hand, similar studies could not establish any relationship between epistaxis as associated with EIPH and sex (Lapointe *et al*., 1994) and epistaxis as associated with

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EIPH and the distance raced (Speirs *et al*., 1982). Furthermore, no association was found between epistaxis as associated with EIPH and trainer or going (Mason *et al*., 1983).

Very few references relating to the inheritance of epistaxis in the horse could be found in the available literature. According to Pfaff (1950) all bleeders could be traced back to the stallion Herod (foaled in 1758), and the names of the stallion, Hermit (foaled in 1864) and that of his grandson, Gallinule (foaled in 1884), appear in the pedigree of many bleeders in South Africa. The author discussed the possibility of a link between epistaxis and heredity (Pfaff, 1950). In a later publication Pfaff (1976) again suggested that heredity might be an important factor in the expression of epistaxis. This corroborated a statement made by Cook (1974) that breeding might be a factor influencing the occurrence of epistaxis in racing Thoroughbreds. However, no recent research into the mode of inheritance of epistaxis has been conducted. Robertson (1913), an expert on racehorse pedigrees, suggested that in the English Thoroughbred, bleeding from the nose is inherited as a simple recessive character, and that horses are not likely to break blood vessels unless they carry a homozygous pair of that recessive gene. However, no further evidence was provided. In a preliminary study (Weideman, unpublished), breeding data of dams and sires that suffered from epistaxis whilst racing were subjected to a series of test crosses in order to establish whether they were possible carriers of a recessive gene for "bleeding". In the case of mares that "bled", and could thus be regarded as homozygous recessive carriers, only 8.1% rather than an expected 50% "bleeders" were produced when bred to heterozygous stallions and an expected 0% "bleeders" when bred to homozygous dominant stallions. Unfortunately, the "bleeding" status of most stallions is unknown because the majority of sires used in the country has been imported. The data of only four sires that raced locally and bled, could be used. Bleeder stallions (assumed to be homozygous recessive) bred to heterozygous or homozygous dominant mares produced 9.7% bleeders against the expected 50% or 0%, respectively. Only one example of an assumed homozygous recessive x homozygous recessive mating could be found in the data and this resulted in a bleeder, as would be expected (100%). Most of the test crosses thus suggested that Robertson (1913) was incorrect in his assumption that epistaxis is inherited as a simple recessive gene. This finding prompted the present study in which it was postulated that epistaxis is inherited in a polygenic manner, expressing itself as a threshold trait (Weideman *et al.*, 2004).

The aim of the present study was to obtain more information on the genetic aspects of epistaxis in Thoroughbreds race horses in Southern Africa.

Materials and Methods

A horse demonstrating epistaxis is reported a "bleeder" by veterinarians employed by the National Horseracing Authority and is recorded in the dataset as such. For the purpose of this study the dataset did not include any data for starts following the first episode of epistaxis. Bleeders are subsequently suspended from racing for periods varying from three months (initial bout of epistaxis) to six months for repeated bleeders and permanent suspension upon the recurrence of any further bleeding. Due to time and cost constraints, on-course endoscopy is not employed as a routine on any of the Southern African tracks and horses that might bleed internally are thus not identified. Data of the National Horseracing Authority's reported cases have been recorded for all the racing centres in South Africa and Zimbabwe. The passport number (identification), name of horse, age, sex, breeding, stud where born and raised, trainer, distance raced, date and race when pulmonary haemorrhage occurred, centre of racing, date of last run before EIPH occurrence, state of going, jockey, weight carried, altitude and the date of return to racing of suspended horses have been recorded and are available from the National Horseracing Authority of Southern Africa's database. Racing in Southern Africa is conducted at sea level (Durban, Cape Town and Port Elizabeth), approximately 1 000 metres above sea level (Pietermaritzburg) and 2 000 meters a.s.l. (Johannesburg, Bloemfontein, Kimberley and Zimbabwe). The data covering the period 1986 – 2002 and involving 51 465 individual runners that raced a total of 778 532 runs were analysed. Pedigree information was made available by the National Horseracing Authority of Southern Africa. This included the initial runners from the data as well as the sire, dam and paternal- and maternal grandparents, and is shown in Table 1. The pedigree-file used in this data set was extended by 11 681 animals in order to create more genetic ties. The total number of animals was thus 63 146. The pedigree depth was, on average, three to four, but not more than six generations.

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The bleeder status of most sires (being imported) and a large proportion of imported fillies and mares that raced overseas was unknown. The data also included a large percentage of unraced dams of runners and grandams of runners, including those in the extended part of the pedigree file.

In order to estimate heritabilities (h^2), including a full pedigree relationship matrix, variance components were obtained by Restricted Maximum Likelihood (REML) procedures after a logit transformation of the data with a linear mixed model using ASREML was fitted (Gilmour *et al*., 1999). The transformation used in logistic regression is a transformation of the predicted scores of Y (Y') and instead of using Y', the log of probabilities is used as shown in the model:

$$\ln\left(\frac{P}{1-P}\right) = a + bX$$

Table 1 Description of the data set for epistaxis as associated with "exercise-induced pulmonary haemorrhage" (EIPH)

Total number of race runs	778 532
Total number of animals	63 146
Animals in extended pedigree file	11 681
Number of sires	1 471
Number of sires producing bleeders	354
Number of dams	16 277
Number of grand sires	2 368
Number of grand dams	10 796
Average incidence for the period	2.1%

The ASREML programme estimates variance components for mixed models by restricted maximum likelihood, employing an average information algorithm (Bunter, 2002). As fixed effects for the model, variables that tested significant ($P < 0.0001$) in a preliminary data analysis were fitted. In this preliminary analysis (PROC GENMOD of SAS, 1990) various combinations of variables, age, weight carried, altitude, sex, month and going were tested. Fixed effects chosen and included in the model were gender (females, gelded males, entire males), going or prevailing underfoot conditions (heavy, soft, good, firm) and altitude (at altitude of ± 2000 metres, mid- altitude or ± 1000 metres, sea-level or ± 0 metres). Although all the fixed effects except month, tested significant, only the above-mentioned three were selected because the inclusion of the remaining fixed effects caused convergence problems. The P-values for these fixed effects are presented in Table 2, indicating that age, altitude and going are the most important fixed effects influencing the occurrence of epistaxis as associated with EIPH.

Table 2 The P-values of fixed effects

Fixed effect	GENMOD P-value
Age	0.0001
Weight carried	0.0073
Altitude	0.0001
Sex	0.0076
Going	0.0001
Month	0.5464

In order to investigate the sensitivity of parameter estimates for epistaxis, a series of alternative random effect models was employed. These included animal, maternal and sire models. Analyses were done using linear, logit and probit procedures. Data were subsequently analysed allowing for a binomial distribution of the observed variables (generalised linear mixed models, GLMM). For the majority of fixed effects, convergence was achieved without needing to fix or constrain variance components in any way.

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Results and Discussion

Heritabilities and variances for epistaxis are presented in Table 3. Heritability estimates varied from 0 to 0.40, depending on the model fitted. The estimated breeding values (on the transformed scale) from the sire and animal models of Southern African sires were predicted and are shown in Table 4 (the top five sires displaying the highest estimated breeding value for epistaxis) and Table 5 (the top five sires showing the lowest estimated breeding value), respectively.

Table 3 Heritability estimates and standard errors (s.e.) for epistaxis using logit transformation for different models

Model	Heritability	s.e.
Animal	0.230	0.0225
Maternal	0.001	0.0026
Sire	0.400	0.0555

Table 4 The highest estimated breeding values (sire model and animal model) for epistaxis of Southern African based sires that produced runners during the period 1986-2002 according to their country of origin, year of birth, number of runners (progeny that raced) and number of bleeder progeny produced by individual sires

Sire	Year of birth	Estimated sire breeding value	Estimated animal breeding value	*Country of origin	Number of progeny that raced	Number of bleeder progeny
Print	1982	-1.38	-2.35	GB	101	11
Lost Chord	1973	-1.26	-2.39	GB	275	17
Al Mufti	1985	-1.24	-2.08	USA	284	21
Folmar	1973	-1.16	-1.98	USA	380	21
Northern Guest	1977	-1.07	-2.15	USA	817	46

*GB = Great Britain, USA = United States of America

Table 5 The lowest estimated breeding values (sire model and animal model) for epistaxis of Southern African based sires that produced runners during the period 1986-2002 according to their country of origin, year of birth, number of runners and number of bleeders produced

Sire	Year of birth	Estimated sire breeding value	Estimated animal breeding value	*Country of origin	Number of progeny that raced	Number of bleeder progeny
Peaceable Kingdom	1966	+ 0.72	+ 0.80	USA	370	0
Gallic League	1985	+ 0.66	+ 1.03	IRE	181	0
Song of Songs	1974	+ 0.65	+ 1.00	GB	277	0
Hobnob	1972	+ 0.63	+ 0.95	FR	384	1
All Fired Up	1981	+ 0.60	+ 1.16	USA	461	3

*GB = Great Britain, IRE = Ireland, USA = United States of America, FR = France

The estimated breeding value for epistaxis for all Southern African sires active during the 1986-2002 period varied from a high of –1.38 to a low of +0.72 while the animal breeding value ranged from a high of -2.39 to a low of 0.46. A frequency distribution of the breeding values fitting an animal model and breeding values fitting a sire model are presented in Figures 1 and 2, respectively, indicating a skewed distribution and a high peak for both sires and animals with approximately zero breeding values. The highest negative values indicated those sires breeding the most bleeders and high positive values those not inclined to breed bleeders.

Unfortunately, for the purpose of this study, probit analyses did not converge, resulting in them not being used and only the logit transformation was applicable for this study. No heritability for maternal

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effects was evident, as shown in Table 3. However, both the animal and sire models yielded heritabilities of 0.23 and 0.40, respectively. Gilmour *et al*. (1985) demonstrated a downward bias of heritability estimates under a sire model if the number of progeny per sire was low or the incidence was extreme, as was the case in this study (2.1%). Conversely, Engel *et al*. (1995) showed an upwards bias in heritability estimates for simulated data sets, attributing the positive direction of bias to the increased number of fixed effects included in the simulation data generated and subsequently the analytical model applied. According to Bunter (2002), the above mentioned factors make the prediction of probable direction of bias in heritability estimates for generalised linear mixed model methodology applied to real data sets a good deal more complicated. It is thus not possible to speculate about the possibility of bias in these estimates. In several other studies on threshold traits the sire model yielded higher estimates than the animal model (Snyman *et al.*, 1998; Rust & Groeneveld, 2002). The regression of breeding values of sires fitting a sire model on the corresponding breeding values fitting an animal model are illustrated in Figure 3 with R^2 = 0.93, indicating a high correlation between corresponding Best Linear Unbiased Prediction (BLUP) estimates and almost no change in rank.

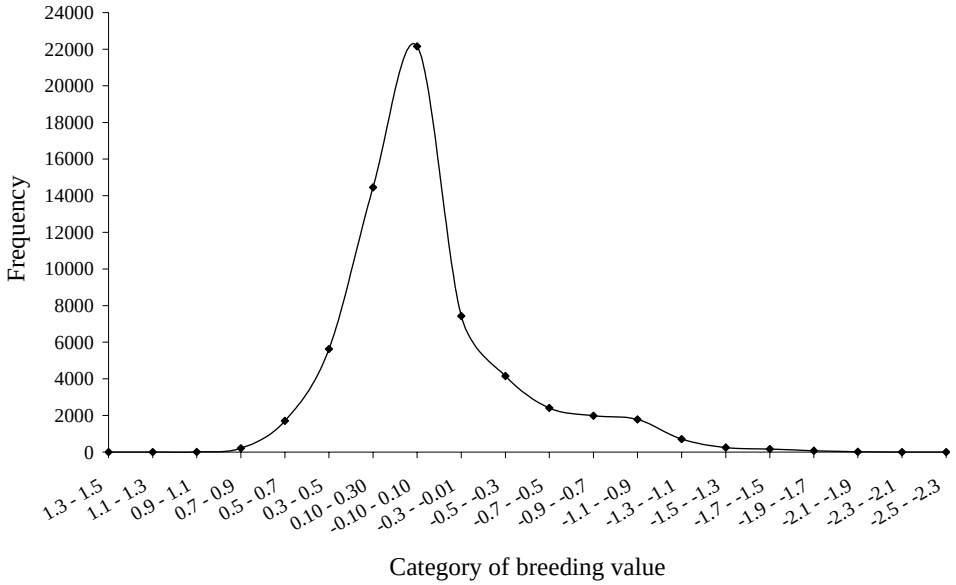

Figure 1 Frequency distribution of Best Linear Unbiased Prediction (BLUP) breeding values fitting an animal model (n = 63 142)

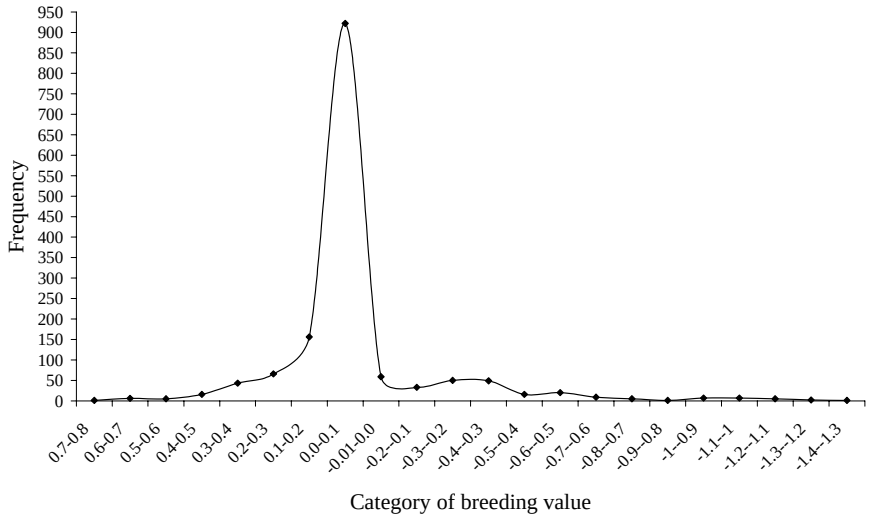

Figure 2 Frequency distribution of Best Linear Unbiased Prediction (BLUP) breeding values fitting a sire model (n = 1 471)

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No references relating to the heritability of epistaxis in the horse could be found in the available literature except for an earlier study by the authors of this study (Weideman *et al.*, 2004). They found the average incidence for epistaxis in Southern African horses to be 2.1% and the heritability of predisposition to epistaxis according to the procedure of Falconer (1989) to be 0.54 for first-degree relatives (coefficient of relationship, r = 0.5), 0.41 for second-degree relatives (r = 0.25) and 0.30 for third-degree relatives (r = 0.125). This study estimated the heritability of EIPH related to epistaxis of sires in Southern Africa at 0.40 and considered this to be high. Selection against this trait should be effective and relatively fast progress could be made at reducing the incidence of this disorder.

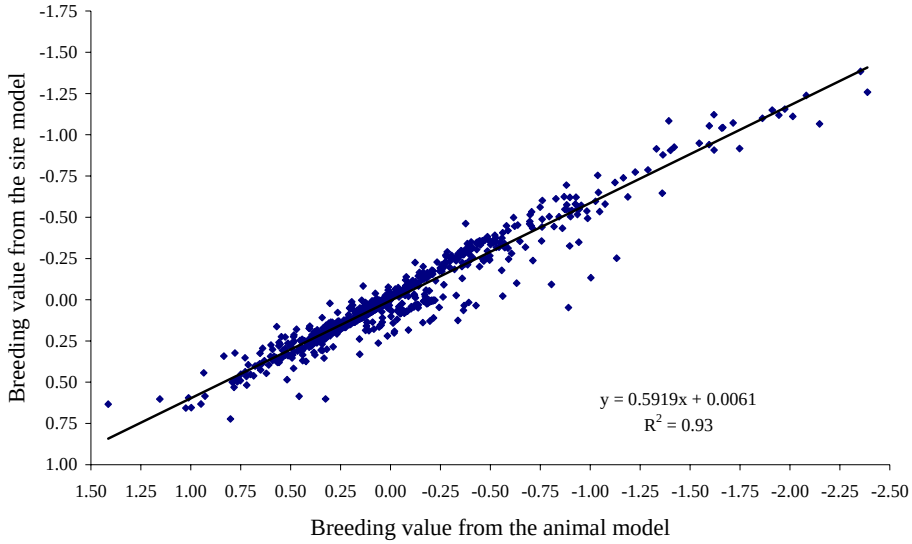

Figure 3 Regression of Best Linear Unbiased Prediction (BLUP) breeding values of the sires (n = 1471) fitting a sire model on the corresponding breeding values fitting an animal model

Most sires used at stud in Southern Africa and a small proportion of fillies and mares are imported. Only a few of the countries from which they were imported have measures in place to record and suspend bleeders, and they are consequently of unknown bleeder status. A number of America states allow horses to race after treatment with furosemide, a presumed suppressor of epistaxis as associated with EIPH. The efficacy of this drug was studied by a number of researchers, notably Sweeney *et al.* (1990), Manohar *et al.* (1994), Barnes (2000), Geor *et al.* (2001) and Kindig *et al.* (2001). Varying degrees of success were noted. The use of furosemide is prohibited in horses participating in races that fall under the jurisdiction of the National Horseracing Authority of Southern Africa.

The increasing prevalence of epistaxis in Southern African Thoroughbreds, as shown by Weideman *et al.* (2003), might be an indication that breeders have, through selective breeding, developed horses whose lungs can no longer sustain the stress of strenuous exercise.

Figures 4 and 5 show the genetic trend for epistaxis breeding values for the animal model and sire model, respectively. The breeding values from the animal model in Figure 4 illustrate that epistaxis has increased from about 0.04 in 1982 to –0.12 in 1998. A similar tendency in the incidence of epistaxis was found in the sire model (Figure 5) where an approximate increase from 0.02 (1970) to -0.08 (1987) was shown. This would indicate that over an 18-year period a consistent increasing genetic trend was experienced. From Tables 4 and 5 it can be seen that both sires having high negative and positive breeding values for epistaxis have large numbers of progeny, and consequently a high level of reliability. From these tables it is obvious that to get a clear picture regarding the breeding values for epistaxis of sires, only those sires that have completed a number of years at stud and produced a large number of offspring were selected. All potential sires have to prove their ability on the racetrack before being selected for stud duties. This amounts to a racing career up to the age of 5-years and being retired to stud at the age of six. Another 4 to 5 years would be required for some progeny (1 to 2 crops) to be born and tested on the racetrack. This implies that relatively accurate breeding values of sires could be estimated only at an age of 10+ years. The

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reliability of breeding values of sires with a few progeny would not be as reliable as values for sires with many progeny.

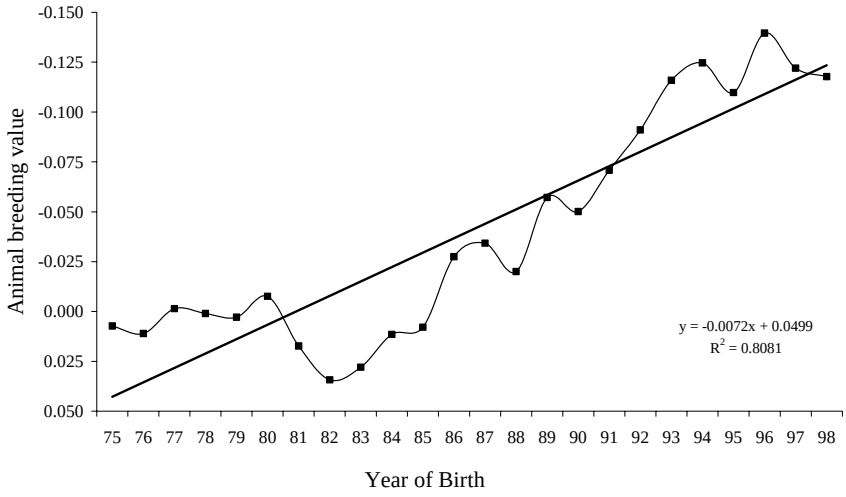
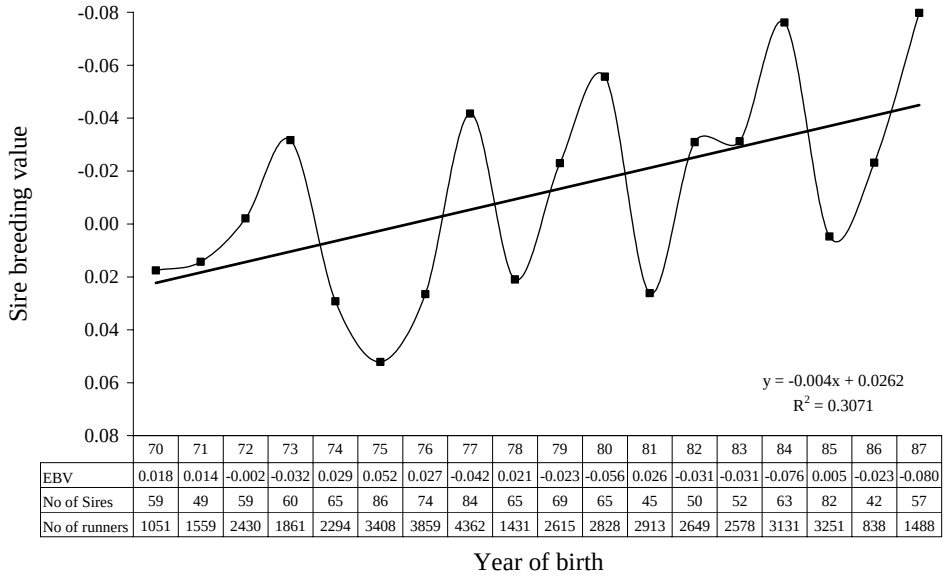


Figure 4 Genetic trend for epistaxis breeding values based on the animal model



	70	71	72	73	74	75	76	77	78	79	80	81	82	83	84	85	86	87
EBV	0.018	0.014	-0.002	-0.032	0.029	0.052	0.027	-0.042	0.021	-0.023	-0.056	0.026	-0.031	-0.031	-0.076	0.005	-0.023	-0.080
No of Sires	59	49	59	60	65	86	74	84	65	69	65	45	50	52	63	82	42	57
No of runners	1051	1559	2430	1861	2294	3408	3859	4362	1431	2615	2828	2913	2649	2578	3131	3251	838	1488

Figure 5 Genetic trend for epistaxis breeding values based on a sire model, with number of sires per year and number of runners sired

Among the sires displaying the highest breeding value for epistaxis, Sportsworld (1988) was the youngest, while Qui Danzig (1987) was the youngest sire displaying the lowest breeding value. Therefore, reliable breeding values obtained by fitting a sire model can only being estimated at a relatively advanced sire age. This is unfortunate, as sires of this age are mostly regarded as past their best and breeders would rather invest in younger, more fashionable and promising sires. Selection bias could also be a problem easily overlooked since this occurs when a sire is represented only by selected daughters at stud (only those females that were good performers on the racetrack or the very well bred ones, - the poor performing females being culled). Very few South African bred males (less than 2%) enter the stallion ranks, and the effect of selection bias on the sires can thus be largely ignored. If a large proportion of a sire's poorly performing daughters

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and sons is culled before being adequately exposed and tested on the racetrack, his evaluation would be appreciably higher than his true genetic value. In contrast to the sire model, the animal model uses information from the maternal side of the pedigree and the genetic merit of all relatives plus the animal's own performance to estimate the animal's genetic merit. Sires can thus be evaluated at an appreciably earlier age when using the animal model. Breeding values obtained by fitting an animal model are thus of great value, provided that sufficient links exist between studs.

An alternative could be to "challenge" sires, as described by De Greef *et al.* (2001). To establish the epistaxis predisposition of sires of unknown status, young outstanding sires could be "challenged" for bleeding by testing their offspring in an epistaxis-challenging environment. Since a positive relationship was established by Weideman *et al*. (2003) between racing at sea level and epistaxis, it might be advantageous to test Southern African sires by comparing all offspring that raced at sea level. According to Falconer (1989) it is possible to control the incidence by external means and to optimize it. If the character is reacting to some environmental factor(s), the effect of the factor(s) can be intensified or reduced so that the incidence is altered. The incidence of the character selected for (epistaxis) can thus be increased through racing horses at sea level (an unfavourable environmental factor). This changed incidence is best regarded as a shift in the threshold relative to the mean liability of the population and by shifting the threshold selection could be more effectively applied.

Conclusions

This study has shown that epistaxis as related to EIPH in the Southern African Thoroughbred sires has a strong genetic basis. The results obtained suggest that genetic determination of epistaxis is strong enough to be considered in breeding strategies. This should include the establishment of an international database for epistaxis and that estimated breeding values be used in the selection of parents. Further steps should include the disqualification of "bleeders" from being registered in the Stud Book and that furosemide be banned internationally from racing, or failing that, breeders should refrain from buying potential sires that have raced on this drug. In an effort to get a clearer picture of stallions transmitting epistaxis to offspring, sires could be challenged through testing their offspring in a challenging environment (racing at sea level *vs.* at altitude). This may shed more light on the predisposition of imported sires with unknown bleeder status and those who raced after treatment with furosemide, to produce a sizeable number of bleeders.

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