

# Increasing Hereditary Health Problems in the Breeding of Purebred Dogs: A Comparative Overview Using Dobermans in Germany, Europe and in the USA as Examples

by Dr. Reinhard Haberzettl

**Introduction** – Linda C Kurz, UDC Recording Secretary and Health Committee Chair.

An early version of the following article originally appeared in August 2002 in “Das Schäferhund Magazin,” a publication for German Shepherd Dog fanciers. Since that time, it has been updated with current information. We are especially grateful to be able to include archival photographs from Dr. Haberzettl's personal collection. UDC members will find a great deal to think about in this article. The author brings us a unique perspective and is not influenced by those currently holding leadership in the European Doberman community.

**Brief C.V. of the Author:** Dr. Reinhard Haberzettl was born in 1952. He obtained his doctorate in biology (equivalent to Ph.D.) in USA) from the prestigious Humboldt University Berlin. He did additional work that focused on animal genetics. In that field, he achieved a Diplom-Biologe (equivalent to USA M.S.) from Martin-Luther University in Halle/Wittenberg.

After completing his formal education, Dr. Haberzettl worked for 5 years in the pharmaceutical industry as a genetic and toxicological scientist and published in the journal “Chemical and Pharmacological Factory Fahlberg-List Magdeburg” (today part of Hexal AG). From 1980-90 he worked at the Institute of Animal Breeding of Humboldt Berlin, doing agricultural-animal breeding research. Since 1991, returning to the pharmaceutical industry, he has worked for Celltech Pharma AG Essen.

In 1990, he served as Breed Warden for the East German Dobermann Verein, the same year that the East and West German Dobermann Verein were reunited. He bred Dobermans from 1970 until about 1990. At the present time he is an active member of the SV and is an ardent breeder of German Shepherd Dogs



*A robust brown male dog from Russian breeding, a very powerful type without inbreeding and without West European ancestors. This photo from the Haberzettl archives was made in 1988 at an International Dog Show in Budapest, Hungary.*

He and his wife, Angelika, operate a retail pharmacy. They have two children Robert, 23 and Uta, 22. Robert is studying pharmacology and Uta is studying theology.

## Short history of the Health of the Doberman

The Doberman was created from mixtures of various breeds and half-breeds at the end of the 19th century (Göller 1912, Dom 1957). This genetic variety (Heterozygosity) was a great health advantage. Up to approximately 1950, there were practically no hereditary health problems, worldwide. The Doberman was vigorous and long-lived. These qualities still remained in the populations of East Germany and Eastern Europe up to the nineties.



*The brown Doberman bitch Freya vom Kohnstein SchHI (born 1969) biting while stopping the helper from fleeing in 1975 in two escape phases. The quickness and high temperament of good Dobermans place severe demands on the helpers' abilities. Photo from Haberzettl archives made at a training day in 1975 in Magdeburg, East Germany.*

Between 1970 and 1990 the Doberman became a vogue breed in the USA; this resulted in mass over-breeding. The causes of the Doberman's boom were on the one hand the growing security need of citizens and on the other hand the successful advertisement of the "protector dog" image of the breed in movies. This role was subsequently taken over by the Rottweiler and the German Shepherd Dog in the nineties in the USA. This over-breeding and inbreeding of Dobermans, taken together with insufficient attention to their hereditary health, has resulted in a North American Doberman population that is highly burdened with 5 hereditary defects (Table 1). Therefore we are forced to acknowledge a strong degeneration in the breed that might be hard to repair by means of conventional animal breeding. The population of German and European dogs is similarly highly burdened with 2 hereditary defects as a consequence of the same bad breeding practices as in the USA. The other 3 hereditary defects are scarcer in Europe, but an increase is now apparent!



*Freya's daughter, Anka von der Domstadt, SchH1 (born 1972) during the attack*

**Table 1: Estimated spread of 7 degenerative hereditary diseases as well as their heritability (estimated value of genetic variance in the total variance) in 3 different Doberman populations. The estimates for East European populations are somewhat more uncertain because an increasing dilution of the indigenous gene pool has taken place as a consequence of imports from Western Europe and the USA and additionally because there are fewer reliable sources**

Disease (Symptoms)	Heritability	Mode of Inheritance	Spread of hereditary defects in:		
			USA	Western Europe (2004)	Eastern Europe (1990)
1. Dilated cardiomyopathy, DCM (Sudden death and congestive heart failure)	high	polygenetic but mostly autosomally (incompletely) dominant (partly X-chromosomal)	very high	very high, but in the individual DCM genotypes, big differences	little
2. Gastric Volvulus (bloat, shock and death)	medium to high	polygenetic	high	high and increasing!	little
3. Hypothyroidism (skin problems but various other severe symptoms possible)	very high	several genes? monogenetic?	high	little, but increasing!	little
4. Von-Willebrand disease = VWD (potentially severe bleeding)	very high	autosomally (incompletely) recessive monogenetic	high	little, but increasing!	little
5. Wobbler syndrome (paralyses)	very high	monogenetic? polygenetic?	high	1989 little, but after then, fast increasing; 2004 medium	little
6. PHTVL/PHPV (ranging from impaired vision to blindness)	high	autosomally (incompletely) dominant	little	little to medium (below 15%)	little
7. Hip joint dysplasia –HD, (crippling arthritis and hip pain)	medium	polygenetic	little	little (phenotype below 5%)	little

For friends of dogs and genetic scientists, it is a completely incomprehensible fact that even today neither inbreeding restrictions, nor exclusions from breeding dogs burdened in genotype and in phenotype has been initiated --neither by breeders nor by the breeding authorities in almost all European countries (including Germany) nor on other continents.

While in North America the great hereditary health problems have been analyzed and some corrective steps taken offensively and honestly, most breeding authorities in Europe have made the unhelpful claim to their breeders and to the public that there are either no or few problems. This is an irresponsible attitude. Uninformed purchasers of puppies are very disappointed when their young dogs fall ill or die of cardiomyopathy or volvulus. These hereditary diseases are appearing more often in Europe, because of the high inbreeding and unsatisfactory health tests done on the popular breeding animals. This causes high monetary costs at the veterinary clinic and for medications. The life quality of the dogs (and owners!!) deteriorates as the life expectancy of dogs shortens by years!

### **Breeding of Pure Bred Dogs and Good Health - Utopia or a Reachable Goal?**

Let me elaborate on some short critical constructive thoughts of mine about how to achieve a higher health rate in the future breeding of purebred dogs because this concerns almost all breeds. Well-known veterinarians, genetic scientists, breeders and experts in animal protection increasingly criticize the quality of the breeding controls imposed by purebred dogs' organizations (AKC, VDH, FCI, etc.) where genetic health is being neglected. We insistently demand that genetic health as a breeding goal must be regarded as just as important as a breed's beauty and character. This demand should be respected for all breeds in the future! The criticism of insufficient care to genetic health in 4 areas is summarized below and the steps to remedy them are outlined:

1. The breeding for a beautiful conformation must not burden the health of a breed as a result of an extreme interpretation of the breed standard, but the breed standard as applied by judges at exhibitions must benefit the health.

2. Hereditary defects must not be tolerated in conformation breeding, but they must be quickly eliminated from the gene pool. Veterinary medical and animal breeding genetic institutes of the universities can offer competent assistance. Breeding authorities, breeding judges, breeding attendants and breeders are not only responsible for the beauty and character of their purebred dogs, but every one of them must take responsibility for countermeasures against genetic health defects in spite of temporary financial and/or prestige losses.

3. In the past, many breeding authorities (not all of them) have not taken sufficient advantage of the offers of universities for assistance to fight hereditary defect or in fact ignored them completely (Eichelberg 1998). This cooperation is logistically expensive and it makes high demands on the responsible persons. It also requires honesty, a sense of responsibility and a readiness to constantly educate oneself!

4. The role of uncontrolled inbreeding should especially be restricted in all breeds, because it has led to an accumulation of

defective genes. In those bloodlines burdened by high numbers of hereditary defects, we must completely waive inbreeding and exclude entirely all breeding of animals with any external and hereditary degeneration.

In this article I am going to confine my discussion to points No. 2 and 3 with the Doberman as example. The insufficient attention to all 4 points of criticism in many other breeds has caused an unnecessary decrease in the public image of the whole of purebred dog breeding in the last few years. Breeders and breeding authorities need more self-criticism and more sense of responsibility towards the breeds as well as towards the buyers of the whelps. If responsible persons of breeds burdened by hereditary health defects do not quickly succeed in limiting the damage, then the situation in Germany and Europe will develop along a path similar to that observed in the USA during the last decade (Günter 1996). The numbers of purebred dogs being registered has dropped rapidly in the USA (this is particularly true for Dobermans) because more and more dog fans prefer to pick up a dog from rescue organizations or to purchase a healthier half-breed. The same situation is now occurring in Germany in 2004.

The different individual breeds and different populations of the same breed can be heavily burdened with hereditary defects nationally and internationally in very different ways. Hip joint dysplasia (HD) is a good example. There are some very heavy burdened breeds: St. Bernard, Leonberger, German Shepherd Dog and others. However, success in the fight against it has been very different according to the commitment of breeders. The Hovawart represents a very especial positive example where much progress has been made. Concerning the German Shepherd Dog, success of the fight against HD is very different from country to country (Willis 1994). In the countries where there are good descendant records, the dogs carrying HD can be identified in spite of an external impeccable hip (phenotype type good, gene type bad) and they could be excluded from breeding with the result that the frequency of diseased hips would strongly decrease. However, at the SV they have been unsuccessful as this recommendation has not been followed. For examples in individual breeds; the Airedale-Terrier and Doberman have a very low HD burden, and almost 100 percent of greyhound breeds and all wild dogs, (e.g. the wolf) are generally HD-free. In the worldwide view, the Doberman has a relatively healthy hip. Since the beginning of X-ray examinations 30 years ago in Germany, the number of Dobermans with normal hips has almost constantly been at over 90 percent; thus breeding practices have caused neither deterioration nor improvement. Table I summarizes this HD problem and the other 6 hereditary Doberman health problems. The following text briefly discusses each of these 6 and makes comparison to other breeds.\_

#### **1<sup>st</sup> feature Sudden death by heart failure (cardiomyopathy)**

Many dog breeds are affected with varying frequency, but the DCM death-rate for the Doberman takes the sad first place both in the USA and in Germany. Mr. Kraft (1989), from the University of Munich, presents heart death statistics from dissection materials of different pathological institutes, where frequently the Doberman rate stands ahead of the Great Dane, the St. Bernard and the German Shepherd Dog. Furthermore, Prof. Kraft presents the result of a regional survey of Doberman



Club (VDH) from South Germany. From 92 cases of death, 24 died of death by heart failure. The extensive statistics of death by heart failure in purebred dogs by Calvert and Pickus (1989) from the USA looks like the situation in Germany. The Doberman by far leads the Great Dane, the Irish Wolf hound and the St. Bernard.

Affected Dobermans can die at 2 months, 12 months, 3 years, 7 years or 11 years –the age of DCM death in any individual affected Doberman depends upon how many of the DCM genes he has received from his sire and dam (van der Zwan 1987). Quantitatively, DCM has a polygenetic mode of inheritance (Table 1). The greater the number of DCM genes inherited, the earlier the dog will die, less than 5 years old. If he has inherited only a few of the DCM genes, then he will not die of it till he is an old dog (over 9 years old) or perhaps not showing DCM at all, dying of something else first.

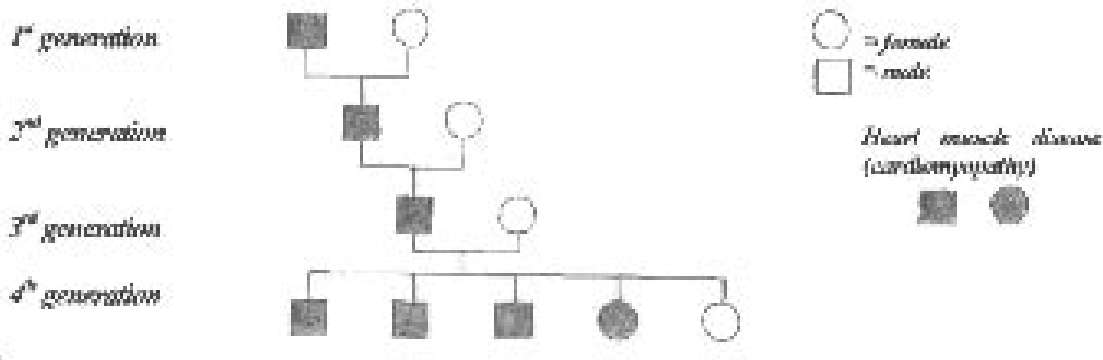
Both in North America and in Europe an increasing reduction in the age of death seems apparent (Kollenberg 1998). In general two forms of disease progression (Type A and B) can be identified in the Doberman depending on how suddenly the dogs die and by other features (van der Zwan 1987, Schüler 1997).

Type A: Arrhythmia. For this type, the death occurs suddenly, allegedly without (??) any warning. The dog collapses and dies. Diagnosis of the dog's arrhythmia is possible by using long-term ECG (Holter recording) as is done for human beings.

Type B: Congestive Heart Failure. This type shows a lingering development of symptoms for a prolonged period. The dogs often cough in the morning as a result of water accumulation in the lungs. Diagnostically, heart enlargements and vessel changes can be detected a long time before the death.

A partly X-chromosome and predominantly autosomally (at different chromosomes) polygene hereditary transmission can be assumed, because according to the statistics approx. 57% of all Dobermans in Germany and 67% in USA, that died by heart failure, are male dogs.

The noticeable frequency of death cases over several generations (Fig. 1) can be identified in the German and American Doberman's pedigree trees. The heritability of the sudden death by heart failure must be assessed as medium to high.



**Fig. 1** Pedigree for heredity of heart muscle disease (cardiomyopathy, type B) from a US breeding pattern according to Calvert and Pickus (1989) over four generations. Grandfather, son and grandson fell ill and each of them was mated with healthy bitches. The grandson begot a litter of 5 whelps, 4 (3,1) of them fell ill



**Jason v. Nymphenburg**, born 1982. His owner Martina Kelb in Berlin, breeder under kennel name "Novlis", made this photo. Many Nymphenburg Dobermans were the major winners of German and European shows in the 80s and 90s: Athene, Athos, Berenike, Bellona, Diana, Farina, Feus, Jaron, Jason, Orpheus and others. They had a great influence on the breed. Most died by DCM or volvulus because they had many genes for both diseases.



**Ilg v. Amtmannsgarten**, born 1985; an elegant but oversized black male dog, strongly inbred from West German breeding, with death by heart failure (Father: Jason von Nymphenburg.) Photo is from the Haberzettl archives



*Eos v.d. Dobermannstadt, born 1986, standing in a show pose at left.*

*The bottom two photos (left) are from his first SchHIII trial in 1988 during the attack by the helper and at the hold and bark. He died of DCM at 8.5 years. Up to his death in 1994, he made SchH III many times with his owner and trainer Vokhard Haun, (breeder, "v.d. Fixe Idee"). Eos' breeder was Walter Hacket, Apolda. Eos' brother Euros also died of DCM at the age of 9. Another brother, Enos, died of bloat at only 9 months of age.*

*Father: Jason v. Nymphenburg, West Germany, died of DCM at 7 years. Jason's three litter-brothers and their mother and one grandfather also died of DCM at ages ranging from 4 to 8 years old.*

*Mother: Asta v.d. Dobermannstadt, East Germany, phenotype and genotype without DCM. (pictured below)*



*The E-litter v.d. Dobermannstadt was an outcross breeding between Asta and Jason. It took place in 1985 in the garden of Haberzettl's in East Berlin. In the resulting litter, one brother and two sisters of Eos and Euros probably have had no DCM in phenotype. Eos was bred with 55 East German bitches (all free of cardio in phenotype and genotype) producing 340 progeny, with not one developing DCM phenotype 10 years after Eos' last pups were whelped! There must have been a dilution effect of the DCM genes from Jason v. Nymphenburg in his pups in the first and second filial generation after crossing Jason and his son Eos to DCM free bitches in East Germany.*



*Asta v.d. Dobermannstadt, Best bitch in Show Biesdorf 1984, Dam of Dobermannstadt E litter. Asta was free of DCM in phenotype and genotype*

## 2<sup>nd</sup> feature: Volvulus (Bloat and Gastric Torsion)

Volvulus appears in many large breeds with differing frequency. It is a life threatening disease and emergency veterinary treatment is required to enable the dog to survive. According to Willis (1994), the German Shepherd Dog, the Berner Sennen dog and the Doberman are most frequently affected in Europe. In the USA, the death statistics of 26 breeds (Schüler 1997), indicate that the German Shepherd Dog, the St. Bernhard and the Weimaraner are especially heavily affected. In breed comparison, the Doberman is only in the 8<sup>th</sup> place here, however a strong increase is evident in Europe. From 1964 till 1994 the clinic admissions of Dobermans for bloat have dramatically increased by 1500%. Of all 92 registered death cases of Dobermans in South Germany, 15 (16%) died of volvulus. In Germany, many Dobermans were even successfully operated on twice during their life and then they were used for breeding. An increase of volvulus within definite bloodlines is noted - the genetic basis is clear, but environmental factors play a role as well. The heredity pattern is not yet exactly known. Because of the fact that volvulus is subject to environmental factors (e.g. filling quantity in stomach, movement or rest after the meals, anatomical special features of the form of the rib cage and rib width and others), we can assume that the heredity is polygenetic. At the moment the heritability can be judged as medium to high. Until 1990, bloat and torsion in the East German Doberman population was under 2 %. After 1990, 20 % of German Dobermans were dying of bloat with torsion. Much more than 20% must have a genotype predisposing to volvulus.

## 3<sup>rd</sup> feature: Hypothyroidism

Besides the Doberman, the Husky, Malamute, Labrador and Beagle are also burdened with this hereditary defect (Willis 1994). According to Dodds (1988), over 60 % of Dobermans in the USA are hypothyroid. Similar to the disease in humans, if it is left untreated, hypothyroidism leads to various, unspecific disease symptoms in the dog, e.g. hair and skin illnesses, muscle and nervous diseases, loss or gain of weight, digestive troubles, infections and other symptoms. Quality of life and life expectancy are greatly reduced without hormone treatment. Hypothyroidism is comparatively easy to diagnose and easy to treat. Giving the hormone daily in pill form benefits greatly the diseased Dobermans. However, if breeding is being pursued with these diseased dogs, then there the breed is existentially endangered. Hypothyroidism can exacerbate the symptoms of other diseases such as: cardiomyopathy, vWD and Wobbler syndrome. The heritability of hypothyroidism is estimated to

be as high as 90 percent (Dodds 1988). Therefore the effect of environmental factors is small (with the exception of hormone substitution!). A mutation of one or only few genes can be assumed.

## 4<sup>th</sup> feature: von Willebrand disease (= vWD-bleeder)

The "von Willebrand disease" is a very special blood coagulation disorder with a significant health risk that can be transmitted autosomally incompletely (Haberzettl 2004) through a recessive simple monogenetic mode of inheritance (Brewer 1999). Completely (100%) recessive and completely dominant monogenetic inheritable diseases and features in both animals and humans are very rare; most monogenetic diseases have a mode of inheritance between recessive and dominant (Freye 1976, Strachan and Read 1996). But most genetic defects in humans and dogs alike are not monogenetic but polygenetic with a complex quantitative mode of heredity, similar to that of hip joint dysplasia. The vWD mode of inheritance is "incompletely" recessive because carrier animals (Aa) have plasma levels of factor 8 (von Willebrand's protein) intermediate between those of "clear" (AA) and "affected" (aa) animals. This is in contrast to a "completely" recessive mode of inheritance as illustrated by the brown (b) and black (B) color genes. BB and Bb dogs are "completely" black and only bb dogs are brown. Bb dogs that are carriers of the brown gene are not of an intermediate color. In contrast, Aa dogs that are carriers of the vWD gene have intermediate levels of the factor in their blood.

Molecular genetics has made the situation relatively clear. This complex situation is now understood to arise because the genetic defect giving rise to vWD (in the Doberman and Manchester Terrier but not in all other breeds affected by vWF deficiency according to VetGen) is not in the gene for the protein factor itself but in the DNA sequence controlling the construction of the gene message from its separate parts on the chromosome. This is called a splice defect. Since the fidelity of this splicing process is never 100%, a small amount of normal factor is produced even in the case of dogs carrying two defective genes (Kurz, 2004). The inheritance of the potential to bleed is simple. Normal levels of the factor are only present in animals with two normal genes. The potential for severe bleeding is most serious in animals with two defective genes but even here it is not automatic because some small amounts of normal factor are still made by "mistake". Whether an affected animal (or less likely a carrier) actually bleeds depends upon other genes or environmental factors. Although confidence in this explanation is high, it has failed to appear in the peer-reviewed scientific literature as a consequence of patent concerns and commercialization of the DNA test.

Breeding results (statistical) in all breeds from the 6 possible genetic combinations "A" represents the normal gene and "a" represents the mutated gene:

Both parents carrier (Aa x Aa): → puppies 25 % affected (aa), 50% carrier (Aa), 25% clear (AA)

Both parents clear (AA x AA): → puppies 100% clear (AA)

Both parents affected (aa x aa): → puppies 100% affected (aa)

One parent carrier and one clear (Aa x AA): → puppies 50% carrier (Aa), 50% clear (AA)

One parent carrier and one affected (Aa x aa): → puppies 50% carrier (Aa), 50% affected (aa)

One parent affected and one clear (aa x AA): → puppies 100% carrier (Aa)



The vWD gene frequency in the American Doberman population is very high: approximately 30% are homozygote affected, 50% are carriers (heterozygote vWD, leaving only 20% of dogs completely clear in phenotype and homozygote free of vWD genes in genotype (Kurz 2004). Probably the European Doberman is still only lightly burdened with vWD genes at the moment but exact figures do not exist. Further increase can be expected also in the European Doberman because the breeders have taken only limited actions towards limiting the damage. The extensive North American experience could be helpful in Europe. The vWD is found in 57 other breeds of purebred dogs: German shepherd dog, Corgi, Sheltie, Golden Retriever, Manchester terrier, poodles and many other breeds (Willis 1992, Schueler 1997), but the gene frequencies are lower than in the Doberman. For several other breeds, the homozygote-affected dogs are subject to potentially lethal episodes of external and internal bleeding that can occur any time and must be treated by a veterinarian. But fortunately homozygote Doberman bleeders can still produce a small amount (5-10%) of normal factor (Kurz 2004) providing some protection against uncontrolled bleeding and making the disease milder for affected Dobermans.

The symptoms (phenotypes) shown by affected and carrier Dobermans have such great variability because the actual vWF levels in both affected and carrier can vary widely in different dogs and also in the same dog at various times in his life. These great phenotypical variations must arise from interactions of the vWD gene with other (unknown) genes and to environmental factors in the individual Doberman (stress, high inbreeding, hypothyroidism, other diseases or factors). There is also the possibility that we have more than one deleterious mutation in the vW genes or different alleles at the same gene locus, so that the proteins in phenotypes of different affected and carrier Dobermans are different. Research is continuing (Cornell Veterinary School).

In genotype, both carriers and affected dogs differ only in the degree of penetrance of the defective gene (Dodds 1988). That is, a carrier although outwardly a healthy Doberman is not vigorous, in the sense that it does not have the same reserves as a normal dog to withstand environmental stress. If a vWD carrier becomes hypothyroid, it may be subject to bleeding episodes unless placed on thyroid supplement.

The exact nature of breeding practice (inbreeding or outcrossing) can have a great influence on vitality. Since 1970 breeding of affected animals has slowed in the US but it is increasing in the Netherlands, Italy, Norway, and Hungary. In Germany, there is almost complete inattention to this problem even by the most prestigious breeders; the vWD status of the European population should be checked most urgently.

The new American DNA test is an excellent, simple method that the European Doberman breeder could use to prevent an increase from the low gene frequency in the European Doberman population to the high gene frequency in North America. The old protein-based method was completely unreliable for carrier detection because of the great individual variation in phenotype (the plasma levels of normal vWD-protein.) The vWD DNA test is valid lifelong for every dog. The DNA test report is given as "clear or "carrier" or "affected". This one result for one dog is definitive and final. Retesting is pointless, because the result will always be the same for a given animal (Brewer 1999).

After identifying all carriers in the breeding population American breeders could quickly reduce the disease gene frequency. First the simple monogenetic mode of heredity and second the DNA test give the American breeder a very good chance to eliminate vWD disease in future generations. In fact, this is occurring (Kurz, 2004).

### **5<sup>th</sup> feature: Wobbler syndrome (paralysis)**

The Wobbler syndrome is a hereditary degenerative disease of the neck spinal column, occurring most commonly in middle age. Deformed intervertebral discs and spine bodies constrict the nerve tissues in the spinal cord, so that pain, movement disorders and paralysis occur. Surgery is the only successful treatment. Besides the Doberman, such cases are also present in the German Shepherd Dog, Basset and Barsoi. According to Schüler (1997), the American Doberman has a higher occurrence than the European. However, exact figures are known neither for North America nor for Europe. The exact heredity of the syndrome is not clear, primarily because there is far too little cooperation of the breeder clubs with the Universities. Van der Zwan (1987) supposes a polygene heredity symptom with probable high heritability.

### **6<sup>th</sup> Feature: Degenerative eye diseases (PHTVL/PHPV)**

Impaired vision up to full loss of the sight is the result of genetic conditioned degeneration of the eye and occurs in several breeds with different frequency. According to Willis (1994) the Staffordshire Bull Terrier, Basenji and the Doberman are affected most strongly by PHTVL/PHPV. This is a disorder of a fetal development of the eye that results in large and small spots and turbidity on the eye; the spots are caused by the persistent (that would normally disappear) embryonic membranes and blood vessels.

It is assumed to be an autosomal incompletely dominant heredity (Schüler 1997) and a high heritability should be expected. The abbreviations mean: PHTVL persistent hyperplastic Tunica vascularis lentis, PHPV persistent hyper-plastic primary Vitreum.

While this disease is presently very rare in the American Doberman, it can be expected to increase with the arrival of European imports unless vigilance is practiced. Dogs with even mild degrees of this disorder should not be bred as a consequence of the incomplete dominant heredity; Dogs with mild degrees of this defect can produce blind whelps.

### **Conclusions for all breeds**

Through the negative example of the Doberman in Germany, Europe and the USA, the reader can see that in less than 20 years the Doberman changed from a very healthy, vital and long-lived dog breed to a breed that is susceptible to disease with an average life-expectancy less than 7 years (West Germany 1989). The genetic degeneration of the whole population worldwide has progressed as a result of breeding with sick animals (phenotype and genotype). The average life-expectancy of over 5 years can be reached for many individual dogs only with great financial veterinarian expenditures (e.g. 1 pill of thyroid gland hormone daily, heart

drugs, frequent surgical operations against Wobbler and Bloat, blood coagulation compounds, drugs against Demodex immunity defect and others)!! Generally for all breeds with frequent health problems, veterinarian-medical measures (diagnostics and therapy) are not enough for the breeding animals, Additionally the gene pool of the whole breeding population must be improved by means of intelligent animal breeding and genetic breeding strategies i.e. especially by an effective selection of breeding dogs without heredity defects. The following veterinary and animal breeding measures should be achieved together:

1. All breeding animals should provide a certificate of standardized examination by University clinics covering all problem features of the breed (e.g. 5 features for the Doberman listed in Table 1). This certificate could also be used to make a close connection between breeding, health and exhibitioning. In other words, winning show dogs must prove their health superiority as well as being beautiful breed specimens.

2. The carrying out of these examinations requires dedicated, logistical preparations and arrangements between breeding authorities and universities, but the first step must always come from the breeding authorities that must provide leadership.

3. Inbreeding and pattern breeding (wide inbreeding) using breeding animals from the bloodlines with increased health risk (e. g. for death by heart failure and volvulus at the Doberman) must stop. The breeding authorities must develop intelligent external breeding strategies for their members, where health testing breeding animals would be the first priority.

4. The VDH (nationally in Germany), the AKC (in the USA) and the FCI (internationally) should write the genetic health as the 1<sup>st</sup> breeding goal of the purebred dog on their banners. For instance, by the reform of conformation shows (Herzog 1998), the registration organizations of the purebred dog breed could mandate favoring show dogs and breeding dogs with health certificates during the award of winner titles and during placing. In such a way the health, as the breeding goal, could be considerably promoted. Innovative, combat strategies against hereditary diseases, specifically for severely affected breeds like the Doberman (promoted and controlled by VDH and FCI) could improve the shaken reputation of the purebred dog in public, but first of all for the benefit of our dogs.

***Tendy vom Schrottturm, SchH III during the attack (right). These photos were taken by Dr. Haberzettl at a SchHIII trial in 1987 in East Berlin. Tendy was purchased as a pup (whelped 1984) and trained for protection by her owner Mr. Przakda, Director of Security at the Berlin-Schoenefeld Airport, after a very brutal hold-up as he was jogging through a Berlin park.***



***Mr. Bernd Przakda with the Doberman bitch Tendy vom Schrottturm, SchH III during the obedience phase. The Schutzhund sport obedience phase in East Germany had some exercises (dog walk) not included in the sport today.***

