Prevalence of inherited disorders among mixed-breed and purebred dogs: 27,254 cases (1995–2010)

Thomas P. Bellumori, MS; Thomas R. Famula, PhD; Danika L. Bannasch, PhD, DVM; Janelle M. Belanger, MS; Anita M. Oberbauer, PhD

Objective—To determine the proportion of mixed-breed and purebred dogs with common genetic disorders.

Design—Case-control study.

Animals—27,254 dogs with an inherited disorder.

Procedures—Electronic medical records were reviewed for 24 genetic disorders: hemangiosarcoma, lymphoma, mast cell tumor, osteosarcoma, aortic stenosis, dilated cardiomyopathy, hypertrophic cardiomyopathy, mitral valve dysplasia, patent ductus arteriosus, ventricular septal defect, hyperadrenocorticism, hypoadrenocorticism, hypothyroidism, elbow dysplasia, hip dysplasia, intervertebral disk disease, patellar luxation, ruptured cranial cruciate ligament, atopy or allergic dermatitis, bloat, cataracts, epilepsy, lens luxation, and portosystemic shunt. For each disorder, healthy controls matched for age, body weight, and sex to each affected dog were identified.

Results—Genetic disorders differed in expression. No differences in expression of 13 genetic disorders were detected between purebred dogs and mixed-breed dogs (ie, hip dysplasia, hypo- and hyperadrenocorticism, cancers, lens luxation, and patellar luxation). Purebred dogs were more likely to have 10 genetic disorders, including dilated cardiomyopathy, elbow dysplasia, cataracts, and hypothyroidism. Mixed-breed dogs had a greater probability of ruptured cranial cruciate ligament.

Conclusions and Clinical Relevance—Prevalence of genetic disorders in both populations was related to the specific disorder. Recently derived breeds or those from similar lineages appeared to be more susceptible to certain disorders that affect all closely related purebred dogs, whereas disorders with equal prevalence in the 2 populations suggested that those disorders represented more ancient mutations that are widely spread through the dog population. Results provided insight on how breeding practices may reduce prevalence of a disorder. (*J Am Vet Med Assoc* 2013;242:1549–1555)

Dogs are second only to humans in the number of hereditary diseases identified in the population.[1] Information about the prevalence and etiology of disorders in dogs may provide insight into preventative measures and possible treatments for dogs with diseases as well as for humans sharing common disorders.[2] Although no single registry maintains a record of genetic disease in dogs, it has been suggested that purebred dogs are more prone to genetic disorders than are mixed-breed dogs.[3] Breeding practices and selection pressures used by breeders of purebred dogs have been implicated in the perceived high frequency of genetic disorders, whereas the random mating practices of mixed-breed dogs have been suggested to increase hybrid vigor (heterosis), resulting in healthier dogs.[4]

The increased homozygosity expected in purebred dogs offers the potential for these animals to have traits influenced by recessive alleles in greater frequency than their crossbred counterparts. The common assumption that a mixed-breed dog is healthier would not be true if both parents carried deleterious mutations for the same disorder. Few data have been compiled to accurately assess the question of whether purebred dogs are at greater risk for genetic disorders, compared with mixed-breed dogs. In a study[5] of dogs affected with hip dysplasia, no significant difference in prevalence was observed between purebred and mixed-breed dogs.

Domestic dogs are thought to be derived from 3 to 5 wolf lineages.[6] Each lineage would be derived from a few common ancestors; thus, one might expect some disorders would be common to all dogs, regardless of breed. Genetic mutations that accompanied the domestication process would be expected to be widely distributed throughout the dog population, affecting dogs of any breed, including admixtures of breeds. In contrast

Abbreviations

AKC	American Kennel Club
CI	Confidence interval
IVDD	Intervertebral disk disease

From the Department of Animal Science, College of Agricultural and Environmental Sciences (Bellumori, Famula, Belanger, Oberbauer), and the Department of Population, Health and Reproduction, School of Veterinary Medicine (Bannasch), University of California-Davis, Davis, CA 95616.

Address correspondence to Dr. Oberbauer (amoberbauer@ucdavis.edu).

to more distant mutations, more recent selection pressure (eg, in Europe during the Victorian era[7]) would influence the distribution of newer mutations, restricting those to subsets of the overall dog population. It is likely that with breed refinement for specific tasks and morphology, some mutations accompanied selection for those traits. Rigorous selection pressures to refine the breeds by inbreeding and bottlenecks[4,8] would contribute to a loss of genetic diversity, thereby increasing the likelihood of recessive disorders within a breed population.

The AKC registers purebred dogs and records ancestors. Although, in 2004, there were > 140 AKC-registered breeds, 10 breeds represented more than half of the reported AKC-registered dogs, whereas the 100 least popular breeds represented < 15% of all AKC registrations.[9] The less popular breeds, with many fewer dogs registered each year, would be expected to have smaller effective gene pools. For example, the current population of Portuguese Water Dogs, ranked 56th in registrations as of 2011, has been traced back to a small number of dogs, mostly from 2 kennels, with approximately 6 ancestors comprising 80% of the current gene pool.[9] Breeds with smaller gene pools and reduced genetic variation are more likely to phenotypically express a recessive disorder.[1]

Many studies have sought to describe the prevalence of disorders among individual breeds. Often, the focus is on a single disorder and its inheritance pattern in a particular breed to define possible mutations. Yet, more global studies designed to assess the proportion of mixed-breed and purebred dogs affected with heritable disorders can prove useful toward reducing the prevalence of those disorders in the dog population. Describing disorders equivalently expressed within purebred and mixed-breed dogs may identify disorders common in the overall population and suggest approaches to reduce the prevalence. In contrast, disorders more prevalent to a particular breed may be reduced by use of concerted breeding practices.

A recent study[10] found a direct correlation between disorders inherited in purebred dogs and the morphological characteristics specified in the breed standard. Although that finding underscores the fact that purebred dogs are considered at risk for disorders, it is unknown whether mixed-breed dogs have the same risk of genetic disorders that is suggested for purebred dogs. The purpose of the study reported here was to describe the prevalence of genetic disorders in the dog population as a whole.

Materials and Methods

Case selection criteria—The data used in these analyses were obtained by searching through the University of California-Davis Veterinary Medical Teaching Hospital electronic records of all patients evaluated from January 1, 1995, through January 1, 2010. The genetic disorders selected for the study represented those expected to be present in the dog population at a measurable prevalence and to be debilitating, with confidence in the reliability of diagnosis. Additionally, disorders that affected a variety of anatomic locations and physiologic systems were chosen. Disorders in the following categories were assessed: cancers (hemangiosarcoma, lymphoma, mast cell tumor, and osteosarcoma), cardiac disorder (aortic stenosis, dilated cardiomyopathy, hypertrophic cardiomyopathy, mitral valve dysplasia, patent ductus arteriosus, and ventricular septal defect), endocrine disorders (hyperadrenocorticism, hypoadrenocorticism, and hypothyroidism), orthopedic disorders (elbow dysplasia, hip dysplasia, IVDD, patellar luxation, and ruptured cranial cruciate ligament), and other (atopy or allergic dermatitis, bloat, cataracts, epilepsy, lens luxation, and portosystemic shunt). Mode of inheritance was not a factor in the selection of the conditions under study.

Medical records review—Patient records contained fields that included pertinent history, clinical signs, clinical diagnosis, and other comments. Searches for keywords and any synonym or alternative representation for the genetic disorders were conducted in all fields. As an example, "Cushings," "Cushing's," "Cushing," and "hyperadrenocorticism" were all keyword searches to extract data related to hyperadrenocorticism. From each individual keyword search, a single database of patients was created for each disorder. In addition to disorder status, patient identification number, breed, sex, species, body weight, date of birth, admissions date, discharge date, search-term field (eg, pertinent history and clinical diagnoses), and keyword in context were captured. Each record was screened for accuracy, and only records with definitive confirmed diagnoses by the veterinary medical teaching hospital staff or the referring veterinarian were included for analyses. Any record that referred to suspected diseases, a presumptive diagnosis pending test results, rule-out diagnosis, or differential diagnosis or that included a diagnosis that was in any other way unconfirmed was omitted from analyses. For example, diagnoses of myxomatous mitral valvular disease were excluded from the mitral valve dysplasia category. The sole exception was epilepsy, for which the disorder was classified into 1 of 3 categories (confirmed, probable, or suspect) on the basis of the recorded information. Because of the nature of the records explaining specific vertebral problems, any dog with a laminectomy was considered to have IVDD, although laminectomy for cervical spondylomyelopathy was excluded. For each disorder, records were excluded such that only patients with a confirmed and reliable diagnosis of a particular disorder were retained. Regardless of the number of visits, a given dog was counted only once for a given disorder. To yield a comparison of healthy or diseased dogs with dogs evaluated at hospital for other reasons, a search for records of all dogs admitted after being hit by a car was also done.

The veterinary medical teaching hospital veterinary medical and administrative computer system was again searched to collect information on all of the dogs evaluated at the hospital from January 1, 1995, through January 1, 2010. This data file contained all dogs evaluated at the clinic, including those with and without the disorders that were under study, yielding information for each of the 268,399 visits. Data from the confirmed disorder files were matched to the full data file. In this way, individual patient records were matched so that all visit records for a single patient had the same diagno-

sis and any patient that may not have had the disorder listed for a specific visit was still classified as having the disorder. A given dog could have been classified as having multiple disorders if > 1 disorder was confirmed via diagnostic evaluation. From this file containing all unique dogs, control dogs were identified for use as hospital controls in accordance with clinical research designs.[11] Specifically, none of the conditions under study were diagnosed in these dogs.

Each patient had a breed designation. Dogs of AKC-recognized breeds, AKC miscellaneous breeds, or Foundation Stock Service breeds were considered to be purebred dogs. All nondomesticated canine patients (dingo or wolf) were removed. Pit bull–type dogs were evaluated independently because of the inability to validate purebred status. Any dog labeled as a mix was considered to be a mixed-breed dog. From the records collected, age at each visit could be calculated. For each dog, the age of first recorded diagnosis at the veterinary medical teaching hospital for each disorder was calculated and a mean age of first diagnosis was determined for each disorder.

Statistical analysis—For each disorder, appropriate population controls were identified from the complete data file containing all dogs evaluated at the veterinary medical teaching hospital in the 15-year time frame. Because the number of dogs lacking a given condition far exceeded the number of dogs with the condition, to create the control population against which the dogs with the condition were compared, it was necessary to randomly sample the dogs lacking the condition. Dogs were first stratified by body weight, sex, and age, and then each dog with a condition was matched to a randomly selected dog from the control group having the same weight, sex, and age classification. This sampling created control sets that represented the same characteristics as the affected dogs except for breed status. Control dogs were matched for age (0 to 2 years, > 2 to 7 years, or > 7 years), weight (0 to 12 kg [0 to 26.4 lb], > 12 to 20 kg [26.4 to 44 lb], or > 20 kg [44 lb]), and sex (male, castrated male, female, or spayed female) to each affected dog for each condition. The control dogs matched by the age, weight, and sex criteria were randomly selected from the complete data file, creating the control group for each disorder in accordance with clinical research designs.[11] Thus, the controls were from the same population base from which the dogs with disorders were derived.

To enhance the reliability of the analyses, the sampling set of healthy control dogs was repeated 50 times for each condition investigated. That is, for any given condition, an equal number of healthy dogs, stratified by the age, body weight, and sex of the affected dogs, were randomly selected 50 times to create repeated control data sets matched to the affected dogs. In this manner, the sole variable between the 50 randomly created data sets representing the control population was the number of mixed-breed or purebred dogs. In this way, 50 estimates (1 from each randomly selected set of controls) of the OR for the comparison of purebred with mixed-breed dogs as well as the mean 95% CI of this ratio and the mean *P* value used to test this ratio against the null hypothesis of 1.0 were calculated. In addition, by counting the number of data sets (of 50), the difference in disease risk between purebred and mixed-breed dogs could be determined.

All analyses were conducted via statistical software[a] with a logit link function for analysis of the binomial variable of disease status. The model included terms for age class, weight class, and sex as well as a term for purebred versus mixed-breed dog. Because each of the 50 data sets was balanced for age, weight, and sex groups, the OR for any of these variables should be 1.0, and this was monitored in all analyses as a test of the sampling process. The OR for purebred versus mixed-breed status for each of the 50 data sets was saved, as were the lower and upper limits of the 95% CI for this estimate and its associated *P* value. Also counted were the number of times (of 50 tests) the *P* value was less than or equal to the commonly used type I error rate of 0.05.

The number of dogs from each breed evaluated at the veterinary medical teaching hospital was determined as well as the number of dogs of each breed that were defined as control (no disorder) or affected (having ≥ 1 disorder). The percentage of each breed that was control or affected was then calculated.

Results

Of the 90,004 dogs examined at the veterinary medical teaching hospital small animal clinic that had an identified breed status (purebred, mixed, or pit bull–type), 27,254 had ≥ 1 of the conditions under study and 62,750 were control dogs (**Table 1**). In terms of the percentage of dogs of each breed with ≥ 1 disorder, 15 breeds had < 20% of dogs with ≥ 1 disorder, 63 breeds had from 21% to 30%, 41 breeds had from 31% to 40%, and 10 breeds had > 40%. The mean age at the first visit (assessed as the first appointment at the hospital with a disorder diagnosis) was calculated for each disorder (**Table 2**). Patent ductus arteriosus and ventricular septal defect were both diagnosed at a mean age of 1.32 years. Hyperadrenocorticism was diagnosed at a mean age of 10.54 years, the oldest age of diagnosis for any disorder. By comparison, dogs hit by a car had a mean age of 4.87 years.

Of the 24 disorders assessed, 13 had no significant difference in the mean proportion of purebred and mixed-breed dogs with the disorder when matched for age, sex, and body weight (Table 2). Disorders without a significant predisposition included all the neoplasms (hemangiosarcoma, lymphoma, mast cell tumor, and osteosarcoma), hypertrophic cardiomyopathy, mitral valve dysplasia, patent ductus arteriosus, and ventricular septal defect in the cardiac category; hip dysplasia and patellar luxation in the orthopedic category; hypoadre-

Table 1—Breed distribution of dogs with (Condition) and without (Control) inherited disorders evaluated at the Veterinary Medical Teaching Hospital, University of California-Davis, in a 15-year period.

Breed	Control	Condition	Total
Purebred	45,015	20,937	**65,952 (73.3%)**
Mixed	16,693	5,990	**22,683 (25.2%)**
Pit bull–type	1,042	327	**1,369 (1.5%)**
Total	**62,750**	**27,254**	**90,004 (100%)**

Table 2—Distribution and descriptive statistics of mixed-breed and purebred dogs with inherited conditions diagnosed over a 15-year period.

Disorder or injury	Mixed (No. of dogs)	Purebred (No. of dogs)	Total (No. of dogs)	Mean age at first diagnosis (y)	Mean OR (95% CI)	Mean *P* value	No. of times breed was significant
Cardiac							
Aortic stenosis*	33	357	390	3.0	3.03 (1.96–4.76)	0.000	50
Dilated cardiomyopathy*	32	329	361	7.23	3.45 (2.22–5.26)	0.000	50
Hypertrophic cardiomyopathy	3	33	36	6.51	2.04 (0.40–10.0)	0.336	9
Mitral valve dysplasia	40	180	220	4.09	1.85 (0.73–1.96)	0.446	5
Patent ductus arteriosus	81	329	410	1.32	0.85 (0.60–1.22)	0.480	3
Ventricular septal defect	16	117	133	1.32	1.72 (0.86–3.45)	0.168	15
Cancer							
Hemangiosarcoma	135	427	562	9.19	1.25 (0.95–1.64)	0.186	17
Lymphoma	392	1,182	1,574	8.0	1.11 (0.94–1.30)	0.271	8
Mast cell tumor	342	1,105	1,447	8.0	1.20 (1.01–1.43)	0.068	32
Osteosarcoma	187	522	709	8.23	1.09 (0.86–1.39)	0.449	3
Orthopedic							
Elbow dysplasia*	191	1,034	1,225	3.54	2.00 (1.63–2.50)	0.000	50
Hip dysplasia	500	1,431	1,931	3.89	1.05 (0.91–1.23)	0.473	4
IVDD*	833	3,658	4,491	7.35	1.41 (1.26–1.56)	0.000	50
Patellar luxation	466	1,710	2,176	5.16	1.04 (0.90–1.20)	0.490	0
Ruptured cranial cruciate ligament†	400	828	1,228	5.95	0.79 (0.67–0.94)	0.031	41
Endocrine							
Hyperadrenocorticism	281	808	1,089	10.54	1.02 (0.84–1.23)	0.593	0
Hypoadrenocorticism	67	228	295	8.72	1.23 (0.83–1.79)	0.354	5
Hypothyroidism*	326	1,369	1,695	6.86	1.56 (1.33–1.85)	0.000	50
Other							
Atopy or allergic dermatitis*	237	1,094	1,331	5.95	1.56 (1.30–1.89)	0.003	50
Bloat*	35	187	222	6.92	1.79 (1.10–2.94)	0.054	36
Cataracts*	734	2,822	3,556	9.21	1.27 (1.12–1.41)	0.000	50
Epilepsy total*	188	749	937	6.24	1.37 (1.10–1.69)	0.016	47
Epilepsy confirmed	146	565	711	6.57	1.33 (1.03–1.79)	0.062	28
Epilepsy probable	24	120	144	5.26	1.61 (0.88–2.94)	0.158	13
Epilepsy suspect	18	64	82	5.32	1.03 (0.48–2.22)	0.536	1
Lens luxation	64	251	315	9.07	1.14 (0.78–1.69)	0.478	2
Portosystemic shunt*	74	608	682	2.39	2.04 (1.49–2.77)	0.000	50
Hit by car†	569	1,069	1,638	4.87	0.59 (0.51–0.69)	0.000	50

Mean *P* value indicates comparison of purebred dogs with matched control sampling sets. Number of times breed was significant = Number of times (of 50) that comparison of affected dogs with matched control sampling sets indicated a significant ($P < 0.05$) difference in probability that mixed-breed and purebred categories differed in expression of the condition. Mean OR (95% CI) indicates comparison of purebred dogs relative to mixed-breed dogs.

*Purebred dogs had a greater probability of expressing the condition. †Mixed breeds had a greater probability of expressing the condition. Epilepsy total consists of the sum of all 3 categories of epilepsy.

nocorticism and hyperadrenocorticism in the endocrine category; and lens luxation in the other category.

In contrast, 10 disorders were more prevalent in purebred dogs, compared with those found in mixed-breed dogs. Aortic stenosis and dilated cardiomyopathy in the cardiac category, hypothyroidism in the endocrine category, elbow dysplasia and IVDD in the orthopedic category, and atopy or allergic dermatitis, bloat, cataracts, total epilepsy, and portosystemic shunt were all diagnosed in a greater proportion of purebred dogs than mixed-breed dogs ($P < 0.05$). The OR for these disorders ranged from 1.27 (cataracts) to 3.45 (dilated cardiomyopathy) for purebred dogs, relative to mixed-breed dogs, indicating a greater probability of the condition in purebred dogs.

Cranial cruciate ligament rupture and being hit by a car were more likely to be observed in mixed-breed dogs than purebred dogs, with a 1.3- and 1.7-fold probability of the condition, respectively. Whereas the percentage of purebred dogs evaluated at the veterinary medical teaching hospital during this time frame was 73.3% and for mixed-breed dogs was 25.2%, the percentage of mixed-breed dogs evaluated after being hit by a car was 35% and significantly ($P < 0.05$) greater than expected (Table 2); a similar higher-than-expected percentage was observed for pit bull–type dogs.

Ten genetic disorders had a significantly greater probability of being found in purebred dogs. For aortic stenosis, the top 5 breeds affected on the basis of the percentage of dogs of that breed affected and mixed breeds were Newfoundland (6.80%), Boxer (4.49%), Bull Terrier (4.10%), Irish Terrier (3.13%), Bouvier des Flandres (2.38%), and mixed breed (0.15%); for dilated cardiomyopathy, breeds included Doberman Pinscher (7.32%), Great Dane (7.30%), Neapolitan Mastiff (6.52%), Irish Wolfhound (6.08%), Saluki (5.88%), and mixed breed (0.16%). Breeds affected with elbow dysplasia included Bernese Mountain Dog (13.91%), Newfoundland (10.28%), Mastiff (6.55%), Rottweiler (6.31%), Anatolian Shepherd Dog (5.41%), and mixed breed (0.90%); for IVDD, Dachshund (34.92%), French Bulldog (27.06%), Pekingese (20.59%), Pembroke Welsh Corgi (15.11%), Doberman Pinscher (12.70%), and mixed breed (4.43%); for hypothyroidism, Giant Schnauzer (11.45%), Irish Setter (7.69%), Keeshond (6.63%), Bouvier des Flandres (6.55%), Doberman Pinscher (6.30%), and mixed breed (1.54%); for atopy or allergic dermatitis, West Highland White Terrier (8.58%), Coonhound (8.33%), Wirehaired Fox Terrier (8.16%), Cairn Terrier (6.91%), Tibetan Terrier (5.86%), and mixed breed (1.08%); for bloat,

Saint Bernard (3.76%), Irish Setter (3.42%), Bloodhound (3.39%), Great Dane (2.80%), Irish Wolfhound (2.70%), and mixed breed (0.20%); for cataracts, Silky Terrier (22.76%), Miniature Poodle (21.49%), Brussels Griffon (20.51%), Boston Terrier (19.61%), Tibetan Terrier (18.92%), and mixed breed (4.04%); for epilepsy (total), Catahoula Leopard Dog (3.90%), Beagle (3.57%), Schipperke (3.42%), Papillon (3.40%), Standard Poodle (3.19%), and mixed breed (0.91%); and for portosystemic shunt, Yorkshire Terrier (10.86%), Norwich Terrier (7.41%), Pug (5.88%), Maltese (5.87%), Havanese (4.35%), and mixed breed (0.35%). No single breed dominated the listings. Labrador Retrievers and mixed-breed dogs were more frequently evaluated at the veterinary medical teaching hospital; therefore, those dogs typically had a greater prevalence of every disorder. However, the most frequent breeds affected by each disorder changed when adjusted for absolute numbers of dogs of that breed evaluated at the clinic. Although some breeds appeared multiple times in different disorders, no breed dominated by the percentage of breed affected.

Discussion

This study characterized the prevalence of genetic disorders among purebred and mixed-breed dogs evaluated at the veterinary medical teaching hospital. The study was designed specifically to evaluate purebred dogs, compared with mixed-breed dogs in total, without attempting to evaluate individual breed prevalence. One concern with this approach is that a breed-specific disorder found in a high-population breed may inflate the prevalence among purebred dogs, unduly influencing interpretation of the results. This did not appear to be the case because in those conditions with a difference in prevalence between purebred and mixed-breed dogs, none of the top 5 breeds (as a percentage of dogs evaluated at the hospital) were high-population breeds.

The results indicated that genetic disorders were individual in their expression throughout the dog population. Some genetic disorders were present with equal prevalence among all dogs in the study, regardless of purebred or mixed-breed status. Other genetic disorders were found in greater prevalence among purebred dogs. Every disorder was seen in the mixed-breed population. Thus, on the basis of the data and analyses, the proportion of mixed-breed and purebred dogs affected by genetic disorders may be equal or differ, depending on the specific disorder.

Although this study evaluated > 90,000 purebred and mixed-breed dogs, there were limitations to the study. The study population represented dogs evaluated at a teaching hospital, and the proportions of the disorders in the purebred and mixed-breed dogs may have been different from that in the general canine population. However, the study population did reflect the proportions of purebred and mixed-breed dogs evaluated at private veterinary hospitals in the United States.[12] In a referral hospital, breeds that are considered predisposed to a certain condition may be evaluated with greater frequency and the condition may be diagnosed at a higher rate than in other breeds or mixed-breed dogs that do not have a recognized predisposition. This would cause an overrepresentation of some disorders in purebred dogs. Additionally, clients are willing to pursue more extensive treatment at a referral hospital.[13] Owners of purebred dogs are more likely to spend more on their dogs than are owners of mixed-breed dogs,[14] which would result in a greater proportion of purebred dogs, as seen in the present study. Some dogs in the present study not classified as having a particular condition may simply not have had that condition confirmed because of the age of onset or the expense of definitive diagnostic procedures. For example, epilepsy, atopy (allergic dermatitis), and hypothyroidism, all of which have higher probability in purebred dogs, require more intensive diagnosis, and there may be sociological aspects in which dog owners who own mixed-breed dogs may have less incentive to confirm the diagnosis.

Data for an acute onset of a disorder may have been underrepresented in our data set if clients preferentially took the dog to their own veterinarian and not a teaching hospital. Furthermore, the Veterinary Medical Teaching Hospital of the University of California-Davis represents a dog population primarily from the west coast and may not represent dog populations in other geographic regions. However, for 1 condition in the present study (portosystemic shunt), the data and the breeds preferentially affected mirrored data for all of North America.[15]

All of these biases would be expected equally among mixed-breed and purebred dogs in the population under study, or a bias specifically against the purebred dog population may have occurred; neither would affect the objective of the study. Although these are potential limitations to the data, overall, the data set that was evaluated is, in the authors' opinion, one of the best representations to include consistent diagnoses in large numbers of purebred and mixed-breed dogs.

A previous study[5] found no difference between purebred and mixed-breed dogs with hip dysplasia. Our results, which corroborate the findings of the previous study,[5] indicated that in addition to hip dysplasia, several other disorders did not predominate among purebred dogs. For genetic disorders that are found in multiple breeds or are equally present in mixed-breed dogs, causal mutations may have arisen multiple times or the progenitors of the affected dogs may have been derived from a common distant ancestor carrying the defect. Mutations introduced into the dog genome early, in an ancestor closely associated with the wolf progenitor, would be spread through the dog population at large. Perhaps the same desired traits that made dogs a favorable species for domestication[16] were linked to alleles for hyperadrenocorticism, hypoadrenocorticism, cancers, hip dysplasia, lens luxation, and some cardiac disorders that were not found to be different between purebred and mixed-breed dogs.

Alternatively, the selection for desirable morphological traits may be linked to the presence of deleterious alleles. Patellar luxation and lens luxation are clear examples of size-oriented predisposition. These disorders did not differ in prevalence between purebred and mixed-breed dogs, yet appear to be more common among smaller dogs. Another potential explanation for a disorder's equal prevalence in purebred and mixed-

breed dogs is that some tissues or organs may be less resistant to genetic aberration and a number of different mutations may induce a similar phenotypic defect, even though the precise mutations differ in the 2 dog populations. Additionally, developmental abnormalities influenced by the environment or stochastic developmental perturbations (eg, certain cardiac conditions)[17] would result in the same disease diagnosis. No significant difference was found for cancers between purebred and mixed-breed dogs. Genes for cancer expression may be spread widely among the dog population as a whole, respond to environmental factors that affect all dogs, or a combination of both.

For disorders that affected purebred dogs in higher proportions, the underlying causal mutations likely occurred more recently, such as after the gene pools for particular purebred dogs were developed, or were characteristic of particular lineages. In this study, 4 of the top 5 breeds (by percentage) affected with elbow dysplasia are characterized as being from the Mastiff-like dog lineage[9]: Bernese Mountain Dog, Newfoundland, Mastiff, and Rottweiler. One could speculate that these breeds, having been derived from a common ancestor,[18] share mutations. Transmission of genetic disorders may not only occur within a single antiquity lineage, but also may occasionally cross to another lineage as a result of desire for particular functional traits.[8] A 1998 study[19] supports this idea by revealing that certain disorders, such as elbow dysplasia and portosystemic shunt, occurred in clusters of highly related dogs, whereas clusters of unrelated dogs were unaffected. Additionally, the purebred population was at greater risk for atopy than was the mixed-breed dogs. The published literature indicates that certain breeds are more likely to have atopy than other breeds,[20,21] suggesting that the high prevalence within individual breeds may result in the overall purebred population being at greater risk than the population of mixed-breed dogs. Reports of mixed-breed dogs having equivalent atopy prevalence to subsets of purebred dogs[22] support the existence of such an effect and underscore the concept of clustering of disorders among highly related dogs.

Disorders may be associated with breed derivation or with breed bottlenecks. Such an example is the Irish Wolfhound, a breed with relatively few dogs registered annually. In the mid-1800s, the Irish Wolfhound underwent a population bottleneck so severe that the breed was thought to be extinct.[23] The reduced effective population size suggests a relationship with the concomitant increased risk of dilated cardiomyopathy in Irish Wolfhounds. Indeed, as many as 1 in 3 Irish Wolfhounds may be affected with this disorder.[23] In the present study, Irish Wolfhounds were in the top 5 purebred dog breeds with dilated cardiomyopathy, corroborating the high prevalence, compared with other breeds.

Other disorders appear to be more generalized and more frequently observed in mixed-breed dogs. For example, metabolic disturbances have been implicated in the onset of canine diabetes mellitus, for which the risk of development is higher in mixed-breed dogs.[24] In the present study, dogs with cranial cruciate ligament rupture included purebred dogs from at least 3 lineages (ie, Mastiff, Akita, and German Wirehaired Pointer),[9] with mixed-breed dogs having a 30% greater risk for this disorder than did purebred dogs. The increased risk may be caused by multiple musculoskeletal alleles from different physical conformations that, when combined, reduce the resilience of the ligament in the context of the joint, as has been suggested for humans.[25]

Purebred dog owners, often devoted to a breed and seeking to track the health of that breed, may have created the impression that purebred dogs are not as healthy as mixed-breed dogs. Overall, the prevalence of disorders among purebred and mixed-breed dogs in the present study depended on the condition, with some having a clear distinction between purebred and mixed-breed dogs and others having no difference. Our results confirmed those of other studies focused on hip dysplasia[5] and congenital portosystemic shunts[15] and expanded the potential for future genetic studies to focus on several breeds when considering at-risk breeds to characterize the underlying genetic change. These results also gave insight on the potential effects of breeding practices to reduce prevalence. Reliable genetic tests or screening at a young age may reduce some disorders in the dog population as a whole. Additionally, some disorders may require breed registry intervention to reduce conformational selection pressures that contribute to predisposing a breed to a disorder.

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From this month's *AJVR*

Efficacy of decontamination and sterilization of single-use single-incision laparoscopic surgery ports

James G. Coisman et al

Objective—To determine the efficacy of decontamination and sterilization of a disposable port intended for use during single-incision laparoscopy.

Sample—5 material samples obtained from each of 3 laparoscopic surgery ports.

Procedures—Ports were assigned to undergo decontamination and ethylene oxide sterilization without bacterial inoculation (negative control port), with bacterial inoculation (*Staphylococcus aureus*, *Escherichia coli*, and *Mycobacterium fortuitum*) and without decontamination and sterilization (positive control port), or with bacterial inoculation followed by decontamination and ethylene oxide sterilization (treated port). Each port underwent testing 5 times; during each time, a sample of the foam portion of each port was obtained and bacteriologic culture testing was performed. Bacteriologic culture scores were determined for each port sample.

Results—None of the treated port samples had positive bacteriologic culture results. All 5 positive control port samples had positive bacteriologic culture results. One negative control port sample had positive bacteriologic culture results; a spore-forming *Bacillus* sp organism was cultured from that port sample, which was thought to be an environmental contaminant. Bacteriologic culture scores for the treated port samples were significantly lower than those for the positive control port samples. Bacteriologic culture scores for the treated port samples were not significantly different from those for negative control port samples.

Conclusions and Clinical Relevance—Results of this study indicated standard procedures for decontamination and sterilization of a single-use port intended for use during single-incision laparoscopic surgery were effective for elimination of inoculated bacteria. Reuse of this port may be safe for laparoscopic surgery of animals. (*Am J Vet Res* 2013;74:934–938)

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