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Ophthalmic disease and screening in breeding dogs

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Canine breed screening ophthalmic examination

The American College of Veterinary Ophthalmologists (ACVO) and the Orthopedic Foundation for Animals (OFA) work jointly to operate the Companion Animal Eye Registry (CAER). Through screening ophthalmic examinations and certifications with associated breeding recommendations, the goal is to reduce the incidence of comfort- and vision-threatening inherited ophthalmic conditions in animals, mainly purebred dogs.

History and current organization

In 1970, Dolly Trauner established the Society for the Investigation of Hereditary Defects in Dogs. This group performed pedigree analyses and determined coefficients of inbreeding to reduce the likelihood that dogs affected with inherited diseases would be bred. However, progress was limited by resistance of owners of affected dogs to make pedigrees available. In 1974, Mrs. Trauner established the Canine Eye Registry Foundation (CERF) in an attempt to mediate the lack of pedigree information. The goals for CERF were to promote recognition of inherited ophthalmic conditions in dogs and implement methods for control of these via breeding practices. (G. Aguirre, personal communication, January 2023) This involved ophthalmic examinations of breeding dogs, which eventually became exclusively performed by Diplomates of the ACVO (DACVO); and the Veterinary Medical Database

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(VMDB) through 2013, and OFA beginning 2012 to present, as registry and certifying organizations for these CERF and CAER exams respectively.

The American Board of Veterinary Ophthalmologists standardizes and oversees the residency training programs and certification of DACVO. Performing and interpreting ophthalmic examinations are foundational to the practice of veterinary ophthalmology and are thus a focus of the training and credentialing process, though residents may or may not be exposed to CAER examinations specifically. Once diplomate status is conferred to candidates, they receive an orientation to the nuances of breed screening CAER ophthalmic examinations. This is provided by the OFA CAER program manager and either the ACVO-OFA liaison or a member of the ACVO genetics committee. The ACVO-OFA liaison and ACVO genetics committee members are all ACVO board-certified veterinary ophthalmologists (DACVO). The OFA CAER program manager, ACVO-OFA liaison, and members of the ACVO genetics committee are also available to answer examiner, owner and breed club questions regarding these exams.

Ocular Disorders Presumed to be Inherited in Purebred Dogs

The ACVO genetics committee, with the help of OFA personnel and registry information, compiles and evaluates information regarding known and presumed inherited ophthalmic conditions in dogs to generate the document *Ocular Disorders Presumed to be Inherited in Purebred Dogs*, often referred to solely as *The Blue Book*.¹ It is available online via the ACVO (acvo.org) and OFA (ofa.org/diseases/eye-disease/blue-book/) websites, and serves as a resource for veterinarians and owners; and as the guide for diagnostic coding of CAER exam findings, and ultimately certification and breeding recommendations. *The Blue Book* is updated annually based on registry data and relevant published literature reviewed by the ACVO genetics committee. Evidence-based breed club requests, available genetic tests and ACVO genetics committee discretionary recommendations may also generate updates to *The Blue Book*.¹ Introduction, 2

American Kennel Club-recognized breeds are each given a specific “page” within *The Blue Book* if warranted by breed CAER data and scientific information. There, specific ophthalmic disorders are listed for each breed if examined dogs are reported in sufficient numbers to detect presumed inherited ophthalmic conditions via registry data or if scientific studies or a genetic test has identified inherited ophthalmic conditions. Sufficient numbers are defined as an incidence $\geq 1\%$ of the examined population with a minimum of 5 affected animals per five-year period or 50 or more affected animals in a five-year period regardless of the population of dogs examined.¹ Descriptive information about the listed disorder are provided, including the mode of inheritance or genetic mutation if known, and cited with appropriate references.¹

Additionally, breeding advice is given for each listed disorder of each breed. “Breeder option” conditions are either unlikely inherited though the possibility cannot yet be definitively ruled out (e.g., suspect not inherited/significance unknown cataracts), or suspected or known to be inherited but not of significant potential threat to ocular comfort or vision in either the individual dog nor potential offspring.¹ Awareness of such conditions

and status enables and empowers breeders to responsibly breed away from the condition as indicated, hence the designation “breeder option.” A “no” or do not breed recommendation is listed on the breed page for disorders with very strong evidence of heritability and significant potential threat to ocular comfort or vision in either the individual or the offspring.¹ “No” breeding advice for a given disorder in a given breed may also be stated per breed club request at the discretion of the ACVO genetics committee. Finally, certain CAER exam diagnoses generate “no” breeding advice irrespective of whether or not the disorder is listed on the dog’s Blue Book breed page because they have both strong evidence of heritability, and frequently result in ocular pain and/or blindness in the offspring and sometimes the individual. (Table 1)

Each breed “page” concludes with its associated complete ocular disorders report, which lists prevalence of all diagnoses for reported examined dogs of the breed.

Ophthalmic examination process and certification

To enhance sensitivity of detection of inherited conditions, which are frequently not congenital, the ACVO recommends serial annual ophthalmic examinations of dogs in active breeding lines of all breeds,¹ Introduction though some breed clubs have their own recommendations based on specific concerns for their breeds. Serial examination with associated breeding advice by the German panel of veterinary ophthalmologist has been shown to reduce incidence of certain inherited ophthalmic disorders (cataract, progressive retinal atrophy) in German dachshunds.³

A breed screening CAER ophthalmic exam thoroughly assesses features of the orbits, eyelids, and globes (from corneas to retinas and optic nerves), and is performed by a DACVO using slit lamp biomicroscopy and binocular indirect ophthalmoscopy. It is performed after pharmacologic mydriasis (pupil dilation) to best allow thorough evaluation of the peripheral/entire lenses and ocular fundi. Dogs of certain breeds with known inherited disorders of the iris should be examined before dilation to best allow thorough evaluation of the anterior segment for the following disorders: dalmatian - iris hypoplasia/sphincter dysplasia; Australian shepherd, miniature American shepherd/minature Australian shepherd, and toy Australian shepherd - iris coloboma.¹ They should subsequently be dilated and otherwise examined fully as above. All findings of the breed screening CAER ophthalmic exam, though with focus on those potentially significant to breeding (i.e., known, suspected or possibly inherited), are noted by the diplomate examiner on a specific CAER exam form. (Figure 1) This allows most appropriate documentation and communication, and ultimately appropriate and consistent diagnosis/coding and certification with breeding recommendations dictated by the ACVO genetics committee via *The Blue Book* through OFA.

It should be noted that a Schirmer tear test (STT), fluorescein staining, tonometry, gonioscopy, high resolution ultrasonographic imaging for evaluation of the iridocorneal angle, electroretinography, and streak retinoscopy to determine refractive error, are not components of the routine CAER exam. Certainly these ophthalmic diagnostic tests may provide additional information relevant to the breed screening CAER ophthalmic exam (i.e.,

diseases they test for may be inherited) or the individual canine patient being examined (i.e., they may warrant intervention to reduce risk of ocular discomfort and or visual impairment). They may thus be performed at the discretion of the examiner or request of the owner and documented as “comments” on the exam form. Additionally, in some cases (e.g., where KCS is suspected but cannot be confirmed solely via examination findings) such tests (in this case, a STT) may be requested or required to be performed before an official diagnosis can be given and thus a certificate or other result generated at OFA. However, because these additional ophthalmic exam components are not included in the vast majority of CAER exams, early cases where physical ophthalmic exam signs are not yet present could be missed.¹

With input from the ACVO Vision for Animals Foundation Canine Glaucoma Consortium, the ACVO genetics committee and OFA have created a separate gonioscopy exam form. Though currently only in the data collection phase, the goal of this form is to establish a standardized database for additional information related to the iridocorneal angle/aqueous humor outflow and potential risk factors for glaucoma in purebred and breeding dogs. Like the general breed screening CAER ophthalmic exam, the ultimate goals of this additional arm of the program are to promote recognition and allow understanding of predispositions to inherited primary glaucoma in dogs (i.e., are there predictable iridocorneal angle factors and exam findings?) and make evidence-based breeding recommendations to reduce such. At present, if performed, gonioscopy and any other iridocorneal angle assessment findings may be written in as comments on the CAER form as above, or a gonioscopy form may be completed.

Though examiners provide and document gonioscopic findings to owners and may interpret and make breeding recommendations regarding these results, no certification or recommendation is provided by OFA/ACVO since both performing that examination itself and interpreting what it means for the individual dog and its breed lines is complicated and lacks consensus. Dogs having had gonioscopy performed and documented on a CAER or gonioscopy form submitted to OFA receive a denotation on their CAER certificates that it was done, but again, no official interpretation or recommendations occur.

It is important to note that like most physical exam subsets, breed screening CAER ophthalmic exams are phenotypic. Dogs may be carriers of inherited conditions or even genetically affected but not (yet) displaying the phenotype since many non-congenital inherited diseases manifest later in life, often after dogs have been bred. In these cases, CAER exams could be normal despite the possibility of dogs passing on problematic genes and ultimately disorders. Many genetic defects with ophthalmic ramifications are not yet known or do not have an available genetic test. In addition, available genetic testing is very specific (e.g., may rule in or out one form of inherited retinal degeneration but not others), whereas the phenotypic screening CAER exam, which looks at the entire ophthalmic system, is broad. Therefore, it is recommended that dogs in active breeding lines receive phenotypic CAER exams that are complemented with genetic testing for specific disorders in dogs of known affected breeds.

Given the screening nature of CAER exams, they attract a biased population and generate biased, likely underestimated, data regarding the true incidence of disorders in affected dog breeds. Dogs with ophthalmic disorders that cause noticeable pain and or vision loss, or even just obviously visible lesions may not be presented for and diagnosed via CAER exams as their severity may have already necessitated veterinary attention. Asymptomatic but phenotypically present conditions are more likely to be detected on this type of exam.

Owner copies of completed CAER exam forms may be submitted to OFA for registry and certification. A database is created from this registry along with CAER exam form research copies which capture instances of examined dogs where owners do not submit results. Review by the ACVO-OFA liaison will clarify unclear diagnoses and cross reference findings with any previous exams on file. Ultimately, diagnoses are finalized and certificate codes and breeding recommendations are generated per *The Blue Book* guidelines and breed pages. Though forms may be submitted any time, certification and breeding recommendations expire a year after the date of the exam to encourage serial screening and enhance the sensitivity of detecting inherited conditions.

Selected specific conditions screened for on a CAER examination

Common or otherwise particularly problematic conditions from each section of the CAER form are described and discussed below.

Multiple canine eyelid conditions are likely inherited though the causative mutations are unknown. These adnexal disorders usually generate a “breeder option” breeding recommendation when diagnosed on a CAER exam because they are complexly inherited and cause less severe pathology than disorders where breeding is not recommended.

- Distichia are abnormal eyelashes that emerge through the eyelid margin, most commonly through the meibomian gland orifices. (Figure 2) They are usually asymptomatic but can cause squinting, tearing and corneal ulceration in some instances.
- Entropion is an inward rolling of the eyelid margin that causes irritation and corneal ulceration when periocular hair rubs on the ocular surface. In contrast, ectropion is an outward rolling of the eyelids that can cause ocular surface irritation via exposure. Both entropion and ectropion are likely influenced by multiple factors related to facial conformation, and therefore may be inadvertently selected for as breeders seek a desired appearance in their dogs. At the request of their breed clubs, entropion in the Rhodesian ridgeback and rottweiler has been designated as a “no” breeding recommendation.¹

Corneal disorders with a presumed or known genetic basis in dogs vary in their clinical significance and therefore also in CAER breeding recommendations.

- Chronic superficial keratitis, also known as pannus, is an immune-mediated corneal disease characterized by vascularization, fibrosis, pigmentation and infiltrate often to the extent of pink proliferative tissue formation. (Figure 3) Pannus of the nictitans can occur in isolation (plasmoma or atypical

pannus) or concurrent with corneal disease and is manifested by a pink nodular appearance to the normally smooth pigmented third eyelid conjunctiva. Blindness can occur when corneal disease is severe. A genetic basis is suspected in German shepherds (GSD), greyhounds, Belgian tervurens, Belgian sheepdogs and Maremma sheepdogs due to breed predilections and sufficient incidence in CAER data.^{1, 4–6} A CAER exam diagnosis of pannus receives a “no” breeding recommendation. This has seemingly improved breeding practices and decreased incidence of the condition in GSD presented for CAER exams: in 2013, 150 GSD were examined and 2% were affected with plasmoma, 1.3% with corneal pannus; in 2022, 209 were examined and 0% and 0.5% were affected with plasmoma and corneal pannus respectively. (J. Pautz, personal communication of breed data report, January 2023)

Epithelial-stromal corneal dystrophy is characterized by the accumulation of white to gray opacities, most often lipid, within one or more corneal layers. (Figure 4) It rarely causes pain or blindness and usually does not require treatment; therefore, for most breeds this diagnosis receives a breeding recommendation of “breeder option”. This still allows breeding practices to select away from the disorder and the cavalier King Charles spaniel (CKCS) provides an example of a commonly affected breed with decreasing incidence in dogs presented for CAER exams: in 2013, with 2520 CKCS examined, 10% were affected with corneal dystrophy; in 2022, with 3192 CKCS examined, 7.6% were affected. (J. Pautz, personal communication of breed data report, January 2023) Exceptions to the breeder option recommendation for corneal dystrophy include “Sheltie corneal dystrophy” in the Shetland sheepdog, which carries a “no” breeding recommendation because the disease results in an unstable overlying tear film and often painful corneal erosions warranting treatment;^{1, 7} and the Siberian husky where the inheritance is presumed to be autosomal recessive, and the opacities are often more severe, potentially resulting in vision impairment.^{1, 8–11}

- Endothelial corneal dystrophy results in progressive corneal edema. When severe, this disease causes pain via chronic corneal ulcerations and vision impairment from corneal opacification. Predispositions have been identified in the basenji, Boston terrier, chihuahua and dachshund, therefore a “no” breeding recommendation is made for these breeds due to the presumption of hereditary disease.^{1, 8} Based on sufficient incidence in CAER data, endothelial corneal dystrophy is also listed as a disorder for the petit basset griffon vendeen with a “breeder option” breeding recommendation.¹ Corneal endothelial degeneration, which is usually age related and may present similarly clinically, does not generate any specific breeding recommendations.
- Macular corneal dystrophy is a unique form of corneal dystrophy in Labrador retrievers with autosomal recessive inheritance from a mutation in the CHST6 gene.¹² This disease usually affects dogs aged 4–6 years and is characterized by bilateral stromal grey-tan circular corneal opacities of varying size with potential associated vascularization.¹³ A commercial genetic test is available, and a “no” breeding recommendation is made if this disorder is diagnosed on a CAER exam.

- Pigmentary keratitis, also known as pigmentary keratopathy, is defined as the deposition of superficial corneal pigment which is usually accompanied by fibrosis and vascularization. (Figure 5) It is most often a problem in brachycephalic dogs, especially pugs, where ocular surface irritation and exposure from facial conformation such as medial lower eyelid entropion and macropalpebral fissure (wide eyelid openings) may play a role.¹⁴ As with the previously discussed adnexal disorders, selection for a certain facial appearance may result in an increased prevalence of pigmentary keratitis. However, one study in pugs did not find an association between facial conformation and corneal pigmentation and therefore surmised that the disorder may have its own genetic basis separate from corneal pigmentation secondary to irritation and exposure considerations.¹⁵ This disorder is categorized as “breeder option” since it is not fully understood, does not threaten ocular comfort, and though may be vision impairing when severe, often is milder. Again, this still allows breeding practices to select away from the disorder and the incidence in pugs presented for CAER exams went from 73.3% (of 101 dogs examined) in 2013 to 46.2% affected (of 173 dogs examined) in 2022. (J. Pautz, personal communication of breed data report, January 2023)

Uveal disorders vary in severity and breeding recommendations.

- Uveal cysts are fluid-filled semi-transparent circular structures arising from the epithelium of the posterior iris or ciliary body. (Figure 6) They can be variably pigmented, vary in size and remain attached or break free to float in the anterior chamber. Cysts may rupture, and deposit pigment on intraocular tissues. Uveal cysts rarely cause clinical problems and in all breeds are receive a breeding recommendation of “breeder option”. However, in the golden retriever uveal cysts may be associated with pigmentary uveitis and secondary glaucoma, and they may be associated with glaucoma in the great dane and American bulldog.¹⁶⁻¹⁹
- Pigmentary uveitis is a poorly understood and potentially devastating condition that occurs in middle-aged to older golden retrievers¹⁶ and carries an OFA “no” breeding recommendation. (Figure 7) It begins with the presence of radial pigment deposition on the anterior lens capsule and can progress to cause posterior synechiae, secondary cataract and in its most advanced form, secondary glaucoma.²⁰⁻²¹ This disease has been associated with the presence of uveal cysts, though not all golden retrievers with uveal cysts will progress to develop pigmentary uveitis.^{17, 21-22} Since dogs in the initial stage of this disease are often asymptomatic, screening ophthalmic exams may allow earlier detection and altered breeding practices. At later stages, affected dogs are usually recognized when clinical signs prompt veterinary attention. However, elimination of this disease has been challenging because it does not yet have a genetic test and affected dogs are usually not diagnosed until after they may have been bred. This said, with recognition and earlier diagnosis of the disorder, the incidence in golden retrievers presented for CAER exams has decreased: 2% (of 6458 dogs

examined) in 2013 to 1.1% affected (of 9619 dogs examined) in 2022. (J. Pautz, personal communication of breed data report, January 2023)

- An iris coloboma is a congenital full-thickness defect of the iris that occurs as a developmental anomaly. It can occur anywhere in the iris though most commonly occurs at the most ventral aspect (typical coloboma) from failure of closure of the optic fissure. Iris colobomas are coded as “breeder option” except for in the Australian shepherd, miniature American shepherd/minature Australian shepherd, and toy Australian shepherd where a “no” breeding recommendation is made due to their potential association with more serious abnormalities in other parts of the eye.¹ This has seemingly affected breeding practices and slightly decreased incidence of the condition in Australian shepherds presented for CAER exams: in 2013, 3742 were examined and 1.5% were affected; in 2022, 3582 were examined and 0.9% were affected. (J. Pautz, personal communication of breed data report, January 2023)
- Congenital partial thickness iridal defects are marked as iris hypoplasia on the CAER form and are considered “breeder option” for all breeds.
- Persistent pupillary membranes (PPMs) are remnants of the fetal anterior segment vascular system that have failed to regress by 3 months postnatally. They can occur unilaterally or bilaterally in a variety of forms: strands that originate from the surface of the iris and connect to the iris (iris-iris PPM), lens (iris-lens PPM) or cornea (iris-cornea PPM), as endothelial corneal opacities without strands, (Figure 8) or most severely as iridal sheets within the anterior chamber. PPMs that contact the lens frequently cause cataract and those that contact the cornea cause corneal opacity. The mildest form, iris-iris PPMs, are coded “breeder option”, but for all other forms a “no” breeding recommendation is made since they can cause vision impairment and or because of potential heritability and more severe forms in offspring. PPMs are found in multiple breeds but have been especially significant in the basenji, where they may also be associated with optic nerve colobomas,^{23–24} and the (English) mastiff and Pembroke Welsh corgi breeds.¹ The incidence of iris-to-iris PPMs in Pembroke Welsh corgis presented for CAER exams decreased from 17.2% (of 529 dogs examined) in 2013 to 14% affected (of 810 dogs examined) in 2022. The incidence of iris to lens PPMs in basenjis presented for CAER exams decreased from 7.7% (of 221 dogs examined) in 2013 to 1.1% affected (of 272 dogs examined) in 2022. The incidence of endothelial corneal opacities without strands from previous PPMs in mastiffs presented for CAER exams decreased from 3% (of 201 dogs examined) in 2013 to 1.9% affected (of 160 dogs examined) in 2022. (J. Pautz, personal communication of breed data report, January 2023)

Disorders of the lens can result in ocular discomfort and vision impairment via opacification, intraocular inflammation and secondary glaucoma. Many severe lens conditions result in a “no” breeding recommendation.

- Lens luxation is a potentially blinding disorder in dogs but fortunately one of the few anterior segment disorders that has a well-defined genetic etiology. An autosomal recessively inherited mutation in *ADAMTS17* causes the development of abnormal lens zonules which break prematurely.²⁵ As a result, the lens becomes loose and can move anteriorly or posteriorly out of position to increase susceptibility to glaucoma, corneal edema, uveitis and retinal detachment. This mutation is widespread across breeds but is especially prevalent in terriers and other dogs bred for short stature.^{26–27} This gene has also been associated with primary open angle glaucoma in a growing number of breeds, including the shar-pei, basset hound, petit basset griffon vendeen and basset fauve de Bretagne.^{27–30} Subtle signs of lens instability, such as a deep anterior chamber, vitreous in the anterior chamber and abnormal movement of the iris or lens with rapid eye movements (iridodonesis and phacodonesis, respectively) may be found on a CAER exam before more devastating disease occurs when the lens has become completely luxated. A “no” breeding recommendation is made for this condition. Commercial genetic testing is available.
- A cataract is an opacity of the lens and the most common presumed etiology of clinically significant cataracts in dogs is heredity. Hereditary cataracts can occur in any part of the lens and vary in size/extent. In general, a cataract that is suspected to be hereditary (without other reasonable, likely explanation such as developmental vascular contact, trauma, age, or previous inflammation, etc., typical in location, shape, etc.) on a CAER exam receives a “no” breeding recommendation. There are some exceptions to this such as posterior Y-suture tip opacities, which are not considered a true cataract, and nuclear fibrillar/pulverulent cataracts, which may be inherited and/or senile. These lesions both do not progress to cause vision impairment and are not associated with risk of more severe forms in offspring. Therefore, these cataract types are coded as “breeder option”, with posterior Y-suture tip opacities receiving their own specific code (E2). In GSD presented for CAER exams, the incidence of nuclear cataracts decreased from 6% in 2013 with 150 dogs examined to 4.3% in 2022 with 209 dogs examined. (J. Pautz, personal communication of breed data report, January 2023)

Mutations in the *HSF4* gene have been associated with cataract development as an autosomal co-dominant trait in the Australian shepherd, miniature American shepherd/miniature Australian shepherd, and toy Australian shepherd (*HSF4-2*) and an autosomal recessive trait in the Boston terrier, Staffordshire bull terrier, French bulldog and miniature pinscher (*HSF4-1*).^{1, 31–34} However, in most breeds the specific gene or genes involved in cataract formation remain unknown. In addition, other genes may be involved within known *HSF4* affected breeds.

Posterior polar cataracts, which are presumed hereditary in multiple breeds, are small, often triangular, and usually do not significantly impact vision in the animal in which they occur. (Figure 9) However, they may be associated with more severe forms of cataract in offspring and hence, a “no” breeding recommendation is made for this diagnosis on a CAER exam.³⁵

This has seemingly affected breeding practices and decreased incidence of the condition in dogs presented for CAER exams of several breeds: golden retrievers (4.1% (of 6458 dogs examined) in 2013 to 3.2% (of 9619 dogs examined) in 2022; cocker spaniels (4.3% (of 1129 dogs examined) in 2013 to 3.1% (of 1085 dogs examined) in 2022; Siberian huskies (4.9% (of 984 dogs examined) in 2013 to 3.8% (of 993 dogs examined) in 2022; and greater Swiss mountain dogs (5.2% (of 115 dogs examined) in 2013 to 1.9% (of 105 dogs examined) in 2022. (J. Pautz, personal communication of breed data report, January 2023)

Another form of cataract, in the equatorial cortex, may be inherited or not. Its incidence has decreased in greater Swiss mountain dogs (6.1% of 115 dogs examined to 2.9% of 105 dogs examined) and Boston terriers (2.8% of 533 dogs examined to 1.7% of 773 dogs examined) presented for CAER exams in 2013 and 2022 respectively. (J. Pautz, personal communication of breed data report, January 2023)

Cataracts can occur secondary to non-hereditary causes, such as trauma, aging, uveitis, nutritional deficiencies and specific metabolic diseases such as diabetes mellitus and hypocalcemia. For those cataracts that the examiner suspects not to be genetic in origin, “significance unknown/suspect not inherited” can be marked on the CAER form and those dogs may receive a breeder option code (E1).

The vitreous, retina, pigmented epithelium, choroid, sclera, and optic nerve comprise the posterior aspect/segment of the globe. These tissues mediate initiation of the visual pathway, transmission of visual information to the brain, recycling of visual pigments, and provision of nutrients/metabolite removal for the metabolically active photoreceptors. Inherited or presumed inherited disorders of these structures are common, and can be congenital (retinal dysplasias, collie eye anomaly, optic nerve hypoplasia or coloboma) or adult-onset (progressive retinal atrophy (PRA), multifocal retinopathy). While genetic tests for some conditions are available, there are many potentially inherited conditions for which no genetic diagnosis (and therefore test) exists. Hence, genetic testing cannot replace yearly hereditary eye disease screening, particularly for posterior segment conditions. For breeding dogs, a clinical diagnosis of a potentially inherited retinal disorder could be confirmed with genetic testing, but a ‘clear’ genetic test does not exclude an inherited component. Most inherited conditions of the canine posterior segment have an autosomal recessive mode of inheritance, although dominant conditions and recessive conditions with additional modifier genes have been described. The CAER exam system does not currently administer breeding recommendations based solely on genetic testing, and owners should seek expert advice from ophthalmologists and theriogenologists when making breeding decisions based on these outcomes. Genetic test results may help further inform or modify a phenotypic CAER exam diagnosis and breeding recommendations.

Vitreous conditions

- Liquefaction of the vitreous is common in aging, but earlier onset vitreous degeneration occurs in specific breeds. The genetics of this condition are not defined but it is presumed inherited in the Italian greyhound, Brussels griffon, shih tzu, whippet, and bichon frise, as well as other breeds, based on sufficient incidence in CAER data and references.^{1, 36} Though this theoretically

predisposes dogs to retinal detachment, glaucoma and lens luxation, in a study of these dogs diagnosed with vitreous degeneration on CAER exams, no such association with these ocular comorbidities nor cataract was found.³⁶ In Italian greyhounds, Brussels griffons and whippets, from 2013 to 2022, incidence of one form of vitreous degeneration (syneresis) in dogs presented for CAER exams decreased from 26% to 4.3% (in 192 and 94 Italian greyhounds examined), 20% to 14.3% (in 60 and 63 Brussels griffons examined), and 4.5% to 2.4% (in 355 and 594 whippets examined). (J. Pautz, personal communication of breed data report, January 2023)

- A group of congenital conditions relate to persistence of the fetal lens vasculature in the vitreous (persistent hyaloid artery (PHA), persistent hyperplastic primary vitreous or persistent hyperplastic tunica vasculosa lentis (PHPV/PHTVL)). These vessels can contribute to cataract formation, and/or vitreal hemorrhage if patent. PHPV/PHTVL is best documented as a presumed inherited disorder in Doberman pinschers and Staffordshire bull terriers, though the genetics of the disorder are not defined.^{1,37–44} CAER exams appear to have decreased the incidence of these conditions in these breeds in that the incidence of PHA in Staffordshire bull terriers presented for CAER exams decreased from 3.4% in 2013 (88 dogs examined) to 2% in 2022 (99 dogs examined), and the incidence of PHPV/PHTVL in Doberman pinschers presented for CAER exams decreased from 1.2% in 2013 (172 dogs examined) to 0.7% in 2022 (287 dogs examined). (J. Pautz, personal communication of breed data report, January 2023)

Congenital retina/choroid/optic nerve conditions

- Retinal dysplasias vary from mild (retinal folds manifesting as small multifocal areas of retinal thickening and/or thinning) (Figure 10), moderate (larger, geographic areas of retinal thickening and/or thinning) to severe (complete detachment of the retina with visual impairment). Although commonly diagnosed clinically in many breeds, mutations have been described in only two genes, *COL9A2* (Labrador retriever) and *COL9A3* (Samoyed and northern Inuit), which also cause an oculo-skeletal dysplasia.⁴⁵ Limb deformities include shortened forelimbs with valgus deformity and ocular abnormalities include cataract, multifocal retinal dysplasia, vitreous degeneration and retinal detachment. Diagnosis of the more severe forms of retinal dysplasia (geographic/complete) on a CAER exam will result in a “no” breeding recommendation. Retinal folds vary in their recommendation from “breeder option” to “no” based on the breed affected, and can be downgraded from a “no” to a “breeder option” for the three already mentioned breeds affected by *COL9A2* and *COL9A3* mutations if a negative genetic test can be provided.¹ The Labrador retriever provides an example of decreased incidence of retinal dysplasia – folds: 1.5% of 6741 dogs examined for CAER exams in 2013 were affected versus 0.8% of 6374 dogs in 2022. The English springer spaniel was similar, with a decreased incidence from 3.2% (of 1618 dogs examined for CAER exams) in 2013 to 2.2%

(of 1077 dogs) in 2022. (J. Pautz, personal communication of breed data report, January 2023)

- Collie eye anomaly is most prevalent in collies and related breeds. Phenotypes range from inadequate development of the choroid (choroidal hypoplasia) to defects of the choroid, sclera, and/or optic nerve (coloboma/staphyloma) to complete retinal detachment (with or without hemorrhage) that may be blinding. Mildly affected animals will have no detectable vision impairment. It is important to screen for this condition in puppies, as the development of pigmentation and the tapetum can sometimes mask milder versions of the condition (the “go normal” phenomenon), limiting clinical detection in adulthood. A mutation in the gene *NHEJ1* is known to be associated with choroidal hypoplasia, however the genes responsible for colobomas are unknown.⁴⁶ A clinical or genetic diagnosis of collie eye anomaly results in a “no” breeding recommendation.
- Optic nerve hypoplasia is a maldevelopment of the optic nerve that results in visual impairment. It is characterized by a very small optic nerve head and blindness or severely impaired vision and can occur unilaterally or bilaterally. Although inheritance is likely autosomal recessive, there may be incomplete penetrance, and the genetics of this condition have not been defined. Commonly affected breeds include the miniature poodle, Belgian tervuren, and shih tzu.¹ Micropapilla is a related condition where a small optic nerve head is observed clinically but without vision impairment. A diagnosis of optic nerve hypoplasia results in a “no” breeding recommendation, whereas a diagnosis of micropapilla results in a breeder option recommendation.

Adult-onset retina/choroid/optic nerve conditions

- Multifocal retinopathy is characterized by numerous distinct (i.e., multi-focal), roughly circular patches of elevated retina with accumulation of material that produces gray-tan-pink colored lesions (multifocal bullous retinal detachments). These lesions, looking somewhat like blisters, vary in location and size, although typically they are present in both eyes of the affected dog. Infrequently or chronically, this disorder can impair vision. There are several forms of canine multifocal retinopathy (CMR) from known mutations in the *BEST1* gene, with commercially available genetic tests. Breeding recommendations vary from “breeder option” to “no” based on breed, and commonly affected breeds include the bullmastiff, (English) mastiff, American bulldog, great Pyrenees, coton de Tulear, Finnish Lapphund, Lapponian herder, and Australian shepherd.^{1, 47–53} The miniature American shepherd/minature Australian shepherd, toy Australian shepherd, boerboel, Brazilian terrier, bulldog, cane corso, dogue de Bordeaux, perro de presa canario and other breeds may also be affected.^{48, 52–53}
- Progressive retinal atrophy (PRA) is a group of hereditary diseases that cause bilaterally symmetric, progressive degeneration of the retinal photoreceptors. More commonly, rod photoreceptors are affected by gene mutations, although cone disorders are described. Cone dysfunction and or degeneration can also be a

consequence of rod dysfunction and or degeneration. Dogs will typically present with night vision changes prior to onset of vision changes in daylight or bright light. Age of onset and rate of progression is highly variable, underpinning the necessity to perform CAER exams yearly. On the CAER form PRA is denoted as “retinal atrophy – generalized.” The most well-known genetic mutation associated with PRA is the autosomal recessively inherited *prcd* mutation, though over 20 different mutations have been discovered to cause this disease in a variety of breeds and many more are yet to be discovered.^{34, 53–54} A clinical diagnosis of PRA results in a “no” breeding recommendation. Because not all PRA causative genes are known, genetic testing may be used to complement but not circumvent a clinical diagnosis. The English cocker spaniel provides an example of decreased incidence of PRA in dogs presented for CAER exams: 2.6% of 116 dogs examined in 2013 versus 0.7% of 268 dogs in 2022. (J. Pautz, personal communication of breed data report, January 2023)

Conclusion

The incidence of comfort- and vision-threatening inherited canine ophthalmic conditions can be reduced with the use of screening examinations and certifications with associated breeding recommendations provided by collaboration between the ACVO and OFA in the form of CAER exams. The document *Ocular Disorders Presumed to be Inherited in Purebred Dogs* (known as “*The Blue Book*”) is the primary reference for these recommendations and can be viewed online.

Acknowledgments

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References

1. Genetics committee of the American College of Veterinary Ophthalmologists. Ocular disorders presumed to be inherited in purebred dogs, 13th ed., 2021, © American College of Veterinary Ophthalmologists.
2. American College of Veterinary Ophthalmologists. American College of Veterinary Ophthalmologists: Operational and committee policies and procedures, 2021, p.35–38.
3. Koll S, Reese S, Medugorac I, et al. The effect of repeated eye examinations and breeding advice on the prevalence and incidence of cataracts and progressive retinal atrophy in German dachshunds over a 13-year period. *Vet Ophthalmol* 2017; 20:114–122. [PubMed: 27073021]
4. Peiffer RL Jr, Gelatt KN, Gwin RM. Chronic superficial keratitis. *Vet Med Small Anim Clin* 1977; 72(1):35–37. [PubMed: 584092]
5. Jokinen P, Rusanen EM, Kennedy LJ, et al. MHC class II risk haplotype associated with canine chronic superficial keratitis in German Shepherd dogs. *Vet Immunol Immunopathol* 2011; 140(1–2):37–41. [PubMed: 21144596]
6. Chavkin MJ, Roberts SM, Salman MD, et al. Risk factors for development of chronic superficial keratitis in dogs. *J Am Vet Med Assoc* 1994; 204(10):1630–1634. [PubMed: 8050943]
7. Crispin SM, Barnett KC. Dystrophy, degeneration and infiltration of the canine cornea. *J Sm Anim Prac* 1983; 24(2): 63–83.

8. Cooley PL, Dice PF 2nd. Corneal dystrophy in the dog and cat. *Vet Clin North Am Small Anim Pract* 1990; 20(3):681–692. [PubMed: 2194353]
9. Waring GO 3rd. Inheritance of crystalline corneal dystrophy in Siberian Huskies. *J Am Anim Hosp Assoc* 1986; 22:655.
10. MacMillan AD, Waring GO 3rd, Spangler WL, et al. Crystalline corneal opacities in the Siberian Husky. *J Am Vet Med Assoc* 1979; 175:829–832. [PubMed: 528326]
11. Waring GO, Elkins MB, Spangler W. Oval lipid corneal opacities in beagles and crystalline lipid corneal opacities in Siberian Huskies. *Metab Pediatr Ophthalmol* 1979; 3:203–213.
12. Tetas Pont R, Downs L, Pettitt L, et al. A Carbohydrate Sulfotransferase-6 (CHST6) gene mutation is associated with Macular Corneal Dystrophy in Labrador Retrievers. *Vet Ophthalmol* 2016; 19(6):488–492. [PubMed: 26585178]
13. Busse C, Kafarnik C, Linn-Pearl R, et al. Phenotype of macular corneal dystrophy in Labrador Retrievers: A multicenter study [published correction appears in *Vet Ophthalmol*. 2020 May;23(3):592]. *Vet Ophthalmol* 2019; 22(3):294–304.
14. Maini S, Everson R, Dawson C, et al. Pigmentary keratitis in pugs in the United Kingdom: prevalence and associated features. *BMC Vet Res* 2019; 15(1):384. [PubMed: 31666065]
15. Labelle AL, Dresser CB, Hamor RE, et al. Characteristics of, prevalence of, and risk factors for corneal pigmentation (pigmentary keratopathy) in Pugs. *J Am Vet Med Assoc* 2013; 243(5):667–674. [PubMed: 23971846]
16. Townsend WM, Gornik KR. Prevalence of uveal cysts and pigmentary uveitis in Golden Retrievers in three Midwestern states. *J Am Vet Med Assoc* 2013; 243(9):1298–1301. [PubMed: 24134580]
17. Holly VL, Sandmeyer LS, Bauer BS, et al. Golden retriever cystic uveal disease: a longitudinal study of iridociliary cysts, pigmentary uveitis, and pigmentary/cystic glaucoma over a decade in western Canada. *Vet Ophthalmol* 2016; 19(3):237–244. [PubMed: 26119416]
18. Spiess BM, Bolliger JO, Guscetti F, et al. Multiple ciliary body cysts and secondary glaucoma in the Great Dane: a report of nine cases. *Vet Ophthalmol* 1998; 1(1):41–45. [PubMed: 11397208]
19. Pumphrey SA, Pizzirani S, Pirie CG, et al. Glaucoma associated with uveal cysts and goniodysgenesis in American Bulldogs: a case series. *Vet Ophthalmol* 2013; 16(5):377–385. [PubMed: 23110479]
20. Townsend WM, Huey JA, McCool E, et al. Golden retriever pigmentary uveitis: Challenges of diagnosis and treatment. *Vet Ophthalmol* 2020; 23(5):774–784. [PubMed: 32639654]
21. Sapienza JS, Simó FJ, Prades-Sapienza A. Golden Retriever uveitis: 75 cases (1994–1999). *Vet Ophthalmol* 2000; 3(4):241–246. [PubMed: 11397310]
22. Esson D, Armour M, Mundy P, et al. The histopathological and immunohistochemical characteristics of pigmentary and cystic glaucoma in the Golden Retriever. *Vet Ophthalmol* 2009; 12(6):361–368. [PubMed: 19883466]
23. Barnett KC, Knight GC. Persistent pupillary membrane and associated defects in the Basenji. *Vet Rec* 1969; 85(9):242–248. [PubMed: 4980462]
24. Roberts SR, Bistner SI. Persistent pupillary membrane in Basenji dogs. *J Am Vet Med Assoc* 1968; 153(5):533–542. [PubMed: 5691151]
25. Farias FH, Johnson GS, Taylor JF, et al. An ADAMTS17 splice donor site mutation in dogs with primary lens luxation. *Invest Ophthalmol Vis Sci* 2010; 51(9):4716–4721. [PubMed: 20375329]
26. Gould D, Pettitt L, McLaughlin B, et al. ADAMTS17 mutation associated with primary lens luxation is widespread among breeds. *Vet Ophthalmol* 2011; 14(6):378–384. [PubMed: 22050825]
27. Jeanes EC, Oliver JAC, Ricketts SL, et al. Glaucoma-causing *ADAMTS17* mutations are also reproducibly associated with height in two domestic dog breeds: selection for short stature may have contributed to increased prevalence of glaucoma. *Canine Genet Epidemiol*. 2019; 6:5. [PubMed: 31131111]
28. Oliver JAC, Rustidge S, Pettitt L, et al. Evaluation of ADAMTS17 in Chinese Shar-Pei with primary open-angle glaucoma, primary lens luxation, or both. *Am J Vet Res* 2018; 79(1):98–106. [PubMed: 29287154]
29. Oliver JA, Forman OP, Pettitt L, et al. Two Independent Mutations in ADAMTS17 Are Associated with Primary Open Angle Glaucoma in the Basset Hound and Basset Fauve de Bretagne Breeds

- of Dog [published correction appears in PLoS One. 2016;11(5):e0156192]. PLoS One 2015; 10(10):e0140436. [PubMed: 26474315]
30. Forman OP, Pettitt L, Komáromy AM, et al. A Novel Genome-Wide Association Study Approach Using Genotyping by Exome Sequencing Leads to the Identification of a Primary Open Angle Glaucoma Associated Inversion Disrupting ADAMTS17. PLoS One 2015; 10(12):e0143546. [PubMed: 26683476]
 31. Mellersh CS, McLaughlin B, Ahonen S, et al. Mutation in HSF4 is associated with hereditary cataract in the Australian Shepherd. Vet Ophthalmol 2009; 12(6):372–378 [PubMed: 19883468]
 32. Mellersh CS, Graves KT, McLaughlin B, et al. Mutation in HSF4 associated with early but not late-onset hereditary cataract in the Boston Terrier. J Hered 2007; 98(5):531–533. [PubMed: 17611257]
 33. Mellersh CS, Pettitt L, Forman OP, et al. Identification of mutations in HSF4 in dogs of three different breeds with hereditary cataracts. Vet Ophthalmol 2006; 9(5):369–378. [PubMed: 16939467]
 34. Donner J, Anderson H, Davison S, et al. Frequency and distribution of 152 genetic disease variants in over 100,000 mixed breed and purebred dogs [published correction appears in PLoS Genet. 2019 Jan 18;15(1):e1007938]. PLoS Genet. 2018; 14(4):e1007361. [PubMed: 29708978]
 35. Kraijer-Huwer IM, Gubbels EJ, Scholten J, et al. Characterization and prevalence of cataracts in Labrador Retrievers in The Netherlands. Am J Vet Res 2008; 69(10):1336–1340. [PubMed: 18828692]
 36. Krishnan H, Diehl K, Stefanovski D, et al. Vitreous degeneration and associated ocular abnormalities in the dog. Vet Ophthalmol 2020; 23(2):219–224. [PubMed: 31464365]
 37. van der Linde-Sipman JS, Stades FC, Wolff-Rouen-daal D. Persistent hyperplastic tunica vasculosa lentis and persistent hyperplastic primary vitreous in the Doberman Pinscher: Pathologic aspects. J Am Anim Hosp Assoc 1983; 19:791.
 38. Stades FC. Persistent hyperplastic tunica vasculosa lentis and persistent hyperplastic primary vitreous (PHTVL/PHPV) in ninety closely related Pinchers. J Am Anim Hosp Assoc 1980; 16:739.
 39. Stades FC. Persistent hyperplastic tunica vasculosa lentis and persistent hyperplastic primary vitreous in Doberman Pinschers: Genetic aspects. J Am Anim Hosp Assoc. 1983; 19:957.
 40. Stades FC, Boeve MH, van den Brom WE, et al. The incidence of PHTVL/PHPV in Dobermans and the results of breeding rules. Vet Quarterly 1991; 13:24.
 41. Anderson DE. The incidence of PHTVL/PHPV in Dobermans and the results of breeding. J Hered 1991; 82:21. [PubMed: 1997589]
 42. Boeve MH, Stades FC. Persistent hyperplastic tunica vasculosa lentis and primary vitreous (PHTVL/PHPV) in the dog: A comparative review. Prog Vet Comp Ophthalmol 1992; 2:163.
 43. Curtis R, Barnett KC, Leon A. Persistent hyperplastic primary vitreous in the Staffordshire Bull Terrier. Vet Rec 1984; 115:385. [PubMed: 6506414]
 44. Leon A, Curtis R, Barnett K. Hereditary persistent hyperplastic primary vitreous in the Staffordshire Bull Terrier. J Am Anim Hosp Assoc 1986; 22:765–774.
 45. Goldstein O, Guyon R, Kukekova A, et al. COL9A2 and COL9A3 mutations in canine autosomal recessive oculoskeletal dysplasia. Mamm Genome 2010; 21(7–8):398–408. [PubMed: 20686772]
 46. Parker HG, Kukekova AV, Akey DT, et al. Breed relationships facilitate fine-mapping studies: a 7.8-kb deletion cosegregates with Collie eye anomaly across multiple dog breeds. Genome Res 2007; 17(11):1562–1571. [PubMed: 17916641]
 47. Guziewicz KE, Zangerl B, Lindauer SJ, et al. Bestrophin gene mutations cause canine multifocal retinopathy: a novel animal model for best disease. Invest Ophthalmol Vis Sci 2007; 48(5):1959–1967. [PubMed: 17460247]
 48. Zangerl B, Wickström K, Slavik J, et al. Assessment of canine BEST1 variations identifies new mutations and establishes an independent bestrophinopathy model (cmr3). Mol Vis 2010; 16:2791–2804. [PubMed: 21197113]
 49. Grahn BH, Philibert H, Cullen CL, et al. Multifocal retinopathy of Great Pyrenees dogs. Vet Ophthalmol 1998; 1(4):211–221. [PubMed: 11397233]

50. Grahn BH, Cullen CL. Retinopathy of Great Pyrenees dogs: fluorescein angiography, light microscopy and transmitting and scanning electron microscopy. *Vet Ophthalmol* 2001; 4(3):191–199. [PubMed: 11722783]
51. Grahn BH, Sandmeyer LL, Breaux C. Retinopathy of Coton de Tulear dogs: clinical manifestations, electroretinographic, ultrasonographic, fluorescein and indocyanine green angiographic, and optical coherence tomographic findings. *Vet Ophthalmol* 2008; 11(4):242–249. [PubMed: 18638350]
52. Hoffmann I, Guziewicz KE, Zangerl B, et al. Canine multifocal retinopathy in the Australian Shepherd: a case report. *Vet Ophthalmol* 2012; 15 Suppl 2(0 2):134–138. [PubMed: 22432598]
53. Donner J, Kaukonen M, Anderson H, et al. Genetic Panel Screening of Nearly 100 Mutations Reveals New Insights into the Breed Distribution of Risk Variants for Canine Hereditary Disorders. *PLoS One* 2016; 11(8):e0161005. [PubMed: 27525650]
54. Online Mendelian Inheritance in Animals, OMIA. Sydney School of Veterinary Science. Available at: <https://omia.org/>. Accessed January 27, 2023.

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Key points:

The Orthopedic Foundation for Animals (OFA) Companion Animal Eye Registry (CAER) is operated to reduce the incidence of comfort- and vision-threatening inherited ophthalmic conditions.

A document written by the American College of Veterinary Ophthalmologists (ACVO) genetics committee called *Ocular Disorders Presumed to be Inherited in Purebred Dogs* guides certification and breeding recommendations of screening ophthalmic exam findings.

Serial screening ophthalmic examinations by Diplomates of the ACVO (DACVO), with breeding recommendations, have decreased the incidence of some inherited ophthalmic disorders in dogs.

Synopsis:

This manuscript describes the history and infrastructure associated with canine breed-related eye screening and certification by Diplomates of the American College of Veterinary Ophthalmologists (DACVO). Some of the common or otherwise particularly problematic specific inherited ophthalmic conditions are discussed.

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Orthopedic Foundation for Animals
2300 E Nifong Blvd, Columbia, MO 65201-3806
Phone: (573) 442-0418; Email: ofa@ofa.org
www.ofa.org. A not-for-profit organization

Call name: _____
Registered name: _____
Breed: _____ Sex: _____
ID Number (if any): ☐ Tattoo ☐ Microchip
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Date of Birth (mm/dd/yy): _____ Date of Exam (mm/dd/yy): _____
Owner Name: _____
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Owner Address: _____
City: _____ State: _____ Zip/postal code: _____
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I hereby certify that the animal examined is the animal described on this application, and understand that the results of this exam will be submitted by the examining ophthalmologist to the database for statistical gathering purposes. I understand that only passing results will be released to the public unless the initials of a registered owner or authorized agent appear in the authorization box below which permits the OFA to release non-passing results to the public.

Signature of owner or authorized agent/representative _____

I hereby authorize the OFA to release the results of the evaluation of the animal described on this application to the public if the results are non-passing (initials) _____

☐ I DID verify microchip/tattoo on this dog
☐ I DID NOT verify microchip/tattoo on this dog
☐ NO MICROCHIP/TATTOO PRESENT

I certify that I have performed this ophthalmic examination using pharmacological mydriasis, ophthalmoscopy, and biomicroscopy.

Signature _____ ACVO # _____ Date _____

Diplomate, American College of Veterinary Ophthalmologists

FEES AND CREDIT CARD INFORMATION ON THE BACK OF THE WHITE (OWNER) COPY

889200

Companion Animal Eye Registry (CAER)

RIGHT EYE GLOBE LEFT EYE

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NICTITANS
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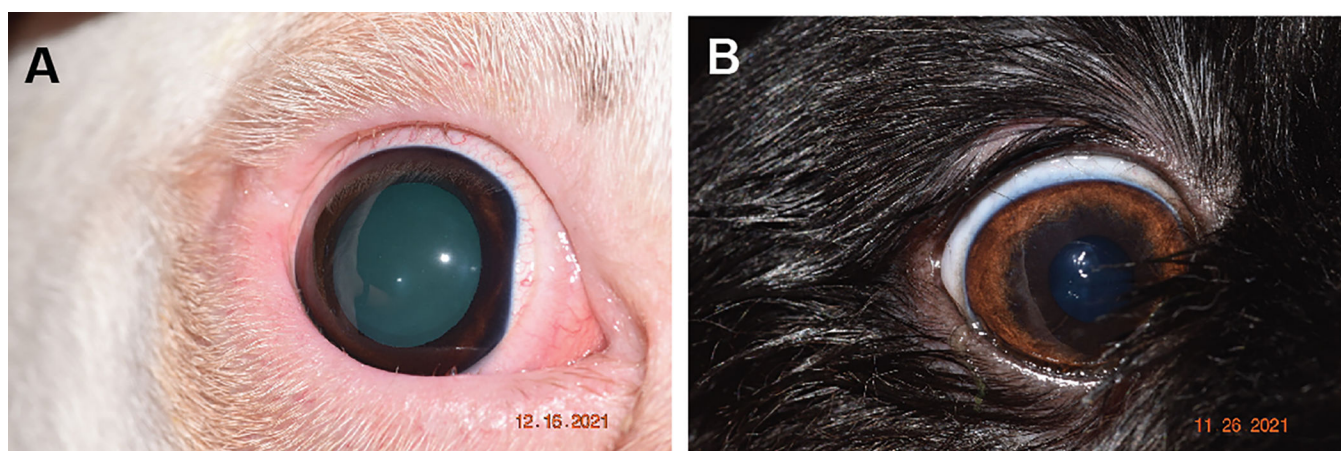


Figure 2:
Distichia in the right eye of a 2-year-old spayed-female boxer (A) and the right eye of a 1.5-year-old spayed-female shih tzu (B). These were bilateral in both cases.



Figure 3:

Pannus in the left eye of a 6-year-old spayed-female German shepherd (A), the right eye of a 4-year-old neutered-male greyhound (B); pannus and atypical pannus in the left eye of 5-year-old neutered-male German shepherd (C). These were all bilateral.



Figure 4:
Epithelial-stromal corneal dystrophy in the left eye of a 6-year-old neutered-male cavalier King Charles spaniel (A) and the right eye of a 4-year-old male Siberian husky (B). These were bilateral in both cases.



Figure 5:
Pigmentary keratitis/keratopathy in the left eye of a 4-year-old neutered-male shih tzu. Note the typical “wedge” of nasal corneal pigment, in this case without vascularization.



Figure 6:

Solitary, heavily pigmented, free-floating (in the anterior chamber) uveal cyst in the left eye of a 6-year-old neutered-male Boston terrier (A). Solitary, mildly-pigmented, free-floating uveal cyst, along with multiple, lightly pigmented uveal cysts “peeking” around the nasal pupillary margin from behind, in the right eye of a 5-year-old neutered-male golden retriever (B). Multiple, mildly pigmented uveal cysts at the nasal pupillary margin in the right eye of an 8-year-old spayed-female golden retriever with pigmentary uveitis and secondary glaucoma in the contralateral eye (C). Note the relatively heavily pigmented iris, and focal dense pigment on the superior-nasal anterior lens capsule, which is a remnant from a ruptured and deflated previous additional uveal cyst.



Figure 7:
Pigmentary uveitis in the right eye of a 9-year-old female-spayed golden retriever. Note the diffuse radial pigment deposition on the anterior lens capsule and early secondary cataract.

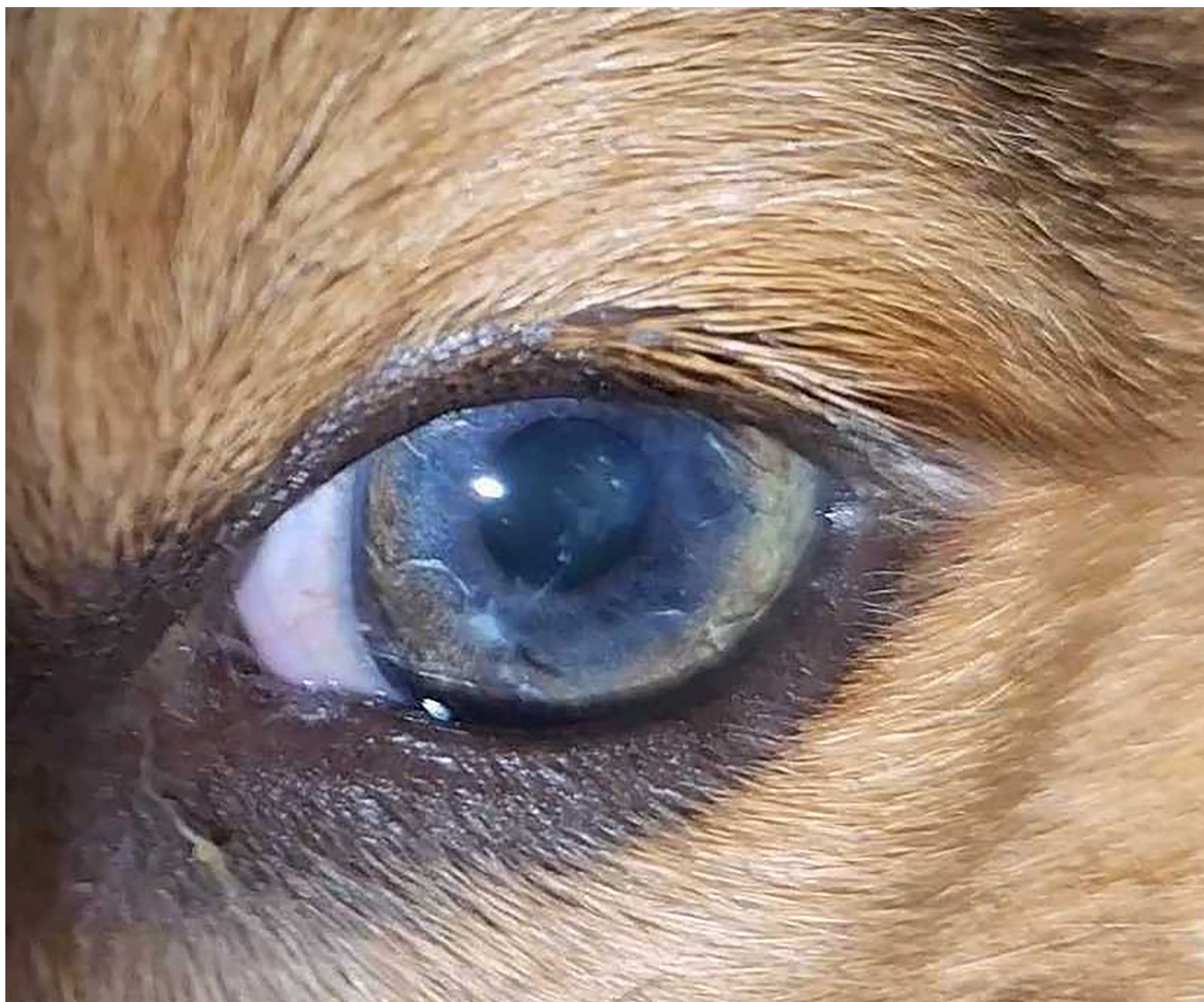


Figure 8: Iris-to-iris persistent pupillary membranes (PPM) and endothelial opacities no strands (remnants of iris-to-cornea PPM) in the left eye of a 4-month-old male corgi. The contralateral eye was equivocally microphthalmic and had iris-to-cornea PPM with associated corneal endothelial leukoma and overlying corneal edema from focal endothelial dysfunction in the region of PPM adherence.



Figure 9:
Posterior polar cataract in the left eye of a 3-year-old female golden retriever presented for CAER examination. This was bilateral.

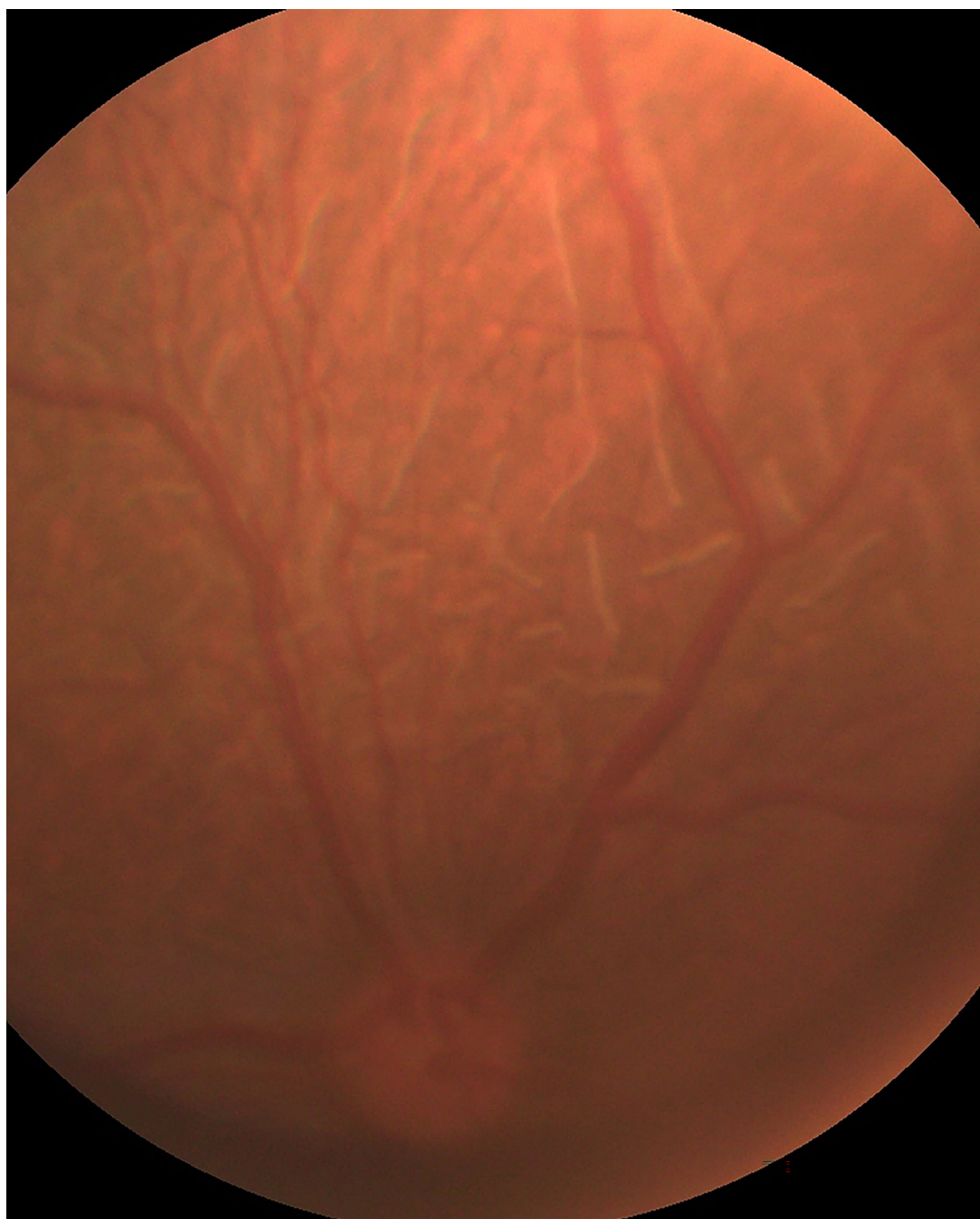


Figure 10: Extensive retinal folds in the right eye of an 8-week-old English springer spaniel presented for CAER litter screening examination. Multiple puppies in the litter were affected with retinal folds or geographic retinal dysplasia in one or both eyes.

Table 1.Unequivocal “no” breeding recommendation conditions¹

Keratoconjunctivitis sicca (KCS or dry eye)	Glaucoma
Certain forms of persistent pupillary membranes (PPM) and associated findings (iris to iris and iris to cornea PPM, iris sheets and endothelial opacity/no strands)	Cataracts without an obvious, reasonable, non-inherited explanation
Lens subluxation or luxation	Persistent hyperplastic primary vitreous (PHPV)/persistent hyperplastic tunica vasculosa lentis (PHTVL)
Retinal detachment	Generalized or diffuse retinal atrophy/retinal degeneration (e.g., the inherited progressive retinal atrophies (PRA))
Geographic or detached retinal dysplasia	Optic nerve coloboma or hypoplasia