Lens disorders in dogs are generally associated with loss of clarity or altered lens position within the globe. The lens is an anatomically simple spheroid structure, comprising protein encased within a capsule; understanding lens embryology and anatomy aids diagnosis of lens disorders. Both cataract and lens luxation are commonly hereditary disorders and lens examination techniques are explained for diagnosis and monitoring.

**Key words:** lens, canine, cataract, luxation, subluxation, ocular embryology, ocular examination

### Introduction

The lens is a unique biological structure both in function and form. It is avascular and of higher protein content than any other tissue of the body. In the dog, this biconvex spheroid measures 7mm (anterior to posterior) by 10mm (equatorial diameter) and volume is around 0.5ml. The structure and form is solely orientated towards an optical function, allowing light to be transferred, refracted and filtered. Although the cornea provides the greatest degree of refraction, it is the lens which does the fine-tuning, further refracting the incoming light to a focal point on the retina. Correct alignment of the lens within the globe and relative to the retina is essential for accurate focus. Lens disorders typically are associated with impaired clarity or altered position and much of the article therefore concerns cataracts and luxation. Initially however, it helps to understand the embryology and anatomy of the lens, so this is covered in the first part of the article, as well as congenital and developmental disorders. Cataracts occur most commonly in dogs as a primary, hereditary disorder or secondary to diabetes mellitus; these are discussed in the second part of the article as well as lens luxation and examination of the lens. Cataracts associated with trauma and secondary to other ocular disease, including uveitis, are covered in the following article in the series, which is focussed on lens disorders in cats.

#### Lens Embryology

The lens is derived embryologically from surface ectoderm, when a focal region comes in to contact with the underlying optic vesicle as it extends from the developing forebrain, derived from neural ectoderm. This lens placode thickens (day 17 of gestation in the dog) and invaginates, passing inside the optic vesicle whilst this also invaginates to form a bilayered optic cup. This contact, as well as correct alignment, ensures appropriate induction of the surface ectoderm, ensuring the correct size and location of the lens vesicle and therefore the later lens (see Figure 1).

**Figure 1:** Embryology of the lens in the dog

1a: The optic vesicle is an out-budding of neural ectoderm which approaches the surface ectoderm, inducing thickening of cells to form a lens placode (day 17)

1b: Invagination of the lens placode occurs within the developing optic cup

1c: The lens vesicle detaches, forming a hollow ball of lens epithelial cells (day 25)
When the lens vesicle detaches from the surface ectoderm (at day 25 of gestation), this is the first step in the formation of the chambers of the anterior segment of the globe. The lens vesicle is comprised of a monolayer of cuboidal epithelial cells, surrounded by a basal lamina. Once detached, the posteriorly placed cells elongate, in response to the adjacent inner aspect of the optic cup (future neural retina). These elongating cells obliterate the lumen and become the primary lens fibres: the embryonic lens nucleus (Figure 2). A layer of cuboidal cells remains anteriorly, and these cells maintain mitotic activity throughout life, differentiating at the lens equator into new lens cells (the lens cortex).

Newly formed lens cells at the lens equator elongate into slender fibres, stretching anteriorly and posteriorly and wrapping around the nucleus to meet the opposing, developing lens cells at a region called the lens sutures. The sutures form a Y-shape anteriorly and an inverted-Y posteriorly, which can be observed in the adult lens with careful observation and slit-lamp biomicroscopy. During this transformation the cells also lose nuclei and organelles – essential for optical transparency: The cell shape becomes hexagonal in cross-section, allowing close packing and a high level of inter-cellular contact, and along the length of the cells, an array of cytoplasmic interdigitations and nexi form. These transformations allow the lens to effectively function as a large lens, as opposed to being biochemically and electrically isolated.

The content of the lens cells is primarily protein, with structural, refractive proteins being predominantly soluble (called crystallins), whilst the membranes are comprised of insoluble proteins (albuminoids). As layer upon layer of fibres accumulate throughout life within the confines of the lens capsule (basal lamina), the inner, nuclear, fibres become increasingly closely packed and the balance of crystallins to albuminoids shifts towards the latter. This process continues throughout life and manifests as a nucleus of altered refraction compared to the foetal nucleus of the lens. Impaction under additional layers of normal lens fibres results in the opacity becoming smaller relative to total lens volume as the dog ages. Merle ocular dysgenesis, associated with the merle gene in Australian Shepherds and other breeds, results in defects including microphthalmos, microcornea and colobomas of the uvea or retina as well as cataract.

Colobomas of the lens are in fact not truly a coloboma (gap or hole) but a defect seen at the equator, appearing as a notch in the lens. These are likely due to a focal zonule defect, resulting in reduced tension in this region. As with other ocular colobomata, they are termed ‘typical’ when located at the six-o’clock position, where they are associated with defective closure of the optic cup and ‘atypical’ if elsewhere around the equator. Rarely are they large enough to cause substantial loss of lens stability or a notable impact on vision.

Vascular anomalies comprise some of the most commonly observed congenital ocular conditions which, although not directly leucocytic, do often impact upon the lens or lens capsule. Persistent remnants of the embryonic vasculature are observed anteriorly in the form of persistent pupillary membrane remnants. These strands of pigmented tissue may be confused with post-inflamatory synechiae, however they arise from the collarette (mid-portion) of the iris. These may insert upon the lens capsule and be associated with a focal cataract (Figure 4). In others, mere punctate or cruciate foci of pigment are noted scattered uniformly upon the axial lens capsule, distinguished from post-inflamatory scarring by the uniform colour, patterning and axial location (Figure 5).

Posteriorly, hyaloid remnants may take the form of ‘island’ of pigment and fibres on the posterior capsule and be clinically insignificant or, at the other extreme, form fibro-vascular opacities, sometimes complete with a patent hyaloid artery. Hyperplasia of these vascular remnants can lead to large, posterior capsular plaques which can block the pupil. This is referred to as persistent hyaloid primary vitreous (PHPV) or persistent hyperplastic tunica vasculosa lentis (PHVTL) and is a recognised hereditary disorder of the Dobermann Pinscher, German Pinscher and Staffordshire Bull Terrier. Secondary cataract is common due to the disruption of the lens capsule, sometimes with uveal tract haemorrhage. Surgery is considered when there is an impact on vision and involves phacoemulsification cataract surgery, posterior capsulotomy and, in some cases, vitreotectomy.

Careful pre-operative assessment includes the use of ocular ultrasound with colour-blood Doppler or microbubbles and contrast techniques. The most common congenital shape deformity is posterior lenticonus, a conical protrusion of the posterior lens. This typically occurs with other congenital abnormalities such as cataract, persistent hyaloid artery or PHPV/PHVTL (as in the Dobermann) or microphthalmos.

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