Health Booklet
For
Soft-Coated Wheaten Terriers

(*An advisory booklet for all Wheaten owners)*

Issue 2 – February 2021

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Introduction

The Soft-Coated Wheaten Terrier (SCWT) is essentially a healthy, hardy dog with an average life span of 13-15 years. However, like most breeds, there are some hereditary diseases which can affect the breed.

This Health Booklet is a brief overview of four diseases, which can affect the SCWT.

Health Testing

Key veterinary researchers recommend that you have an annual health screen on your Soft-Coated Wheaten Terrier. This gives a snap-shot for you and your veterinarian on the general health of your Wheaten, but more specifically it can indicate if your Wheaten has any evidence of the hereditary diseases: renal dysplasia (**RD**), protein losing nephropathy (**PLN**), protein losing enteropathy (**PLE**) and canine degenerative myelopathy (**DM)**, all of which can occasionally affect the breed.

Quick Definitions:

- **RD** – is the abnormal development of the kidney in utero. This malformation can result in early renal failure.

- **PLN & PLE -** are syndromes characterised by the loss of protein. **PLN** is the loss of protein from the kidneys and **PLE** is the loss of protein from the gastrointestinal tract.

- **DM** – is a progressive loss of coordination (ataxia) in the back legs. The disease is chronic and progressive, resulting in paralysis.

Clinical Signs

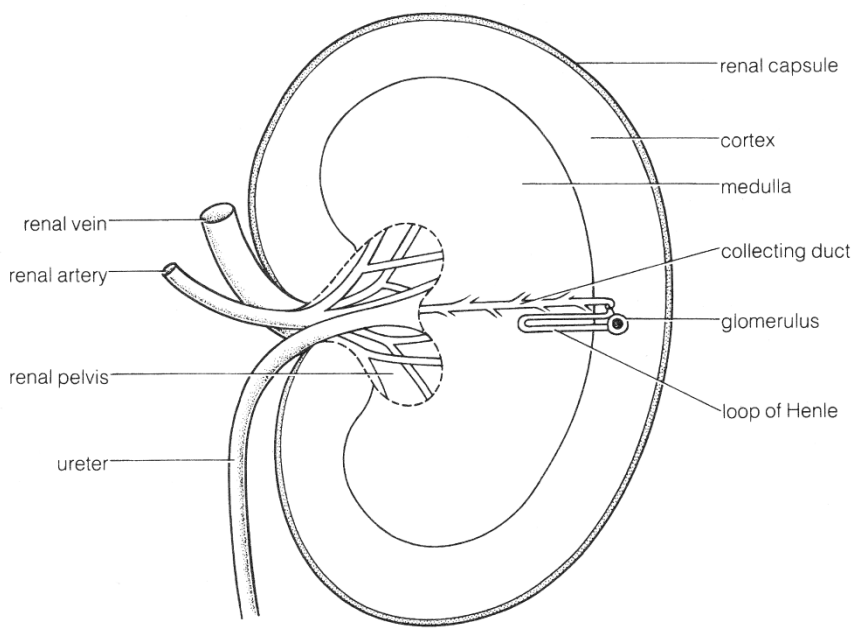
Clinical signs of a disease are the things you can see or that your vet may discover on his/her physical examination of your Wheaten.

Testing is important

In many diseases, clinical signs do not show up until well after tests show signs of the disease. Also, many diseases have very similar signs.

Physiology of the kidney

The kidney is involved in two of the hereditary diseases**: RD & PLN**



Microscopic glomerulus, loop of Henle and collecting duct have been magnified out of proportion for clarity

Kidney anatomy and function

The kidney has a very rich blood supply. This enters via the renal artery and passes through a complex network of arteries, capillaries and veins before leaving through the renal vein. During its passage through this system, the blood is cleansed of the toxic by-products of protein metabolism.

Filtration takes place in minute tufts of blood vessels. Each tuft is called a glomerulus and together with its associated tubules and blood vessels forms the nephron. Normally large molecules such as blood proteins and the red and white blood cells, all essential for health, remain in the blood while small molecules not required for body function (e.g. urea, creatinine) are excreted into the urine. Some small molecules which have passed through the filters are reabsorbed into the blood as they are necessary for a healthy chemical balance in the body (e.g. glucose, salt etc.).

Kidney failure and disease

There are a number of different disease processes which can damage the glomeruli thereby causing kidney failure. Glomerulonephritis (the inflammation of kidney tissue) and glomerulosclerosis (scarring or hardening of the blood vessels in the kidney) are broad terms for many forms of this damage.

Ingestion of chemicals such as antifreeze, some foods, plants etc., can cause kidney damage.

Sometimes kidney failure is caused by a defect in the filtration mechanism itself allowing large protein molecules such as albumin to remain in the blood. This is typical of protein losing nephropathy.

Occasionally the kidney doesn't develop properly before birth with the result that the kidneys never work efficiently. This varies from individual to individual with some animals badly affected living for a few months while others may live for many years. This is what occurs with renal dysplasia.

Kidney failure may be 'silent' for many years as approximately 70% of the kidney can be damaged before any physical signs show themselves.

The effects of some forms of kidney disease can be slowed down with treatment and diet, but scarred glomeruli can never be repaired. This is what makes early detection so vital. The treatment given in the early stages of kidney failure depends on the disease causing the damage.

Renal Dysplasia (RD)

History

In the 1960s a number of Wheatens in Great Britain died from renal failure. These dogs ranged in age from very young puppies to approximately 8 years.

In the 1970s reports came from Scandinavia and Holland of Wheatens dying from kidney disease. A study of these affected dogs led to the diagnosis of hereditary renal cortical hypoplasia (another name for RD). All those affected at that time had pedigrees containing English lines.

In 1978, Dr D F Kelly, pathologist at Liverpool Veterinary College, diagnosed a young British dog as having inherited kidney disease. He was asked by the SCWT Club to investigate further. Other affected litters were later identified, confirming that indeed, inherited kidney disease existed among UK Wheatens.

In December 1983, the SCWT Club committee approved a health directive stating that no dog or bitch which has produced any offspring diagnosed as being affected by RD should be bred from further.

Professor Andrew Nash, at the University of Glasgow Veterinary School, treated several affected Wheatens from a breeder near Glasgow. His assistance was therefore sought and in 1984, a litter monitoring scheme was set up with the help of funds from the Clinical Studies Trust Fund and the SCWT Club of GB.

Around the same time, the Club asked Dr Bruce Cattanach, Boxer breeder and geneticist at the Medical Research Council Radiobiological Unit, Harwell, for his help to establish the mode of inheritance of this kidney disease and to help the Club set up a testing scheme to control its spread. All this was done with great success and the number of puppies dying reduced dramatically.

In 1986, Club members agreed at an AGM that the Club should adopt a policy of publishing all results of hereditary defect tests following relevant experts' confirmation.

General

Renal dysplasia in the SCWT is recognised as a familial, inherited disease and the mode of inheritance is thought to be via a recessive gene. However, it should be noted that there is no clear agreement on the mode of inheritance. There are other kidney diseases, including RD, that are caused by other external factors, rather than by genetic inheritance. A diagnosis of RD can only be confirmed following a post-mortem examination of the deceased dog's kidneys or by analysing a wedge biopsy taken from a living dog. There is currently no genetic/DNA test for RD.

The kidneys appear abnormally small when seen on ultrasound examination.

The symptoms of RD are similar to other forms of kidney disease as well as other diseases and are extremely variable. Affected dogs can exhibit any combination of the following:

Renal Dysplasia (RD)	Symptoms	Laboratory abnormalities often associated with this disease
Renal dysplasia is a congenital or neonatal disease which causes maldevelopment of the kidneys in utero, or early in life	Increased water consumption Increased urination (dilute urine) "Poor doer" Decreased appetite Vomiting Possibly prone to urinary infections	Low urine specific gravity (USG) Elevated creatinine and urea (BUN) Small kidneys Small hyperechoic kidneys with or without cysts seen via abdominal ultrasound

Treatment

Most forms of kidney disease are progressive but with proper treatment and dietary support many animals can lead relatively normal although shorter lives. If the disease is detected before serious damage to the kidneys has occurred, a change of diet to a lower protein food may well be all that is necessary. Veterinary advice is essential since many specially formulated kidney diets are available.

Fresh, clean water should be available at all times and the dog should be protected from undue stresses such as extremes of temperature, over-excitement, excessive exertion, etc.

Any course of medication prescribed by the veterinary surgeon should be followed carefully even though the dog appears better. Obviously, any changes or problems should be quickly reported to the vet.

Recommendations for testing

As there are still occasional cases of RD being reported, it is likely that the gene is still present in the gene pool and it is **imperative** that we remain vigilant.

1. As signs of RD do not always become evident before breeding age **it is strongly advised** that all potential breeding stock should be blood and urine tested *prior to mating.*

2. Breeders should ensure puppies are **blood tested** at 7-8 weeks of age before going to their new homes. If a test result is not normal that puppy should be 'run on' and retested in another 7-10 days. It is probable that the test result will then be normal. If it is not, then the puppy should not be sold.

3. Please increase awareness of the RD control scheme. When you give each new owner their Puppy Handbook, ensure this booklet is enclosed.

Controlling RD

The main objective of the Club scheme is to prevent further cases of RD occurring and to try to reduce the number of carriers in the breed. This should be done in a manner which does not limit the gene pool unnecessarily or entirely eliminate the use of valuable breeding stock.

Another equally important aim is to identify affected animals, to help these dogs achieve a reasonable lifestyle and life span with treatment and diet.

It must be remembered that **the <u>only</u> way to confirm RD as the cause of death of a Wheaten is by post mortem examination of the kidney.**

Breeding recommendations:

1. Parents of Wheatens diagnosed with RD or having died and RD confirmed by post mortem should be withdrawn from further breeding. As proven carriers, they represent known identified sources of RD in the breed.

2. The following dogs have an unacceptably high risk of being carriers and should be withdrawn from breeding:
   a. Brothers and sisters of proven carriers – 50% risk
   b. Progeny from any mating with proven carrier – 50% risk
   c. Brothers and sisters of Wheatens with RD, whether from the same, or separate litters

3. Wheatens with a carrier in the second to fifth generations of their pedigree still have a risk of being carriers. With two or more carriers in their pedigrees, the risks increase greatly. Therefore, especially with the higher risk animals, it is recommended that they should only be mated with Wheatens that do not have any known carriers in their **five generation** pedigrees.

4. Stud dog owners have equal responsibility and should check the bitch's pedigree carefully to assess the risks of RD before accepting her for mating.

If you are unsure of your dog's health ancestry please contact your breeder or the Club's health team who can advise you of any carriers in your dog's pedigree.

A free publication: 'Renal Dysplasia (RD) in the Soft Coated Wheaten Terriers. An advisory booklet for all Wheaten Owners' is available from the SCWT Club Health team if you or your veterinary surgeon would like further information.

Protein Losing Nephropathy (PLN)

Protein Losing Nephropathy (PLN)	Symptoms	Laboratory abnormalities often associated with this disease
PLN is difficult to diagnose. The initial stages of the disease may be mistaken for liver, glandular or other enteric or kidney diseases. Wheatens with PLN may have serious thromboembolic events before renal failure starts, even before there is increased serum creatinine or BUN. An abnormality of the glomeruli usually causes PLN.	Listlessness/depression Decreased appetite, vomiting, weight loss Ascites, oedema, pleural effusion Increased water consumption, Increased urination (less common) Thromboembolic phenomena and hypertension (less common)	*Note that not all of the laboratory abnormalities are seen in every case. The most important are indicated by an asterisk*. Hypoalbuminemia* Elevated serum creatinine, BUN (later) Hypercholesterolemia Elevated MA (Microalbuminuria) Elevated urine protein/creatinine ratio*

PLN can be a late onset disease and the damage to the kidney may not show until the end stages of the disease. It is a condition in which large plasma proteins are lost from the blood into the urine.

Glomerulonephritis is the general term for PLN syndrome. This can be any of a group of infectious, inflammatory or cancerous diseases causing a reaction from the body's immune system and the formation of immune complexes which affect the most important components of renal tissue, glomeruli and adjacent structures.

Wheatens affected with PLN have a defect in one of the components of the glomeruli filtration mechanism in the basement membrane. This defect is a slit or hole big enough to allow large molecules such as blood proteins (i.e. albumin) to escape into the urine.
Urinalysis is very important as protein loss can be detected via urine, in some cases years before any changes to blood results occur.

Mode of Inheritance

It was noted in North America that there were an unusually high number of Wheatens with this disease. After years of research, Dr Meryl Littman and Dr Paula Henthorn identified mutations associated with PLN in Wheatens and Airedale Terriers. As a result, there is now a DNA test using a non-invasive cheek swab which any owner can use and submit to a testing laboratory.

Any dog, of any breed can develop PLN due to any number of causes. The mode of inheritance of this disease in the Wheaten is only partially understood. The presence or absence of the predisposing gene does not determine whether the dog ***will*** develop PLN but rather determines the ***risk*** that the dog has of developing the disease.

PLN - DNA Testing

The SCWT Club of GB has a specifically lettered set of PLN DNA test application forms from Laboklin Laboratories, Manchester. The unique forms should eventually provide statistical information of the number of Wheatens with two normal genes or one or two copies of the predisposing gene. These forms and swab kits are available from the Health Team.
The non-invasive cheek swab test is also available from the University of Pennsylvania School of Veterinary Medicine: http://www.scwtca.org/health/dnatest.htm

PLN-Associated Variant Genes Test Result Definitions -This table clarifies the reporting formats between PennGen and Laboklin.

Genetic Term	Definition	What does this mean?	Other Common Terms	Test Results from PennGen	Test Results from Laboklin
Homozygous Negative	A dog **without any** of the variant alleles	A dog that has no copies of the variant alleles is at the least risk of developing PLN	• 0 • 0/0 • No copies • "Normal • "Clear" • Homozygous	1/1	N/N (Clear)
Heterozygote	A dog **with one copy** of the variant alleles	A dog with one copy of the variant allele is at medium risk of developing PLN	• 1 • 0/1 • "Carrier" • 1 Copy • Heterozygous	1/2	N/PLN (Carrier)
Homozygous Positive	A dog with **two copies** of the variant alleles	A dog with two copies of the variant alleles is at the highest risk of developing PLN, but this does not mean it will develop PLN	• 2 • Both copies • Homozygous for the PLN causative mutation	2/2	PLN/PLN (Affected) Affected refers to both copies of the allele, **it does not mean this dog is currently or will be affected with PLN**

A simple way to monitor your dog's health is to have a urine dipstick test when your dog has its annual booster/health check-up.

Treatment

If caught early, dogs with PLN can have a reasonably normal life with moderate exercise and play to give increased longevity.

The treatment for PLN consists of:

- Diet
- Medication
- Stress free lifestyle

Further information:

'Recommendations Concerning Protein-Losing Nephropathy (PLN) in Soft Coated Wheaten Terriers', Dr Meryl Littman, Professor Emerita of Medicine (Clinician-Educator) 4 August 2016 Available to download: http://bit.ly/2oR125C

"Protein Losing Nephropathy and your Wheaten" and "Protein Losing Nephropathy and your Wheaten – A letter for your veterinarian" *available from the SCWT Club of GB Health Team.*

RD and PLN can easily be mistaken for each other. The following Chart explains these subtle differences between the two diseases.

Differences between RD and PLN

Renal Dysplasia (RD)		**Protein Losing Nephropathy (PLN)**
Dogs *generally* die between the ages of 6 weeks to 3 years.		Dogs tend to show their illness at 5-7 years old, but onset can be both earlier and later than this.
Dogs tend to lose little protein in the urine and the serum albumin stays normal.		Dogs lose large quantities of protein in the urine and their serum albumin drops. They also have a high protein/creatinine ratio.
Dogs eventually have high serum creatinine and Urea (BUN) Dogs **do not** have low albumin and high cholesterol.		Dogs eventually have high serum creatinine and urea (BUN). Dogs have low albumin readings and high cholesterol.
Dogs are born with small, malformed kidneys.		Dogs usually have normal sized kidneys until later stages of the disease.
In the renal cortex are microscopic cystic lesions, decreased and immature foetal glomeruli and cystic glomeruli.		Dogs show glomeruli changes, such as glomerulonephritis and/or glomerulosclerosis. They do not have many foetal glomeruli.
Dogs are not usually predisposed to effusions and thromboembolism (clots).		Dogs can throw clots, in the lung, heart, brain, portal vein or distal aorta (saddle).

Protein Losing Enteropathy (PLE)

Digestive System

Your dog's body produces a number of extremely important proteins, called enzymes. One group of these are the digestive enzymes that participate in the breakdown and digestion of food.

In humans, digestion begins in the mouth where saliva contains digestive enzymes. Dogs, however, don't chew their food they gulp it down in chunks. Dog saliva serves in digestion only to moisten and lubricate the mouth and food as it is pushed back into the oesophagus for its journey to the stomach.

The gastrointestinal tract has to perform many functions in order to absorb food then excrete the waste products. The mucosal layer lines the inner surface of the tube and is responsible for secretion and absorption of nutrients to the body. The surface area of the mucosa contains villi. Damage to the villi can cause villous atrophy which leads to malabsorption and diarrhoea.

The major digestive and absorption processes occur in the small intestine. Several digestive enzymes are mixed into the food along with bile. These secretions are used to breakdown carbohydrates, proteins and fats into smaller molecules and proteins into amino acids. The amino acids are absorbed and re-circulated to be made back into proteins while the food mixture is churned and pushed along by intestinal muscle contractions. A dog's intestines are relatively short (about five times their body length) so complex foods have a short time to be broken down and absorbed.

By the time this mixture reaches the large intestine, the final leg of its journey, there should be little of nutritional value left. The large intestine completes the absorption of water and electrolytes and any remaining undigested food is then filtered and stored for elimination in the colon.

When a dog suffers from malabsorption, as in the case of PLE, digestive enzymes fail to absorb protein into the body and it is, therefore, passed through the large intestine into the faeces. A forerunner to PLE can be irritable bowel disease (IBD).

Protein Losing Enteropathy (PLE)	Symptoms	Laboratory abnormalities often associated with this disease
PLE is usually caused by inflammatory bowel disease (IBD) or lymphangitis /lymphangiectasia. In affected Wheatens there is a stimulation of the immune system in the bowel wall	Vomiting Diarrhoea Lethargy Weight Loss Ascites Oedema Pleural effusion	Note that not all of the laboratory abnormalities are seen in every case. The most important are indicated by an asterisk. Hypoalbuminemia* Hypoglobulinemia* Hypocholesterolaemia Eosinophilia Lymphopenia

Protein Losing Enteropathy

- PLE is a condition in which protein is lost excessively into the intestine and can represent a number of abnormalities, which result in the loss of plasma proteins from the gastrointestinal tract.
- The loss of the healthy mucosal layer allows the leakage of vital protein-rich fluids. This is a hallmark of PLE.
- The liver and other cleansing systems are unable to compensate for the loss.
- Mechanisms for gastrointestinal protein loss include lymphatic obstruction, mucosal disease with erosions, or ulcerations.
- PLE is probably related to immunological defence of the intestinal tract.
- This can be a late onset disease, which means that the dog develops it in maturity.
- Tests necessary to detect the presence of PLE are blood, urine and, if necessary, endoscope biopsy and faecal investigation.
- The mode of inheritance **for PLE** is not known.
- At the present time, there is no genetic test available to know if a dog is carrying or affected by this disease.

Note: PLE & **PLN** are difficult to diagnose. The initial stages of the disease may be mistaken for liver, glandular or other enteric or kidney diseases. Wheatens with PLE and/or PLN may have serious thromboembolic events (such as pulmonary embolism) before symptoms or renal failure start, even before there is increased serum creatinine or BUN.

Canine Degenerative Myelopathy (DM)

Failing Back Legs

Degenerative Myelopathy (DM) is a non-painful degenerative disease of the spinal cord which typically affects dogs over 8 years of age. Initial signs are often slight, such as muscle weakness or trembling in a hind leg or wear on the nails of one or both hind feet. This is followed by progressive deterioration in the dog's ability to coordinate the use of its back legs, leading to eventual paralysis.

Weakness and atrophy in the muscles often leads to faecal and urinary incontinence**:** the dog cannot stand unaided to go outside and loss of sensation impairs the brain's ability to receive the messages that the dog 'needs to go'.

The disease will eventually progress and affect the front legs although this can take several years.

There are any number of conditions which cause loss of co-ordination or weakness in the back legs; arthritis, slipped disks, tumours, cysts, infections, stroke, or as a result of injury. Before a diagnosis of DM can be made, each of these conditions will need to be excluded.

DM is thought to be caused by the dog's own immune system attacking the myelin nerve sheath, which surrounds and protects the nerve fibers of the spinal cord. Destruction of the sheath leads to progressive nerve tissue damage affecting voluntary and involuntary motor control and interrupting receipt of messages from the brain.

S**ymptom**s *(progress slowly over time)*

- Hind end weakness (failing back legs), tremor in rear legs.
- Weakness may initially occur in one hind limb, but both will become affected.
- Worn nails, one or both rear feet dragging, knuckling' over of paws.
- Wobbling when walking (drunken sailor effect), falling over when changing direction.
- Difficulty rising, walking up steps, getting in the car, squatting to defecate.
- Urinary & faecal incontinence
- Weakness in front legs leading to complete paralysis.
- A dog does not suffer pain with DM.

Treatment:

No specific treatment is available at this time although there are a number of ways a dog can be supported to help maintain an acceptable quality of life for months to years, and there are steps the owner can take to improve the quality of life for the dog.

Good nursing care and some physical rehabilitation, such as gentle exercise, can help. Hydrotherapy may be more useful as the dog's weight is supported by the water. Belly slings are available which help get the dog to the garden for the necessary "comfort breaks'. There are harnesses and special carts to increase mobility, although not all dogs get the message that these are a way to get from A to B. Introducing a cart for short periods at an early stage seems to be more successful than waiting until it is necessary.

Testing

DM was once thought to be a German Shepherd Dog disease but it is now known to affect over 30 breeds including the Wheaten Terrier.

DM in dogs is associated with a genetic abnormality and there is now a test available for an inheritable gene at least partly responsible for its development. This is a single genetic marker showing a 'risk' of developing the disease**:** no markers are least risk, one marker medium risk, and two markers indicate a greater risk. However, some dogs that have one or two of the genetic markers may never develop the problem.

The genetic test for DM is available in the UK from Laboklin and is listed on our 'special' DNA application form. The forms are available from the Club's Health team health@wheaten.org.uk There is a discount for having both the PLN test and the DM test carried out at the same time.

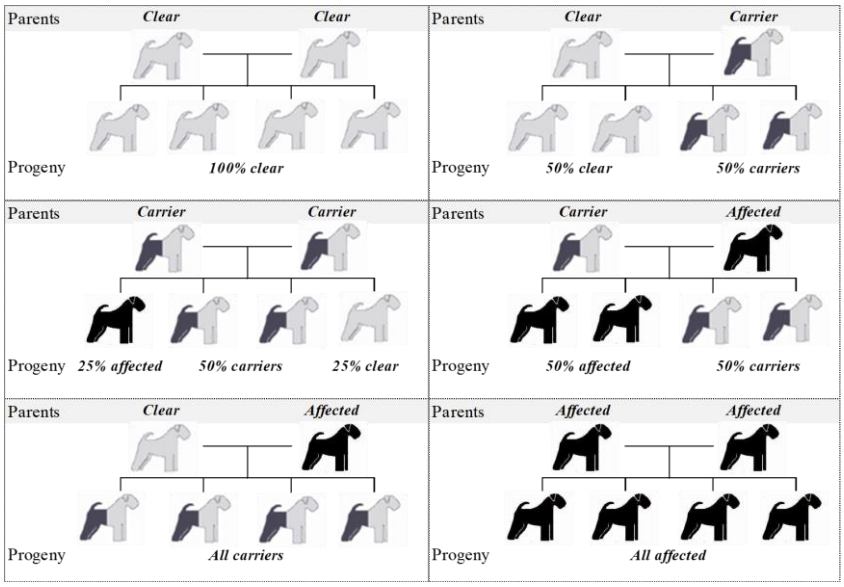
It is too soon to know how common the genetic markers are in the UK. However, from Laboklin's recent Europe-wide DM test results and the few that have been reported by our owners, it does appear that the genetic markers for DM are more prevalent than previously thought.

The current genetic marker only identifies the 'risk' of DM. However, research is continuing at the University of Missouri who have found that a biomarker test, which identifies a specific protein in blood and spinal fluid and is used to diagnose Amyotrophic Lateral Sclerosis (ALS) in people (similar to motor neurone disease), appears to also be applicable to identifying DM in dogs. It is hoped that this eventually may serve as a diagnostic tool.

Further reading on this condition

www.fitzpatrickreferrals.co.uk/neurology/canine-degenerative-myelopathy
www.dogsnaturallymagazine.com/degenerative-myelopathy-in-dogs
www.thekennelclub.org.uk - search under 'Health' for Myelopathy

Inheritance of Recessive Genes



Note: This would apply over a large number of matings of the same pair of animals. Very few litters would demonstrate these precise proportions.

The following points should be noted:

- To produce an affected puppy, both parents must carry the affecting gene.
- It is clinically normal healthy carriers which spread the disease.
- Since carriers are only identified when they produce affected offspring, the number of carriers is potentially far greater than the few that have been identified.
- The diagram shows the statistical expectations, but it should be noted that, as with the tossing of a coin, the ratios shown above do not necessarily occur in each individual litter. Therefore, especially in small litters, the expected 25% affected progeny from carrier matings will not regularly occur, e.g. one litter may be totally unaffected and the next all affected.
- It must be stressed that it is only ADVISED to assume the mode of inheritance of renal dysplasia (RD) is via a recessive gene.
- The mode of inheritance for protein losing enteropathy (PLE), and protein losing nephropathy (PLN) is not yet understood.

Post Mortem Requirements

Every Wheaten is an important link.

The kidneys of any Wheaten dying of kidney failure or anaemia should be sent to the University of Cambridge, as this is the **only way of confirming RD**. This should be done for any dog up to the age of 8 or so for RD. It does not mean a full post mortem for the dog.

If your dog is so ill that euthanasia seems likely and you are willing to have a post mortem, it is strongly advised that you provide your vet with this information in advance.

For renal dysplasia (RD) and protein losing nephropathy (PLN)

The **tissue sample** required is: -

Two **kidneys**, cut in half, and preserved in 10x volume of 10% buffered formalin.

(If the death of the dog occurs at the weekend, the body should be refrigerated, **never frozen** *and the samples taken on Monday for despatch to the University of Cambridge)*

The following should be collected **before euthanasia** and sent with the other items:

Blood: 5ml serum **AND** a maximum of 5ml in EDTA (anticoagulant).

If possible, a 5ml sample of **urine** from the bladder should be included:

Urine: A sample of at least 5ml.

Please note: If there has been a presence of GI disease previous to clinically evident PLN, this could be a sign of both conditions (PLE and PLN). In this case intestinal samples should be included below.

For protein losing enteropathy (PLE):

The **tissue samples** required are: -

Two **kidneys,** cut in half, and preserved in 10x volume of 10% buffered formalin.

Half-inch long sections of the **duodenum, jejunum and ileum** preserved in neutral buffered formalin solution. *Please Note: these samples should be taken within an hour or two after death due to rapid deterioration of the gut.*

The following should be collected **before euthanasia** and sent with the other items:

Blood: 5ml serum **AND** a maximum of 5ml in EDTA (anticoagulant).

If possible, the following should be included:

Urine: A sample of at least 5ml.

The following details should accompany all post mortem samples:

- Name of dog (pet name and pedigree name)
- Date of birth
- Owner's name and address
- 5 generation pedigree *(contact the Health Team if you require a copy)*
- Daily fluid intake and diet fed
- Dog's approximate weight
- Copies of any tests (blood, urine etc.) done on the dog during its recent illness
- Any other information the owner/vet may think appropriate

All samples should be carefully packed and sent first class post to:

The University of Cambridge

Central Diagnostic Services,
Department of Veterinary Medicine,
Madingley Road,
Cambridge CB3 0ES

Tel : 01223 337625
Email : clinpath@vet.cam.ac.uk
Fax : 01223 339090

It is advisable that your veterinarian notifies Cambridge that a sample is being sent for post mortem. **As this is a new service, arranged following the closure of the AHT, we ask that you notify Kate Watkins katerriers@gmail.com OR Tracy Hammond calvenace@gmail.com so they can alert Cambridge Diagnostic Services that a Wheaten sample will be arriving.**

The SCWT Club Health fund is available to help with post mortem costs in cases where inheritable disease may be present. The Club in return requires a copy of the post mortem report.

The post mortem report will be sent to the referring veterinary practice by Cambridge who will then relay the results to the owner of the dog. Owners are encouraged to tell their breeder of the results and to send a copy of the report to the SCWT Club for the Health files.

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Contacts

Veterinary advisors

Should your veterinarian require further information he/she can **contact directly** the Club's veterinary advisors for further advice –

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Diagram of a longitudinal section through the centre of a kidney from *The Veterinary Notes for Dog Owners* editor Trevor Turner BVet Med, MRCVS

The Soft Coated Wheaten Terrier Club of America (SCWTCA): www.scwtca.org RD/PLE/PLN Charts
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