WHEATEN HEALTH INITIATIVE

An Independent Health Group

'To provide a platform for the reception and transmission of information about the health and well-being of the Soft-Coated Wheaten Terrier'

The

Soft-Coated Wheaten Terrier

HEALTH HANDBOOK

Web Site:  www.wheatenhealthinitiative.com
E-mail:  wheatenhealth@aol.com

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An Independent Health Group

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Wheaten Health Initiative is a UK based organisation which was formed in 2003 and is an autonomous health group working independently of any other organisation or club. Our sole aim is caring for the health and well-being of the Soft-Coated Wheaten Terrier (SCWT).

Preface:
Please, remember, the majority of Soft-Coated Wheaten Terriers, will live long and active lives, because on the whole, SCWT’s are healthy and robust dogs but they do have genetic predispositions to certain diseases and owners need to be aware of these.

We hope that the information written in this Health Handbook will help you to understand the known hereditary diseases, which can affect the breed, and it is written within the context of the body systems that they affect.

The handbook also includes ‘Other Medical Conditions’ which have occasionally been known to affect the SCWT.

This handbook also incorporates our previous publication ‘Medical Terms’, which was compiled by the late Roni Andrews, Soldiersong Soft-Coated Wheaten Terriers, USA.

If you have any concerns about any health issues regarding your dog, which you would like to discuss with a member of WHI please contact us. We will, if you wish, keep this confidential.

If you have any questions on any of the topics discussed in this handbook please contact us.

Disclaimer:
Every effort has been made to ensure that there are no errors in this document. If any errors have occurred or the information becomes obsolete due to new developments, WHI will amend the information at the earliest possible time.

All Links within this Health Handbook are reproduced in good faith to provide a diversity of Wheaten and other canine related information. However WHI cannot verify the robustness and accuracy of these sites

Wheaten Health Initiative
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  ©Produced from information on the SCWT Club of America web site, with their kind permission.
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  ©Produced from information on the SCWT Club of America web site, with their kind permission.

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INTRODUCTION:
Have you ever thought that you really should know more about the problems which may affect your Wheaten, started to read information, and suddenly found that it is not written in the same language that you normally use?

Wheaten Health Initiative (WHI), hope that the information within this Health Handbook will help you to understand more about the words and terms that are used by the professionals caring for your dog.

Educating and providing information are Wheaten Health Initiative’s principal objectives, aiming in this way to keep breeders, owners and the veterinary professionals up to date with the latest research and testing procedures.

We believe EVERYONE needs the facts about the hereditary diseases that can affect the breed.

"Their Health In Our Hands"

Our Logo is designed to remind us all that the health of this beautiful breed, is literally, in our hands, owners and breeders alike. It also depicts our vision of global co-operation.

In owning a Wheaten, you take on not just a dog, but a shared responsibility for the future of the breed.

Why do you need to monitor your dog’s health?
It is important owners learn to recognise the signs of the diseases that may affect their Wheaten. In this way the chances of catching a disease in its early stages are increased and therefore the opportunity to do something to prevent the situation from becoming any more serious or life threatening may present itself.

- Firstly educate yourself about your breed.
- Read the material available on Wheaten health and canine health in general.
- Learn the symptoms of those problems that could affect your dog’s health.
- Test your dog to establish the baseline for your dog and then test on a regular basis (if possible annually).
- Learn how to monitor your Wheaten’s health i.e. the various tests you can do.
- Familiarize yourself with the purpose of each test and learn what each result means.
- Keep records of all testing in a file or by using WatchDog* (UK) Health Tracker. (*Email: www.wheatenhealth@aol.com for further information on the Health Tracker.)
Why it is important for you to establish what is the baseline for your dog?

Every dog is an individual in its own right and what may be considered “normal” for one dog may differ slightly for another. If you were to compare any of your dog’s test results, including temperature and respiration rates, with another owner’s results, you might find this was the case. Therefore, there should be no cause for alarm, although any large discrepancies in values may need further investigation.

However, having done that first blood/urine test you will have established a ‘baseline’ for your own dog. Each test you do can be seen as a “snap shot” of your Wheaten’s health. So, in order to monitor your dog’s health correctly, it is important to ask for a copy of the test results from your vet and to keep them on file or use WatchDog*(UK) Health Tracker, so that you can compare later test results with earlier ones.

In this way you will be able to identify any variations that may indicate a change, either up or down, in your Wheaten’s health. Again there is no cause for alarm, bearing in mind that your dog may just be having an “off” day at the time of the test. However, if later testing shows a developing trend, you would be wise to consult with your vet.

You and Your Vet:
- Try to develop a good working relationship with your vet.
- When choosing a vet ask if they are familiar with the Soft-Coated Wheaten Terrier and its medical conditions.
- A copy of ‘Testing Protocols’ should be taken to your Vet and you should ask if they are able to follow these Breed health protocols.
- If your vet is not familiar with the breed, ask if he/she would be happy to receive further information from you on Wheaten health issues.
- Keep clear records to ensure that testing takes place annually.

Provide your vet with a copy of the following to retain for his/her future information:
1. Recommended Health Screening for Renal Dysplasia (RD), Protein Losing Enteropathy (PLE), Protein Losing Nephropathy (PLN), and Addison’s disease. (Comparison Chart of Hereditable Diseases is provided in this handbook)
2. Annual Health Screening Schedule for PLE/PLN in the SCWT.
3. *Protocol for the Alpha Protease Inhibitor (API) test. Fecal API Collections screening test for PLE.
4. MA test for Microalbuminuria.

Tests referred to in 3 is only available in North America, refer to www.scwtca.org for more information.
Normal Parameters for a Healthy Dog:
The average canine Gestation Period is: approximately 63 days

Body Height/Weight Ratio:
The UK Kennel Club Breed Standard for the SCWT states:

**Point of Withers**

**Height:**
- **Dogs**: approximately 18-19½ inches (46-49cms), i.e. measured at the point of the withers
- **Bitches**: Slightly less

**Weight:**
- **Dogs**: 35-45 pounds (16-20.5 kilograms)
- **Bitches**: Somewhat less.

Note: The above is approximate; weight should be in relation to height.

Every dog is an individual therefore the following are approximations:

**Normal Canine Body Temperature:**
- 38-39.2°C
- 100.5-102.5°F

A dog’s body temperature can vary between 38°C to 39.2°C (100.5°F to 102.5°F), this can be dependent on a number of reasons; emotional state, level of activity, environment and even time of day.

A video giving tips on how to take a dog’s temperature is available on our website: [http://www.wheatenhealthinitiative.com/Pages/healthkeyfacts.html](http://www.wheatenhealthinitiative.com/Pages/healthkeyfacts.html)

Please remember – Temperatures outside these values do not automatically indicate that a disease or disorder is present.

However, if your dog’s temperature drops below 37.2 (99°F), or rises above 40°C (104°F), then this could give cause for concern and **you should contact your vet immediately**.

**What Your Dog’s Temperature may mean:**

<table>
<thead>
<tr>
<th>Degrees Centigrade (°C)</th>
<th>Degrees Fahrenheit (°F)</th>
<th>Possible cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>36.6</td>
<td>98</td>
<td>Hypothermia keep your dog warm</td>
</tr>
<tr>
<td>37.2</td>
<td>99</td>
<td>Abnormal</td>
</tr>
<tr>
<td>38 – 39.2</td>
<td>101.5 – 102.5</td>
<td>Normal temperature</td>
</tr>
<tr>
<td>39.4</td>
<td>103</td>
<td>Moderate fever</td>
</tr>
<tr>
<td>40</td>
<td>104</td>
<td>High fever</td>
</tr>
<tr>
<td>40.5</td>
<td>105</td>
<td>Dangerous</td>
</tr>
<tr>
<td>41.1</td>
<td>106</td>
<td>Heatstroke cool down immediately</td>
</tr>
</tbody>
</table>

**Overheating:**
Dogs do not have sweat glands, other than on their footpads, they have to pant in order to reduce their body temperature. Be aware that panting would not help in reducing the dog’s temperature if a dog is suffering from heatstroke.
What you should do:
Remove your dog from the direct sunlight and try to establish a good flow of air around the dog – use an electric fan if possible. The dog should not be immersed in ice or ice-cold water. To decrease the dog’s temperature use cool water and damp cloths or a spray bottle if available, particularly under the front armpits, the groin and the flanks.

When the dog’s temperature has decreased to 39°C (103°F), or below, take the dog to the vet so that the core body temperature can be assessed and veterinary treatment given.

Never leave your dog in a car on hot day.

Pulse Rate = 70-120 beats/minute
Pulse Rate is the number of heart beats per minute. Larger dogs have slower rates than small dogs, and dogs that are in good physical condition will have lower heart rates than dogs of similar size and age that are not physically fit. Puppies typically have higher heart rates, up to one year of age.

Respiration Rate = 18-34 breaths/minute
Respiration rate is the number of breaths per minute. Normal respiratory rates are taken when a dog is resting. A dog that is in pain, having heart or respiratory problems, or suffering from heatstroke, or is excited will usually have an increased respiratory rate. It is therefore important to look at the overall situation, and condition to assess the respiratory rate correctly.

Health Testing:
Testing Protocols – Information for Owners (Adult Dogs):
There is, at the present time, no definitive test for any of the hereditary diseases. However, the Key Veterinary Researchers recommend that you perform 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; Protein Losing Enteropathy (PLE), Protein Losing Nephropathy (PLN), Renal Dysplasia (RD) and Addison’s Disease which can affect the breed.

Quick Definitions:
- PLE & PLN are syndromes characterised by the loss of proteins from the gastrointestinal tract (PLE); or the kidneys (PLN).
- RD – Renal Dysplasia is the abnormal development of the kidney. This malformation can result in early renal failure.
- Addison’s Disease - Addison’s Disease (Hypoadrenocorticism) is the insufficient production and secretion of hormones (glucocorticoids, mineralocorticoids) by the adrenal gland cortex.

Clinical Signs: Clinical signs of a disease are the things you can see or that your veterinarian may discover on his/her physical examination of your Wheaten.
**Testing is important:** In many conditions, clinical signs do not show up until well after tests may show signs of the disease. Also, many clinical signs of one disease can also be signs of another.

<table>
<thead>
<tr>
<th>RD</th>
<th>PLE</th>
<th>PLN&lt;sup&gt;1&lt;/sup&gt;</th>
<th>Addison’s&lt;sup&gt;2&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Increased water consumption</td>
<td>• Vomiting</td>
<td>• Listlessness/ depression</td>
<td>• Listlessness/ depression</td>
</tr>
<tr>
<td>• Increased urination (dilute urine)</td>
<td>• Diarrhoea</td>
<td>• Decreased appetite, vomiting, weight loss</td>
<td>• Decreased appetite, vomiting, weight loss</td>
</tr>
<tr>
<td>• “Poor doer”</td>
<td>• Weight loss</td>
<td>• Ascites, edema, pleural effusion</td>
<td>• Inability to handle stress</td>
</tr>
<tr>
<td>• Decreased appetite</td>
<td>• Ascites</td>
<td>• Increased water consumption</td>
<td>• Sudden collapse</td>
</tr>
<tr>
<td>• Vomiting</td>
<td>• Edema</td>
<td>• Increased urination (less common)</td>
<td>• Slow heart rate</td>
</tr>
<tr>
<td>• Possibly prone to urinary tract infection</td>
<td>• Plural effusion</td>
<td>• Thromboembolic phenomena and hypertension (less common)</td>
<td></td>
</tr>
</tbody>
</table>

<sup>1</sup>**PLE and 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 - lung, heart, brain, portal vein or distal aorta (saddle) before symptoms or renal failure start, even before there is increased serum creatinine or BUN.

<sup>2</sup>The clinical signs of **Addison’s Disease** are often non-specific and can mimic those of multiple other medical disorders.

**Laboratory Tests Run By Your Vet:**
Your veterinarian can check for signs of these diseases with blood and urine tests. It is important the panels run by your Vet test for everything listed here.

**Note:** that not all routine blood and urine tests do, so you must make sure the ones listed here are undertaken.

Blood and urine tests cannot predict whether a dog will develop these diseases. But they can determine whether or not a dog is now clear of signs of disease and establish baseline values for future comparison. Early detection can offer more choices for treatment and can often provide longer and better quality of life.

Your Veterinarian can undertake these tests ‘in-house’, or they may use an external Laboratory service.

Please include:

1. **Biochemical profile, including:**
   - Total protein (TP)
   - Albumin (Alb)
   - Globulin
   - Creatinine (Cr)
   - **Blood Urea Nitrogen** (BUN)
   - Cholesterol (Chol)
   - Sodium (Na)
   - Potassium (K+)
   - Phosphorus (Phos)
2. Complete Blood Count

3. Routine Urinalysis, including:
   - Specific gravity
   - Dipstick
   - Urinary sediment

4. Urine Protein/Creatinine Ratio, Vets - please refer to Health Tests>Information for Veterinarians on our website for information on ‘Pooled UPC’

5. *RECOMMENDED but OPTIONAL (North America only)
   - MA test for microalbuminuria or ERD

6. *RECOMMENDED but OPTIONAL (North America only)
   - Fecal API test

If you or your veterinarian suspects RD or Addison’s, the following tests can be undertaken:

**Renal Dysplasia (RD)**
- Abdominal radiographs/Ultrasound
- Final confirmation of RD, kidney biopsy (wedge, not Tru-cut).

**Addison’s**
- ACTH stimulation test

*Optional Tests (North America):*
The two optional tests may be early indicators of PLE and PLN. You can arrange for these through your veterinarian at the same time you do your annual screening. Alternatively, you can take the samples at home and ship them for testing in a kit SCWTCA has developed. For further information on these two tests visit the SCWTCA web site: www.scwtca.org

- **PLE:** Fecal API (FAPI) Test
- **PLN:** Microalbuminuria (MA) test
Diagnosing: RD; PLN & PLE & Addison’s Disease

These diseases can be difficult to diagnose and can be confused with each other. Here are some of the similarities and differences.

<table>
<thead>
<tr>
<th></th>
<th>RD</th>
<th>PLN</th>
<th>PLE</th>
<th>Addison’s</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age of Onset</strong></td>
<td>&lt;1-3 years</td>
<td>Mean ~ 6 years</td>
<td>Mean ~ 4.5 years</td>
<td>Young (in general)</td>
</tr>
<tr>
<td><strong>Sex Predilection</strong></td>
<td>None noted</td>
<td>Female: male=1.6</td>
<td>Female: male=1.7</td>
<td>Female (in general)</td>
</tr>
<tr>
<td><strong>Polyuria/Polydipsia</strong></td>
<td>Yes</td>
<td>Only25% had PU/PD</td>
<td>No, unless on steroids</td>
<td>Yes</td>
</tr>
<tr>
<td><strong>Vomiting/Diarrhoea</strong></td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td><strong>Ascites/Edema</strong></td>
<td>No</td>
<td>Possibly</td>
<td>Possibly</td>
<td>No</td>
</tr>
<tr>
<td><strong>Azotemia</strong></td>
<td>Yes</td>
<td>Eventually</td>
<td>No</td>
<td>Possibly (pre-renal)</td>
</tr>
<tr>
<td><strong>Kidney Size</strong></td>
<td>Small</td>
<td>May be normal</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>Hypoalbuminemia</strong></td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Possibly (melena)</td>
</tr>
<tr>
<td><strong>Hypoglobulinemia</strong></td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>Possibly (melena)</td>
</tr>
<tr>
<td><strong>Hypercholesterolemia</strong></td>
<td>No</td>
<td>Yes</td>
<td>Hypcholesterolemia</td>
<td>No</td>
</tr>
<tr>
<td><strong>Low Na/K ratio</strong></td>
<td>Not noted</td>
<td>Rarely (~10%)</td>
<td>Rarely (~10%)</td>
<td>Yes</td>
</tr>
<tr>
<td><strong>Urine Specific Gravity</strong></td>
<td>Isosthenuria</td>
<td>Mean 1.023</td>
<td>Mean 1.033</td>
<td>Low (medullary washout)</td>
</tr>
<tr>
<td><strong>Proteinuria</strong></td>
<td>None or mild</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td><strong>Histopathology</strong></td>
<td>Foetal Glomeruli,</td>
<td>Glomerulonephritis,</td>
<td>IBD, lymphangiectasia,</td>
<td>No</td>
</tr>
<tr>
<td></td>
<td>Foetal mesenchyme (K)</td>
<td>Glomerulosclerosis (K)</td>
<td>lymphangitis (I)</td>
<td></td>
</tr>
</tbody>
</table>

Source 1999 ACVIM PROCEEDINGS Soft Coated Wheaten Terrier PLE-PLN; Dr Meryl P. Littman VMD DACVIM, Philadelphia PA

Other important lab findings:

<table>
<thead>
<tr>
<th>RD</th>
<th>PLE</th>
<th>PLN¹</th>
<th>Addison’s²</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Elevated Creatinine</td>
<td>• Eosinophilia</td>
<td>• Elevated Serum Creatinine</td>
<td>• Elevated Serum Creatinine</td>
</tr>
<tr>
<td>• Elevated BUN</td>
<td>• Lymphopenia</td>
<td>• Elevated BUN</td>
<td>• Elevated BUN</td>
</tr>
<tr>
<td>• Low total protein</td>
<td></td>
<td>• Elevated Urine Protein Creatinine Ratio*</td>
<td>• Elevated BUN</td>
</tr>
<tr>
<td></td>
<td></td>
<td>*very important!</td>
<td></td>
</tr>
</tbody>
</table>

Remember: diagnosis of PLE/PLN, RD, or Addison’s is dependent on evaluating everything – test results, clinical signs and symptoms – so do not assume one “bad” test means your dog has these diseases.
What to do next?

- Make sure you test every year and have your veterinarian compare results.
- Keep a copy of the results in a file at home so you can always refer back to them or provide them to a new veterinarian if you move.
- Some people keep a spreadsheet on their computer with all the test results. The *Watchdog Health Tracker* is available in the UK, via WHI and is available in the USA through the SCWT Endowment Fund.

*SCWTCA and WHI neither recommend nor endorse this tool but provide this information for your assistance.*

If these results show any abnormalities, you and your vet need to take immediate action.

1. **You** – Contact your breeder immediately, he/she will want to know in order to help you and to take action on other dogs in their breeding program.
2. **Your veterinarian** – Contact a Veterinary Specialist in your area.

*WHI would like to thank: Dr Allenspach, Dr Littman and Dr Vaden and the ©Soft-Coated Wheaten Terrier Club of America (SCWTCA – visit [www.scwtca.org](http://www.scwtca.org)) for their kind permission to reproduce the ‘Testing Protocol’ information.*

**Tests recommended - Prior to breeding your Soft-Coated Wheaten Terrier - UK:**

Should you choose to breed, it is recommended that the sire and dam have the following tests prior to mating:

- Blood and urinalysis in accordance with the ‘Testing Protocols’.
- *Eye tested (eye certificate - issued annually).*
- *Hip scored (this only need be undertaken once in a dog’s lifetime).*
- Hearing Test – this is recommended for dogs from ‘high risk’ lines (For more information read the section on ‘Other Conditions’ - ‘Deafness’ within this Health Handbook).
- Genetic PLN-Associated Variant Gene Test (this only need to be done once in a dog’s lifetime).

*Eye & Hip Testing are part of the British Veterinary Association (BVA) Health Schemes. Contact details are listed at the back of the Health Handbook.*

**Genetic PLN-Associated Variant Gene Test:**

**UK Swab Kits:** available from the Health Team of The Soft Coated Wheaten Terrier Club of GB. Contact details are listed at the back of the Health Handbook.

**USA:** Contact details are listed at the back of the Health Handbook.
Health Testing Puppies:
Puppies’ body systems are immature and to undertake a full blood and urinalysis before the age of 12 to 18 months could possibly produce spurious results. Therefore, unless a Vet advises a full blood and urinalysis this is not required.

Responsible breeders, prior to homing their puppies, undertake tests on their litters. These may include:

- **Basic kidney function** - This is a blood test taken at approximately 7 weeks of age to assess kidney function and can usually be completed by your vet 'in-house'.
  - Creatinine
  - Urea (BUN)
  - Phosphate levels

- **DNA storage** - At the same time as the basic kidney function test an additional amount of blood can be taken and sent to the *Animal Health Trust (AHT)* for DNA storage. Cheek Swabs are also available to undertake DNA Storage. (see 'Information about DNA Storage at the AHT, UK' below)

- **Eye test** - At approximately 6-8 weeks of age by a BVA approved Ophthalmic Vet, who checks for retinal folds and other eye diseases. (For more information, go to the section on ‘Eyes’ within this Health Handbook.

- **Hearing Test** – This is recommended for puppies that have ‘high risk’ ancestral lines. However, the last reported cases of deafness or hearing impairment to the SCWT Club of GB were in 1998. (For more information, go to the section on ‘Deafness’ within this Health Handbook).

Animal Health Trust (AHT) – Diagnostic Services UK:
The AHT are an external Laboratory that has facilities to undertake testing and screening.
http://www.aht.org.uk/cms-display/diagnostic_services.html

The AHT also stores DNA for SCWT’s.

Information about DNA Storage at the AHT, UK:
Please consider having DNA from all Wheatens’ that you own or breed stored at The Animal Health Trust (AHT).

**Why is DNA storage important?**
DNA is important for the future of the breed. **ALL** dogs, even those who are never bred from, are important. It is hoped that this DNA will enable researchers to find the deleterious (bad) mutations which cause hereditary diseases.

**What will this mean for breeders and owners?**
In the future, if breeders have ALL health information, including if a dog has these deleterious mutations, it will enable the breeder to select ‘safer’ breeding combinations. Over time, the deleterious mutations may be eliminated from the gene pool.

**How often do I have to collect DNA from my dog?**
Just once, DNA can be stored indefinitely.
How do I store DNA?
First check that the breeder has not already stored DNA prior to purchase.

1. **Single Dog(s):**
   Storage can be achieved 2 ways:
   - **Cheek swab** - which is simple and easy and does not require a visit to the Vet
   - **Blood sample** - if your Wheaten is having blood taken for the recommended annual testing an additional amount of 5ml blood sample in an EDTA Tube can be sent to the AHT for DNA Storage purposes

2. **Litters:**
   DNA from family groups will be vital for future research and many breeders have DNA stored from each of the puppies in their litters, prior to the pups leaving for their new homes.
   - **Cheek swab** - which is simple and easy and does not require a visit to the Vet,
   - **Blood sample** - if you are having your puppies’ kidney function tested then an additional amount of 5ml blood sample in an EDTA Tube can be sent to the AHT for DNA Storage purposes

**AHT Forms & Swab Kits** are available from The Soft Coated Wheaten Terrier Club of GB. Contact the Health Team on their website: [http://wheaten.org.uk/index.php/contact/club-committee-contacts](http://wheaten.org.uk/index.php/contact/club-committee-contacts)

**Send to the AHT:**
- Cheek swab(s) or blood sample
- Completed AHT Form(s)
- *5 generation pedigree

*WHI can supply a pedigree upon request. WHI will require your dog’s Kennel Club name, parent names and date of birth.

**Who should I inform if I store DNA?**
Your breeder and the SCWT Club of GB if you have your Wheaten’s DNA stored at the AHT.

**Please inform the AHT of any illnesses that your dog(s)/Puppy(s) has, and keep them updated of any major health changes after the DNA has been submitted.**
UK - Post Mortem Information:

Dear Owner

Nobody likes to think of the time when we might lose our Wheaten friend and companion. The moment we do lose them is very emotional and sensitive for us and a time when the pain of grief can cloud our better judgement.

At a time like this, suggestions of a post mortem are almost unthinkable, but the last loving act could be to allow your Wheaten to provide valuable medical information that may benefit the breed in future; the health of every Wheaten is important, even if it has not been bred from.

Of course, this is a difficult task to take on board so soon after death, but it may help and reassure you that it may not be necessary for a 'full body' post mortem to be carried out. Often, all that may be required, unless there are serious concerns over the cause of death, is to take a sample, using keyhole surgery, from the kidney and/or intestine.

If you would like your Wheaten to make this valuable contribution to wheaten health, it may help to discuss the possibility with your Veterinary Practice so that they can be aware of your wishes well in advance so he/she can discretely carry out the necessary procedures on your behalf, when the time comes.

Importantly, if it is suspected that your dog has died of a hereditary disease, please inform your breeder, and the SCWT Club of GB as this may provide valuable information for future breed health.

Further details about the PM process are given below.

Thank you for taking the time to consider this matter.

Regards
Wheaten Health Initiative

--------------------------------------------

Information for Owners and Veterinarians:

The request for pathological examination should be made through your veterinary surgeon, who will make contact with either the Animal Health Trust or the Royal Veterinary College, as they have experience in performing Post Mortems on Wheaten Terriers.

The AHT and the RVC will charge for their services, as will your Vet. and the cost of the process will depend on whether a whole body or tissue samples are required.

Should your Wheaten have a hereditary disease, or is strongly suspected to have died of a hereditary disease, then please contact the Soft Coated Wheaten Terrier Club of GB as you may qualify for assistance from the health fund. Contact details are at the back of this Handbook and also available on our web site.

Your post mortem report will go back to the referring veterinarian, who would then relay the report and results to you.

DNA will also be collected on PM (if it has not already been previously stored).
Important:
Please give your Vet all this information so he/she knows the correct storage procedure and who to contact – this is very important.

Ideally the post mortem needs to take place:

- Within 48 hours of death or euthanasia. **However, in the case of samples of the intestine, it is important that these are taken as soon as possible after death.**

- The body should be refrigerated (**not frozen**)

- If death occurs over a weekend, please do not send anything to the RVC or AHT until Monday morning.

- Please include a summary of the clinical history leading up to death, or euthanasia, with the body/tissue samples.

- If a whole body post mortem is not necessary, advice regarding the appropriate tissues required for collection can be obtained by contacting one of the pathologists at the RVC or the AHT.

- The RVC and the AHT are able to give advice to the referring veterinary surgeon, on a case by case basis, submission of the whole body or parts thereof and the cost of the procedure.

Who to contact:
The pathology departments at the Royal Veterinary College and the Animal Health Trust have experience of the diagnosis of Protein Losing Enteropathy (PLE), Protein Losing Nephropathy (PLN), Renal Dysplasia (RD) and Addison’s disease, including the criteria used to distinguish between a kidney affected by RD and one which is affected by PLN.

**Vet Referral Only:**

**Animal Health Trust** - Diagnostic Laboratory Services
Lanwades Park
Kentford
Newmarket
Suffolk,
CB8 7UU
**Telephone:** 01638 552993  **Fax:** 01638 555643
E-mail: diagnostics@aht.org.uk  Web site: www.aht.org.uk

**Royal Veterinary College**
Professor Ken Smith, BVM&S, PhD, FHEA FRCPath, FRCVS
Professor of Companion Animal Pathology
The Royal Veterinary College
Hawkshead Lane
North Mymms
Hatfield
Hertfordshire
AL9 7TA
**Telephone:** 01707 666208  **Fax:** 01707 661464
Email: ksmith@rvc.ac.uk  Web site: www.rvc.ac.uk
HEREDITARY DISEASES

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 break down carbohydrates, proteins and fats into smaller molecules. These molecules are absorbed by special cells while the mixture is churned and pushed along by intestinal muscle contractions. Dogs 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 inflammatory bowel disease (IBD).

Protein Losing Enteropathy (PLE):
- 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 Protein Losing Enteropathy (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.
- Please refer to the Comparison Chart of Hereditable Diseases for signs and symptoms of this disease.
- 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 test available to show if dogs are carrying the deleterious (bad) mutations which cause this disease.
**Endocrine System:**

The adrenal cortex produces, among other things, steroid hormones which regulate carbohydrate and fat.

**Addison’s Disease:**

Addison’s disease is the common name for Hypoadrenocorticism. Addison’s disease can occur in dogs of any age, sex or breed although more females are affected than males. It usually is a disease of young and middle aged dogs. The disease is a ‘great mimic’ and can be very difficult to diagnose as there is no one clinical sign specific to Addison’s and these may resemble signs of other illnesses.

The adrenal gland can be damaged by approximately 90% before signs of the disease are seen. The hormones produced by the adrenal glands are important for life. This disease, once diagnosed, can be treated by replacing the hormones produced by the glands which are required for survival.

The adrenal glands secrete adrenal hormones which modify the body’s response to inflammation, stimulate the liver to raise the blood sugar, and also help to control the amount of water and salt in the body which affects blood volume and blood pressure. Addison’s disease is a severe or total deficiency of the adrenal hormones.

Adrenal insufficiency can be primary or secondary. Primary adrenocorticism affects the salt/potassium balance in the body and glucocorticoid as well. Secondary adrenocorticism usually affects glucocorticoids. It is not known why primary adrenocorticism occurs but it is thought it might be an immune mediated process. Secondary adrenocorticism probably occurs most often when prednisone or other cortisones being administered for medical reasons are suddenly withdrawn. It can occur if, for example, pituitary cancer interferes with the production of hormones that stimulate the adrenal glands.

Signs can be pretty vague; more severe signs occur when a dog with hypoadrenocorticism is stressed or when potassium levels get high enough to interfere with heart function. Dogs will sometimes suffer severe shock symptoms when stressed which can lead to rapid death. When potassium reaches high levels heart stoppage can occur which can be fatal. In some cases, particularly regarding secondary Addison’s disease, there are no detectable electrolyte changes.
Signs & Symptoms:
- The clinical signs are variable
- Initially the signs may be mild and very vague
- With an acute crisis the signs are more pronounced
- Lethargy & weakness
- Poor appetite
- Vomiting
- Diarrhoea
- Weight loss
- Depression
- Dehydration
- Excessive thirst and water intake (polydipsia)
- Low body temperature, shaking, collapse, low heart rate
- Please refer to the Comparison Chart of Hereditable Diseases for signs and symptoms of this disease.

It is **not known** if Addison’s disease is an inherited disease in the Wheaten Terrier, although there appears to be a higher than average predisposition for it.

Diagnosis:
This disease can be hard to differentiate from renal failure as the symptoms and even the blood work can be similar. Electrolyte levels can show as normal but Addison’s can sometimes be diagnosed by picking up the changes in the ratio between sodium and potassium levels, this can be easily missed unless it is specifically looked for.

**ACTH Response Test** – The ACTH response test will be necessary to make an accurate diagnosis.
Dogs are usually admitted to the vet’s surgery for a couple of hours. Blood is taken for analysis, followed by an injection which stimulates the production of adrenal hormones. After approximately 1½ -2 hours blood is again taken for analysis, if the production of the adrenal hormones is negative then Addison’s disease (hypoadrenocorticism) is diagnosed.

Treatment:
This depends on whether the onset of illness is acute with severe symptoms, or whether more mild chronic signs are present. For acute signs, i.e. Addisonian crisis, treatment would be administered by emergency admittance to the vet’s surgery. This may include intravenous fluid therapy, electrolyte and acid-base monitoring, and corticosteroid and mineralocorticoid replacement therapy. For chronic disease it may include corticosteroid and mineralocorticoid replacement therapy and daily salt supplementation. Hopefully, the disease will be diagnosed before an Addisonian crisis occurs and treated with prescribed medications.

At home the dog needs a stress reduced environment since its glands cannot produce the hormone that helps it handle stress. Stress can cause relapses of symptoms if not properly treated.
Urinary System:

Kidney Anatomy:
The kidneys filter waste and extra fluid from the blood. The filtering process takes place in the nephron where microscopic blood vessel filters called glomeruli, are attached to fluid-collecting tubules. A number of different disease processes can damage the glomeruli, thereby causing kidney failure.

Glomerulonephritis and glomerulosclerosis are broad terms that include many forms of damage to the glomeruli.

Some forms of kidney failure can be slowed down but scarred glomeruli can never be repaired. This is what makes early detection so vital. Treatment given in the early stages of kidney failure depends on the disease causing the damage.

Kidney failure may be ‘silent’ for many years. Approximately 70% of the kidney can be damaged before any physical signs show themselves.

Glomeruli
The glomeruli are the filters of the kidneys (imagine a water filter in a jug), they filter the blood and make urine. Normally, large molecules such as proteins, and cells such as red blood cells or white blood cells, do not pass through the filters and are retained within the blood because they are so important for health. Small molecules pass completely through the filters. Some of these are completely reabsorbed back into the blood since they are so important in maintaining the right chemical balance of the body e.g. glucose, salt etc. Other molecules, which are not required for body functions are passed freely into the urine, for example Urea, Uric Acid and Creatinine.

There are two main effects of damage to the glomeruli. Substances, which are normally retained in the circulation escape into the urine through the filtration mechanism, one of these is Albumin. As a consequence, protein and red cells appear in the urine and can be detected by a dipstick urine test. Protein in the urine is called proteinuria. Normally there is very little protein in the urine. If the damage gets worse, the filter shuts down and that function of the kidney is lost. If sufficient damage occurs to enough glomeruli kidney failure may occur.

Glomerulonephritis
Glomerulonephritis is the inflammation of the membrane tissue in the kidney that serves as a filter, separating wastes and extra fluid from the blood. Glomerulosclerosis describes the scarring or hardening of the tiny blood vessels within the kidney.
Protein Losing Nephropathy (PLN):

- This can be of late onset and the damage to the kidney may not show until the end stages of the disease.
- PLN is a condition in which plasma protein is lost to excess in the kidney.
- In the SCWT the most common disease causing PLN syndrome is glomerulonephritis.
- The term “glomerulonephritis” defines a group of inflammatory diseases of the kidneys affecting the most important functional components of renal tissue, the glomeruli and adjacent structures.
- There are several immune mechanisms involved in this inflammatory disease.
- SCWTs affected with PLN have damaged glomeruli because the ‘holes’ in the sieve (basement membrane) are too large and allow more than waste to pass through.
- One of the larger molecules that pass through the faulty sieve is protein. That is why excess protein (Albumin) is found in the urine of SCWTs who have one of the diseases that causes PLN.
- Please refer to the Comparison Chart of Hereditable Diseases for signs and symptoms of this disease.
- Tests necessary to detect the presence of PLN are blood, urine and if necessary, endoscope biopsy.

- The mode of inheritance for PLN is not known, but in 2012, Dr Meryl Littman and Dr Paula Henthorn identified mutations associated with PLN. As a result, there is now a test using a non-invasive cheek swab, which an owner can use and submit to the University Of Pennsylvania School Of Veterinary Medicine for interpretation. Please visit the WHI web site for further details of the test: http://www.wheatenhealthinitiative.com/Pages/PLNResearch.html

For the PLN-Variant Gene Test it is preferred that Penn Vet Laboratory is used as this enables continuity of research. In Europe and Scandinavia Laboklin Laboratories also offer the test but the result does not directly aid this research.

PLN-Associated Variant Gene Test Result Definitions - this table clarifies the reporting formats between Penn Vet and Laboklin:

<table>
<thead>
<tr>
<th>Genetic term</th>
<th>Definition</th>
<th>What does this mean?</th>
<th>Other Common Terms</th>
<th>Results Penn Vet</th>
<th>Results Laboklin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Homozygous Negative</td>
<td>A dog without any of the variant alleles</td>
<td>A dog that has no copies of the variant allele is at the least risk of developing PLN</td>
<td>0, 0/0, No copies, ’Normal’, ’Clear’, Homozygous</td>
<td>1/1</td>
<td>N/N (Clear)</td>
</tr>
<tr>
<td>Heterozygote</td>
<td>A dog with one copy of the variant alleles</td>
<td>A dog with one copy of the variant allele is at medium risk of developing PLN</td>
<td>1, 0/1, ’Carrier’, 1 Copy, Heterozygous</td>
<td>1/2</td>
<td>N/PLN (Carrier)</td>
</tr>
<tr>
<td>Homozygous Positive</td>
<td>A dog with two copies of the variant alleles</td>
<td>A dog with two copies of the variant allele is at the highest risk of developing PLN, but this does not mean it will develop PLN</td>
<td>2, Both copies, Homozygous for the PLN causative mutation</td>
<td>2/2</td>
<td>PLN/PLN (Affected)</td>
</tr>
</tbody>
</table>

Affected refers to both copies of the allele, it does not mean the dog is currently or will be affected with PLN.
Renal Dysplasia (RD):

**History** - During the 1960s and 70s breeders noted that an unusually high instance of puppies and young Wheatens were dying, and a small number of dedicated breeders began seeking a reason for this. The SCWT Club of GB with help from Professor Andrew Nash, Glasgow University, and Geneticist Dr Bruce Cattenach, set up a testing and breeding programme in the early 1980s to identify the cause and to try and eliminate the frequency of the disease. This programme was very successful and there is now only an occasional case reported in the UK. Although the researchers advised that RD probably occurred via a recessive gene, no test has been developed to identify this gene. Because of the small gene pool available within the breed the gene is still present within the dog population, consequently breeders must remain vigilant and careful breeding is required to try to prevent this disease re-occurring.

Renal dysplasia is a developmental or genetic defect of the kidneys. Dogs affected with renal dysplasia have kidneys that did not properly develop when the foetus grew in the uterus and so are born with the problem.

Unhealthy or malformed nephrons in the kidney are replaced by fibrous tissue and microscopic cystic lesions in the renal cortex and decreased immature foetal glomeruli and cystic glomeruli. Eventually the kidney cannot do its job of cleansing the blood.

Dogs with Renal Dysplasia need to drink and urinate frequently. They cannot concentrate their urine making it very dilute and pale in colour.

There are various levels of arrested development in affected puppies. Therefore, some puppies show symptoms of kidney disease at, or shortly after birth, while others develop symptoms later in life.

Up to 70% of the kidney can be damaged before any signs of illness can occur.

- Renal dysplasia is a developmental or genetic defect of the kidneys. Dogs affected with renal dysplasia have kidneys that did not properly develop when the foetus grew in the uterus.
- The damage to the kidneys is present at birth.
- Tests necessary to detect the presence of RD are blood, urine and if necessary endoscope biopsy.
- Please refer to the Comparison Chart of Hereditable Diseases for signs and symptoms of this disease.
- The disease is genetic and the mode of inheritance is thought to be caused by a recessive mutation. This means that both parents must carry the gene for a puppy to be affected. (Inheritance of Recessive Genes chart refers).

*At the present time there is no test available to show if dogs are carrying the deleterious (bad) mutations which cause this disease.*
Wheatens who exhibit signs of kidney failure need to have careful diagnosis made, as RD and PLN can be mistaken for each other in the later stages of the disease process. The following chart assists with this comparison.

### Differences between RD and PLN

<table>
<thead>
<tr>
<th>Renal Dysplasia (RD)</th>
<th>Protein Losing Nephropathy (PLN)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Usually referred to as Juvenile Renal Dysplasia. Dogs generally die between the ages of 6 weeks to 3 years.</td>
<td>Dogs tend to show their illness at 5-7 years old, but onset can be both earlier and later than this.</td>
</tr>
<tr>
<td>Dogs drink large amounts of water. Their Urine Specific Gravity (USG) is often low and the urine is dilute.</td>
<td>Dogs may not have these symptoms and can usually concentrate their urine until they reach end stage renal failure.</td>
</tr>
<tr>
<td>Dogs tend to lose little protein in the urine and the serum albumin stays normal.</td>
<td>Dogs lose large quantities of protein in the urine and their serum albumin drops. They also have a high protein/creatinine ratio.</td>
</tr>
<tr>
<td>Dogs eventually have high serum creatinine and Urea (BUN). Dogs do not have low albumin and high cholesterol.</td>
<td>Dogs eventually have high serum creatinine and Urea (BUN). Dogs have low albumin readings and high cholesterol.</td>
</tr>
<tr>
<td>Dogs are born with small, malformed kidneys.</td>
<td>Usually have normal sized kidneys until later stages of the disease.</td>
</tr>
<tr>
<td>In the renal cortex are microscopic cystic lesions, decreased and immature foetal glomeruli and cystic glomeruli.</td>
<td>Dogs show glomeruli changes, such as glomerulonephritis and/or glomerulosclerosis. They do not have many foetal glomeruli</td>
</tr>
<tr>
<td>Dogs are not usually predisposed to effusions and thromboembolism (clots).</td>
<td>Dogs can throw clots, in the lung, heart, brain, portal vein or distal aorta (saddle).</td>
</tr>
</tbody>
</table>
Inheritance of Recessive Genes

<table>
<thead>
<tr>
<th>Breed</th>
<th>Breed</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Affected</td>
<td>Affected</td>
<td>100% Affected</td>
</tr>
<tr>
<td>Affected</td>
<td>Carrier</td>
<td>50% Affected, 50% Carriers</td>
</tr>
<tr>
<td>Affected</td>
<td>Clear</td>
<td>100% Carriers</td>
</tr>
<tr>
<td>Carrier</td>
<td>Carrier</td>
<td>25% Affected, 25% Clear, 50% Carriers</td>
</tr>
<tr>
<td>Clear</td>
<td>Carrier</td>
<td>50% Clear, 50% Carrier</td>
</tr>
<tr>
<td>Clear</td>
<td>Clear</td>
<td>100% Clear</td>
</tr>
</tbody>
</table>

NB: This would apply over a number of matings, not all litters would manifest themselves in these proportions.

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.
This chart is not intended to alarm you or to suggest that your Soft-Coated Wheaten Terrier has inherited any of the diseases it describes. It is purely to provide information for your Vet and yourself.

### Comparison Chart of Hereditary Diseases

There are four hereditary diseases known to affect the breed.

<table>
<thead>
<tr>
<th>DISEASE</th>
<th>SYMPTOMS</th>
<th>LABORATORY ABNORMALITIES OFTEN ASSOCIATED WITH THIS DISEASE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Renal Dysplasia (RD)</strong></td>
<td>Increased water consumption</td>
<td>Low urine specific gravity</td>
</tr>
<tr>
<td></td>
<td>Increased urination (dilute urine)</td>
<td>Elevated creatinine and BUN</td>
</tr>
<tr>
<td></td>
<td>Poor doer, decreased appetite</td>
<td>Small kidneys</td>
</tr>
<tr>
<td></td>
<td>Vomiting</td>
<td>Small, hyperechoic kidneys with or without cysts seen via</td>
</tr>
<tr>
<td></td>
<td>Possibly prone to urinary tract infection.</td>
<td>abdominal ultrasound</td>
</tr>
<tr>
<td><strong>Protein Losing Enteropathy (PLE)</strong></td>
<td>Vomiting, Diarrhoea, Weight loss, Ascites, oedema, pleural effusion</td>
<td>Note that not all of the laboratory abnormalities are seen in every case. The most important are indicated by an asterisk. Hypoalbuminemia* Hypoglobulinemia* Eosinophilia Hypocholesterolemia Lymphopenia</td>
</tr>
<tr>
<td><strong>Protein Losing Nephropathy (PLN)</strong></td>
<td>Listlessness/depression, Decreased appetite, vomiting, weight loss, Ascites, oedema, pleural effusion, Increased water consumption, increased urination (less common), Thromboembolic phenomena and hypertension (less common)</td>
<td>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*</td>
</tr>
<tr>
<td><strong>Addison's Disease</strong></td>
<td>Listlessness/depression. Decreased appetite, vomiting, diarrhoea, weight loss. Inability to handle stress. Sudden collapse. Slow heart rate</td>
<td>Decrease in Na/K ratio (Sodium/potassium ratio) Abnormal ACTH stimulation test Elevated serum creatinine, BUN Sometimes, low urine specific gravity</td>
</tr>
</tbody>
</table>

Further information on all of these diseases can also be found in the 'Health Matters' section at: www.wheatenhealthinitiative.com and the health section of the SCWT Club of America's Web Site: www.scwtca.org

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Additional questions and answers relating to the hereditary diseases:

Soft-Coated Wheaten Terrier health information refers to PLE and PLN as syndromes. What is the difference between a Disease and a Syndrome?

**Disease** – illness or sickness often characterised by typical patient problems (symptoms) and physical findings (signs).

**Syndrome** – a combination of signs and symptoms that occur together.

What is a sub-clinical disease?
A sub-clinical disease is an illness that stays below the surface of clinical detection. A sub-clinical disease has no recognisable clinical findings. It is distinct from a clinical disease which has signs and symptoms that can be recognised. Many diseases, including diabetes, hypothyroidism, and rheumatoid arthritis, can be sub-clinical before surfacing as clinical diseases. Both PLE and PLN are sub-clinical diseases.

What is Protein Loss?
When a dog is losing protein into the urine or faeces there are several possible reasons. If the protein is being lost via the kidney then damage to the glomeruli is the cause. If the protein is being lost from the intestine it is a result of either malabsorption or maldigestion.

If my dog is diagnosed with RD, PLE or PLN what should I do?
If your dog is diagnosed with RD, PLE or PLN then ask your vet to contact a Veterinary Specialist in these diseases to provide your vet with advice on testing and treatment and discuss with him/her a course of treatment and diet suitable for the dog.

What should I do if my dog is diagnosed with Addison’s Disease?
Your vet, (or a veterinary specialist) and you can develop a diet and medication regime that, if followed, should allow your dog to lead a normal, active life.

Does stress affect how my dog feels if it has RD, PLE, PLN or Addison’s Disease?
Yes it does. Dogs should be maintained with a modified normal lifestyle. They will feel their best for the longest period of time if stress is managed and moderate exercise and play is provided.

Can a dog have RD and a Protein-losing disease at the same time?
A dog can have one or any combination of the diseases. Wheatens have been diagnosed with both RD and PLN and with PLN and PLE.

How do I know if my dog has PLN and not RD?
The kidneys of a dog with RD are quite different from a dog with PLN. The damage to the glomeruli in RD cases is, under microscopy, different to the damage shown with dogs with PLN. There are differences seen with blood chemistry and urinalysis. See Diagnostic Chart “Differences Between RD & PLN”

How can I find out if my dog has PLE?
PLE may be present in your dog long before clinical signs manifest, or urine and blood testing show protein loss. The best testing method for PLE is the Fecal API test, but this is only available in North America. Also an Endoscopic biopsy can be an additional aid to help confirm the diagnosis, this is the safest way as surgical biopsy can carry more risk.

How does my dog feel if he has PLE?
Since PLE can be a disease in which symptoms occur in mid to late life, we must assume that early stage PLE is not unpleasant for your dog. In North America PLE can be detected by Fecal API testing in a dog as young as 3 months old however, the
A dog may appear physically healthy for a number of years even with the disease present.

When clinical signs occur the dog does feel some physical symptoms. These symptoms vary with each dog and with the progression of the disease.

Symptoms can include: chronic diarrhoea, vomiting, bloody stools, abdominal pain and weight loss. Your dog may have one or several of these symptoms or other symptoms. How many and which symptoms and the severity of the symptoms depends on the type and severity of the disease.

**My dog hasn't had regular bouts of diarrhoea or vomiting, could it have PLE or PLN?**
These are classic signs that something is wrong but they are by no means always seen. Vomiting and diarrhoea are only two of the many signs, which might indicate PLE and or PLN. The absence of such symptoms does not necessarily indicate the absence of hereditary disease.

**My dog is middle-aged and appears healthy but could he/she still develop PLE or PLN?**
Dogs as old as 14 and previously healthy have been diagnosed with PLE or PLN. Many of the older dogs are asymptomatic (do not show physical signs of the illness), only bloods, urinalysis and in some cases biopsy can tell if a dog is affected.

**What is the point of testing if my dog is going to get PLE or PLN it will still do so?**
The outlook for dogs diagnosed with PLE and PLN is improving constantly due to ongoing research. Early diagnosis is essential; diet, medication, etc. can in many cases improve a dog’s lifestyle and its longevity. Dogs have been known to survive years after diagnosis.

**My puppy got sick at 7 months so will this be RD?**
Protein-losing disease can occur at any age. Although in the SCWT, RD is more common between the ages 7 weeks to 3 years, protein losing disease can occur at any age.

**Does a dog have to have a post-mortem before a definite diagnosis can be given?**
Although post mortems can provide additional proof, a diagnosis can be made while the dog is still living by blood tests, urinalysis, endoscopy or biopsy and ultrasound.

**A lot of old dogs die of kidney disease – could it be just old age not PLN?**
Changes in the kidney due to old age cannot be mistaken for those caused by PLN. Examination of the kidney in post mortem procedures will identify the distinct changes due to deterioration in old age from those caused by PLN.

**My dog is very healthy and does not drink a lot, so it does not need to be tested?**
A dog in the early stages of disease will not drink a lot. At this stage the dog is frequently in a state of ‘compensation’ where signs such as excessive drinking will not be apparent.

**My dog is healthy and does not pass urine frequently so it does not need to be tested?**
A dog in the early stages of disease will often not show obvious signs. This is not an indication that the dog is clear of hereditary disease.
Could my dog be a ‘carrier’ because it is a parent of a dog diagnosed with PLE or PLN?

There is no such certainty as we do not understand how the disease is inherited. Unfortunately, as yet there is NO identified mode of inheritance. The only way of being absolutely certain of whether a dog is a ‘carrier’ lies in the future with the identification of the gene(s) responsible.

Can I use my dog for breeding as a litter-mate has been diagnosed with a protein-losing disease?

There is no way of knowing at present if the litter-mate of an affected dog is ‘safe’ to breed from or not. It might be wise not to breed from a litter mate of an affected animal but, as the mode of inheritance is not yet established; it is not certain if every other dog in a litter could pass on the deleterious mutations.

Do I need to worry about protein-losing disease as I don’t have North American dogs in my pedigree?

There is NO foundation for assuming that only dogs born in North America are at risk of PLE/PLN. ALL the diseases are recognised hereditary diseases of ALL Soft-Coated Wheaten Terriers, no matter what their nationality or place of birth or coat type is etc.

Should I stop giving vaccines if my dog is affected by PLE, PLN or RD?

The research vets working on the SCWTCA health projects advise vaccines should not be given to a dog suffering from PLE, PLN or RD. The vets have determined that vaccines cause too much stress to the system of affected dogs. However, you may titre test to make certain your dog has immunity to those diseases for which you would normally vaccinate. Consult with your vet about titre testing.

What is a Fecal Alpha Protease Inhibitor (API) Test?

A Fecal API test is an extremely valuable method of detecting early stage PLE, even before symptoms or other testing can detect the syndrome. This test detects protein in the dog’s faeces. Faeces is collected, frozen, packaged and shipped in a prescribed manner. This is only available in North America at present; please refer to Protocol of Fecal API Collections, on the SCWT Club of America Web site: www.scwtca.org

Can anything be done to stop these diseases in the Wheaten Terriers?

- Keep up to date with the latest health information.
- Test regularly and repeatedly throughout the dog’s life.
- Be open and honest about the results.
- Make your breeding choices with care and use dogs from other people who are taking the same precautions with regard to their dogs.
- Choose not to breed from affected dogs, or littermates of affected dogs.
- Ask your vet to send a blood sample or cheek swab from your dog (for DNA storage) to the Animal Health Trust to further aid in the research into the treatment, cause and prevention of disease in the SCWT. North America is also storing DNA, visit the SCWTCA web site www.scwtca.org for information.

**Only by taking part in this collective effort and working together openly and honestly, can we safeguard the future of the Soft-Coated Wheaten Terrier.**
Glossary of terms:

Key words with regard to SCWT health:
- Renal Dysplasia (RD)
- Protein Losing Nephropathy (PLN) - *Glomerulonephritis* and *Glomerulosclerosis*
- Protein Losing Enteropathy (PLE) - *Inflammatory bowel disease (IBD)*, *Lymphangiectasia* and *Lymphangitis*.
- Addison’s disease
- Hyper – *high* (too much)
- Hypo – *low* (too little)

**Effusion** – an outpouring or escape of fluid into a part or tissue. Ascites (also called hydroperitonia) is the abnormal build up of effused fluid in the abdomen.

**Embolism** – the obstruction of the blood vessel by a foreign substance or a blood clot blocking the vessel. Something travels through the bloodstream, lodges in a vessel and plugs it. Blood clots are the most common cause of embolism. A pulmonary embolus is a blood clot that has been carried through the blood into the pulmonary artery (the main blood vessel from the heart to the lung), or one of its branches, plugging that vessel. The term “embolus” refers to the plug itself obstructing the blood vessel while “embolism” refers to the process by which this happens.

**Enteric** – of/or relating to the small intestine.

**Enteritis** - is the inflammation of the small intestine.

**Eosinophil** – is a type of white blood cell. The numbers of Eosinophils in blood often rise when there is an allergic reaction in progress. Eosinophilia is the formation and accumulation of an abnormally large number of eosinophils in the blood. Eosinopenia is a deficiency of eosinophilic cells in the blood.

**Granulomatous** - a granuloma is one of a number of forms of localised nodular inflammation found in tissues.

**Granulomatous Enteritis** - (Crohn’s Disease in humans) is a chronic inflammatory disorder, primarily involving the small intestine only. In mild form, it causes small, scattered shallow crater-like areas (erosions) called apthous ulcers in the inner surface of the bowel. In more serious cases, deeper and larger ulcers can develop, causing scarring, stiffness and possibly narrowing of the bowel, sometimes leading to obstruction.

**Granulomatous Peritonitis** - is a severe affect of Granulomatous enteritis in which deep ulcers puncture holes in the bowel wall, leading to infection in the abdominal cavity (peritonitis) and in adjacent organs.

**Hyperphosphatemia** – is higher than normal blood level of phosphate. Hyperphosphatemia is generally a condition in dogs with chronic or end stage renal failure. It is often a key diagnostic tool in evaluating renal function.

**Hypoproteinemia** – low blood protein in the blood. Sometime resulting in oedema and fluid accumulation.

**Inflammatory Bowel Disease (IBD)** - the term used to describe a group of chronic intestinal diseases characterised by inflammation of the bowel – the large or small intestine. (Also see Lymphocytic/Plasmacytic Enteritis and Lymphangiectasia below).
**Isosthenuria** - the excretion of urine with fixed specific gravity. It may occur in terminal renal disease when the specific gravity reaches that of the glomerular filtrate, 1.010.

**Lipogranulomatosis** - is a condition of faulty lipid (fat) metabolism in which nodules of lipid matter are deposited in the skin and mucosa, causing granulomatous reactions.

**Lymph** – an almost colourless fluid that travels through vessels called lymphatics in the lymphatic system and carries cells that help fight infection and disease. Lymphangitis involves the lymph vessels/channels, with inflammation of the channel and resultant pain and systemic and localised symptoms.

**Lymphangiectasia** - is obstruction and dilation of the lymphatic vessels in the digestive system. It is a congenital or acquired disorder of the lymphatic system resulting in fat and protein malabsorption and a protein losing enteropathy. It is another form of IBD that often underlies PLE in the SCWT.

**Lymphocyte** – a small white blood cell (leukocyte) that plays a large role in defending the body against disease.

**Lymphocytic-plasmacytic enteritis** – is a form of Inflammatory Bowel Disease. This form of IBD is one of the diseases that can develop into PLE. Although the exact cause is unknown, one favoured by most academicians is that this disease is an immune-mediated hypersensitivity to some enteric bacteria and dietary components. It is characterised by the presence of inflammation of the cells lining the intestine.

**Maelena (Melena)** - a darkening of the faeces by blood pigments. Typically the faeces have a black colour with a red tinge at the edges and are soft and almost slimy.

**Malabsorption** – is poor intestinal absorption of nutrients. This is caused by any blockage of properly digested nutrients. It may be a symptom of a number of diseases which manifest as PLE, or PLE and PLN.

**Maldigestion** - indicates the system is not properly breaking down nutrients. This is caused by a lack of Pancreatic enzyme. This is not generally a factor of either PLE or PLN.

This disorder is directly related to the excessive leakage of plasma proteins into the lumen of the gastrointestinal tract. 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.

**Mesenchyme** - is the meshwork of embryonic connective tissue in the mesoderm, from which are formed the muscular and connective tissues of the body and also the blood vessels and lymph vessels.

**Nephron** – one of a million tiny filtering units in each kidney. Each nephron is made up of both glomerulus and a fluid collecting tubule that processes extra water and wastes.

**Nephropathy** – is a medical word for kidney disease. Nephropathy can be applied to any disease of the kidney.

**Pancreas** – the organ that makes pancreatic juices and hormones, including insulin. Pancreatic juices, also called enzymes, help digest food in the small intestine. Insulin controls the amount of sugar in the blood. Both enzymes and hormones are
needed to keep the body working correctly. Pancreatitis is an inflammation of the pancreas.

**Proteinuria** – large amounts of protein in the urine. Some protein is normal in the urine. Too much means protein is leaking through the kidney, most often through the glomeruli. The main protein in human blood and the key to the regulation of the osmotic pressure of blood is albumin. Proteinuria is synonymous with albuminuria.

**Thrombus** - a clot in a blood vessel or the heart. The formation, development or presence of a thrombus is called Thrombosis.

**Titre** - tells if an animal has had an immune response to the material (antigen) in the vaccine and that it has made antibodies (which we measure in the titre). Immunologists have equated the presence of certain levels of titre to immunity.

**EXPLANATION OF BLOOD CHEMISTRY:**

Blood tests are often performed as a biochemistry profile, or chemistry panel, which is a collection of blood tests to screen several organs at one time. The makeup of a biochemical profile varies with the laboratory in which it is performed. The following are some of the most commonly performed chemical tests.

**Albumin** – is a small protein produced by the liver. Albumin acts as a sponge to hold water in the blood vessels. When blood albumin is decreased, the pressure created by the heart forcing blood through the blood vessels causes fluid to leak out. This fluid then accumulates in body cavities such as the abdominal cavity or in tissues as oedema.

Albumin is decreased if the liver is damaged and cannot produce an adequate amount of albumin or if albumin is lost through damaged intestine or the urine due to kidney disease. The only cause of increased albumin is dehydration.

**AG Ratio** – A ratio of albumin compared to globulin

**Alkaline phosphatase** – is an enzyme made by the biliary tract (liver), bone and placenta and normally present in high concentrations in growing bone and in bile. It originates from many tissues in the body. When alkaline phosphatase is increased in the bloodstream of the dog the most common causes are liver disease, bone disease or increased blood cortisol either because Prednisone or similar drug is being given to the pet because the animal has Cushing’s disease.

**Alanine Aminotransferase (ALT)** – is an enzyme normally present in liver and heart cells that is released into the bloodstream when the liver or heart is damaged. ALT is also called serum glutamic pyruvic transaminase (SGPT). Liver damage causes ALT to increase in the bloodstream. ALT elevation does not provide information as to whether the liver disease is reversible or not.

**Aspartate Aminotransferase (AST)** – is an enzyme normally in liver and heart cells. AST is released into blood when the liver and heart is damaged.

**Amylase** – is a digestive enzyme formed in the pancreas. Amylase helps the body breakdown sugars. In cases of pancreatitis high levels of amylase are found in the blood.
B/C Ratio (BUN/Creatinine Ratio) – is the ratio of BUN and Creatinine in the urine. This is a very important ratio for Wheatens, since an improper ratio is one of the key indicators of protein losing syndromes.

Bile acids – are produced by the liver and are involved in fat breakdown. A bile acid test is used to evaluate the function of the liver and the blood flow to the liver. Patients with abnormal blood flow to the liver, a condition known as portosystemic shunt will have abnormal levels of bile acids.

Blood Urea Nitrogen (BUN) or Urea – is nitrogen in the blood. This is a waste product produced by the liver from proteins from the diet, and is eliminated from the body by the kidneys. A low BUN can be seen with liver disease and an increased BUN is seen in pets with kidney disease. The kidneys must be damaged to the point that 75% of the kidneys are non-functional before BUN will increase. Pets that are severely dehydrated will have an increased BUN, as the kidneys of a dehydrated patient do not get a normal amount of blood presented to them, so the waste products do not get to the kidneys to be eliminated.

Bilirubin – is a yellow fluid produced when red blood cells break down. Bilirubin is further broken down and eliminated in both the urine and stool. Bilirubin is increased in the blood in patients with some types of liver disease, gallbladder disease or in patients who are destroying the red blood cells at a faster than normal rate (haemolysis). Large amounts of Bilirubin in the bloodstream will give a yellow colour to non-furred parts of the body, which is called icterus or jaundice. Icterus is most easily recognised in the tissues around the eye, inside the ears and on the gums.

Calcium – is a mineral found mainly in the hard part of bones. The body has hormones, which cause bone to release calcium into the blood and to remove calcium from the blood and place it back into bone. Abnormally high calcium in the blood occurs much more commonly than low calcium. High blood calcium is most commonly associated with cancer. Less common causes of elevated calcium are chronic kidney failure, primary hyperparathyroidism, which is over-function of the parathyroid gland, poisoning with certain types of rodent bait and bone disease. One cause of low blood calcium is malfunction of the parathyroid glands, which produce a hormone (PTH) that controls blood calcium levels. Animals poisoned with antifreeze may have very low blood calcium.

Carbon Dioxide (CO₂) – measures a buffer system in the blood. A normal CO₂ level keeps the blood acidity at the correct level.

Chloride – is the major anion found in the fluid outside of cells and in blood. An anion is the negatively charged part of certain substances. Elevations in chloride may be seen in diarrhoea, certain kidney diseases and sometimes in over activity of the parathyroid glands. Decreased chloride is normally lost in the urine, sweat and stomach secretions. Excessive loss can occur from heavy sweating, vomiting and adrenal gland and kidney disease.

Cholesterol – is the most common type of steroid in the body. Cholesterol is carried in the bloodstream as lipoproteins. Cholesterol can be increased in the bloodstream for many reasons in dogs. Some of the diseases that cause elevated cholesterol are hypothyroidism, Cushing’s disease, diabetes and kidney diseases that cause protein to be lost in the urine. High cholesterol does not predispose dogs to heart and blood vessel disease as it does in people.

Creatinine Phosphatase (CK) – is a muscle enzyme.

Creatinine – is a waste product in the blood that results from the normal breakdown of muscle. Healthy kidneys filter creatinine from the blood. An elevation
of creatinine is due to kidney disease or dehydration. Both creatinine and Urea (BUN), increase in the bloodstream at the same time in patients with kidney disease. An elevation of Phosphorus with Creatinine and Urea (BUN) indicate a long standing kidney problem.

**Electrolytes** – are related to fluid balance in your cells. They are especially important if you become dehydrated or have kidney problems. Electrolytes include sodium, potassium, chloride, and bicarbonate.

**Gamma Glutamyl Transpeptidase (GGT)** – is a liver enzyme. High level can indicate liver damage.

**Globulin** – measures the protein in antibodies produced by the immune system.

**Glucose** – is the sugar that is the chief source of energy. Glucose is considered a simple sugar. Found in the blood, it is the main sugar that the body manufactures. High glucose levels in the blood indicate diabetes. It may be mildly increased in dogs with Cushing’s disease. Glucose can temporarily increase in the blood if the dog is excited by having a blood sample drawn. Low blood sugar occurs less commonly and can be a sign of pancreatic cancer or overwhelming infection (sepsis). Low blood sugar can cause depression or seizures.

**Lactic Dehydrogenase (LDH)** – is an enzyme that is elevated if kidney, skeletal muscles, liver or myocardium is injured.

**NA/K Ratio** – A low sodium potassium ratio can be a very important indicator for Addison’s Disease, although it is possible to have a normal sodium and potassium values. Note: To confirm Addison’s disease you may require the ACTH Stimulation test.

**Phosphate (Phosphorus in USA)** – is an essential element in the diet and a major component of bone. Phosphorus in the bloodstream originates from bones. Phosphorus is increased in the bloodstream in patients with chronic kidney disease. Like BUN and creatinine, phosphorus increases in these patients when about 75 percent of both kidneys are damaged.

**Potassium** – affects several major organs including the heart. Potassium is increased in the bloodstream in the pet with acute kidney failure such as kidney failure caused by antifreeze poisoning, in dogs with Addison’s disease and in animals with a ruptured or obstructed bladder. Potassium is lost from the body in vomit, diarrhoea and urine. Pets that are not eating may have low blood potassium. Low blood potassium can cause the pet to feel weak.

**Sedimentation Rate or Sed Rate** – measures how quickly red blood cells settle in a tube of blood. A high sed rate indicates some type of inflammation.

**Sodium** – levels indicate your balance of salt and water. They also are a sign of the functioning of your kidneys and adrenal glands. Sodium may be slightly increased in the blood if the patient is dehydrated although many dehydrated dogs have normal blood sodium. Low blood sodium is most commonly seen with Addison’s disease.

**Total Protein (TP)** – protein includes albumin and larger proteins called globulins. Included in the globulins are antibodies, which are protein molecules. Total protein can be increased if the dog is dehydrated or if the pet’s immune system is being stimulated to produce large amounts of antibody. Total protein is decreased in the same situations which reduce albumin or if the pet has an abnormal immune system and cannot produce antibodies.
**Uric Acid** – comes from the breakdown of DNA (genetic material in the cells), the kidneys normally remove it. High levels of uric acid are fairly common. Very high levels can be caused when the kidneys are unable to remove uric acid from the blood or by leukaemia or lymphoma.

**EXPLANATION OF COMPLETE BLOOD COUNT (CBC):**

The complete blood count measures the number of cells of different types circulating in the bloodstream. There are three major types of blood cells in circulation; red blood cells (RBC); white blood cells (WBC) and platelets. Red blood cells are produced in the bone marrow, which is the soft centre of bones. RBC’s pick up oxygen brought into the body by the lungs, and bring that oxygen to cells throughout the body.

The complete blood count also includes a measure of haemoglobin, which is the actual substance in the red blood cell that carries oxygen.

**Basophils (Bas)** – are not well understood but they are involved in long-term allergic reactions such as asthma or skin allergies. It is a component of Granulocytes and is calculated as a % of WBC.

**Blood Cell Count (RBC)** – is the total number of red blood cells.

**Eosinophils (Eos)** – are normally 1% to 4% of WBC’s. They are involved with reactions to parasites (flea infestation); allergies; inflammation of the GI, urogenital or respiratory tract; or inflammation of the skin.

**Haemoglobin (HGB)** – is the actual carrier of the oxygen on the red blood cell. It is a measurement of the red cell mass.

**Hematocrit (HCT)** – measures the percentage of blood volume taken up by the red blood cells.

**Lymphocytes (lymphs)** – are white blood cells produced in the lymph glands of the body. Lymphocytes fight infection and produce antibodies against infectious agents.

**Lymphopenia** – is a decrease in the number of proportion of lymphocytes (one of the white blood cells) in the blood.

**Mean Corpuscular Haemoglobin (MCH) and Mean Corpuscular Haemoglobin Concentration (MCHC)** – measure the average concentration of haemoglobin in erythrocytes. The MCH is calculated by dividing total haemoglobin by the total number of red blood cells.

**Mean Corpuscular Volume (MCV)** – measures the average volume (size) of individual red blood cells. A low MCV means that the cells are smaller than normal. This is usually caused by an iron deficiency or chronic disease.

**Mean Platelet Volume (MPV)** – is a measurement of the average size of platelets found in blood.

**Monocytes or Macrophages (Monos)** – make up 2% to 8% of WBC’s. They fight infection by “eating” germs and telling the immune system what germs they have found.
Neutrophils or polymorphonuclear cells (Polys) – is the most common type of white blood cells and cause the body to fight bacterial infections. Neutrophils can be decreased in pets with bone marrow disease, in some viral diseases and in some pets receiving cancer chemotherapy drugs. Neutrophils are increased in pets with inflammation or infection of any part of the body and in pets receiving Prednisone or other cortisone type drugs.

Packed Cell Volume (PCV) – is another measure of red blood cells. A small amount of blood is placed in a tiny glass tube and spun in a centrifuge. The blood cells pack to the bottom of the tube and the fluid floats on top. The PCV is the percent of blood that is cells, compared to the total volume of blood. In normal dogs, 40–50% of the blood is made up of blood cells and the remainder is fluid.

Platelets (PT) – are the third type of blood cell examined in CBC, which are produced in the bone marrow and are involved in the process of making blood clot.

Red blood cells (RBC) – are produced by the bone marrow, and are responsible for carrying oxygen throughout the body. This is measured by three main tests.

Red Blood Cell Distribution Width (RDW) – is a measure of the variation of red blood cell (RBC) width that is reported as part of a standard complete blood count.

White blood cells (also called leukocytes) – are produced in the bone marrow and are important for the immune system as they help fight infections in the body.

White Blood Cell Count (WBC) – is the total number of white blood cells. A high WBC usually means that the body is fighting an infection. A very low WBC can be caused by problems with the bone marrow.
Urinalysis:

Explanation of Urinalysis:
A urine sample can provide information about several organ systems. The concentration, colour, clarity and microscopic examination of the urine sample can provide diagnostic information.

Urine may be obtained by catching a sample during normal urination, or alternatively, by your vet passing a catheter into the bladder or by placing a small needle through the body into the bladder, a procedure called cystocentesis.

Depending upon why the urine sample is being collected, one collection method may be preferred over another. Enquire at the time you make an appointment for veterinary care if a urine sample may be collected. Preventing your pet from urinating prior to the appointment will assure that your pet’s bladder will contain urine for sampling.

The following refers only to the Urine Protein and Creatinine, a common urinalysis performed on the Wheaten Terrier.

Urine Protein - Proteinuria is excess protein in the urine. Some protein is normal in the urine. Too much means protein is leaking through the kidney, most often through the glomeruli. The main protein in human blood and the key to the regulation of the osmotic pressure of blood is albumin. Proteinuria is synonymous with albuminuria.

Creatinine - Creatinine is a chemical waste molecule that is generated from the muscle metabolism. Creatinine is produced from creatine, a molecule of major importance for energy production in muscles. Approximately 2% of the body’s creatine is converted to creatinine every day. Creatinine is transported through the bloodstream to the kidneys. The kidneys filter out most of the creatinine and dispose of it in the urine.

Although it is a waste, creatinine serves a vital diagnostic function. Creatinine has been found to be fairly reliable indicator of kidney function. As the kidneys become impaired the creatinine will rise. Abnormally high levels of creatinine warn of possible malfunction or failure of the kidneys, sometimes even before symptoms are evident.

Urine Colour – Normal colour is yellow to amber. Red is caused by blood, dark yellow to brown is caused by Bilirubin, reddish brown is caused by haemoglobin/myoglobin.

Urine Transparency – Normal is clear. Cloudy urine is caused by crystals, bacteria, cells, blood, mucous or casts.

Urine Specific Gravity (USG) – Specific gravity is a measurement of how concentrated the urine is. Renal impairment and diabetes insipidus affect a dog’s ability to concentrate urine.

Urine Protein - is usually only seen in trace amounts as the kidney normally does not allow protein to get through. When protein is present it indicates damage to the kidneys.

Urine sediment - can be examined for solid material such as cells, bacteria, crystals and casts. Red blood cells in the urine indicate inflammation, certain tumours or blood clotting disorders. White blood cells and bacteria are seen in infection. Crystals may be normal or may indicate infection, liver disease, toxin ingestion or bladder stones.
**Glucose** - should not be seen in the urine. However, when blood sugar levels become very high as with diabetes mellitus, it exceeds the kidney’s capacity to keep sugar out and glucose is seen in the urine.

**PH Levels** – should be a little on the acidic side, i.e. 6.2 – 6.5

**The Animal Health Trust (AHT)**

**Guide to Interpretation of Urine Protein/Creatinine Ratio (UPC)**

Results of <0.5: Normal Result.

Results of <0.5 – 1.0: Indeterminate result. May or may not reflect abnormality.

Results of >1.5: Abnormal. Consistent with loss of protein within urinary system. Considerations should include pre-glomerular, glomerular or post-glomerular causes.

Results of >5.0: Abnormal. Elevations of this degree are often associated with primary glomerular disease. Correlation with serum biochemistry. Urinalysis and clinical findings is recommended.

Results of >13.0: Abnormal. Elevations of this degree are often associated with renal amyloidosis or severe glomerulonephropathy. Correlation with serum biochemistry, urinalysis and clinical findings is recommended.

Further Reading regarding UPC:

https://ahdc.vet.cornell.edu/sects/clinpath/test/urine/protein.cfm
GENETICS

We hope that the following information which includes a two page chart and glossary of terms will help you to understand the complex and complicated subject of Genetics.

DNA:
DNA is a long fine fibre made up from two strands that stick together with a slight twist to form a helix shape.

DNA is found in cells and is organised into stretches of genes where the base proteins attach to coil the DNA to fit into each cell, giving rise to structures known as chromosomes.

Along these stretches are instructions to ‘turn a gene on’ and ‘turn a gene off’; and large stretches whose purpose is not even known.

Genes:
Genes are made from Deoxyribonucleic Acid (DNA). DNA is made up of four nucleotides which are individual chemical structures known as bases. These four nucleotides are, Adenine ‘A’, Thymine ‘T’, Cytosine ‘C’ and Guanine ‘G’, they are joined end to end.

Genes carry the instructions or ‘plans’, for the making of thousands of proteins that are found and deciphered by the cell. The random combination of these bases determines what the cell will look like and what job that cell will do and how the many different cells of the body will be arranged.

Each cell has a nucleus containing 78 chromosomes, the exception to this being red blood cells (which have no chromosomes), and the reproductive cells, eggs and sperm (which have 39 chromosomes each). For example, how to make haemoglobin; haemoglobin is the protein that carries oxygen around the bloodstream. The body needs to constantly make haemoglobin.

Chromosomes:
A chromosome is made up of DNA and the proteins attached to it. Each chromosome has a thread of DNA running along its length and the genes are arranged along this thread.

This resembles beads on a string. Chromosomes are arranged in pairs and in each cell of a dog there are a total of 78 chromosomes; 39 from the sire and 39 from the dam. These are 38 pairs of autosomes and two chromosomes involved in specifying sex, i.e. X or Y chromosomes.

Sex determination in canines is exactly the same as in humans; bitches have two X chromosomes whilst the dog has one X and one Y chromosome.

Each set of 39 chromosomes contain approximately 20,000 genes, representing a sequence of 3 billion bases. These 20,000 different genes are required to specify the dog.
Gene Mutations:

The ‘plan’ embedded in the gene can become altered by a process called mutation. This can involve change in the sequence of the bases by adding or removing some of the base sequence within the gene. Considering the times a gene has to copy and reproduce itself it’s not surprising that mistakes (mutations) can occur.

*On the first ‘ladder‘ is a ‘normal’ strand of DNA. The other three show various mutations, as indicated by the boxes. On the second strand, a substitution has occurred, changing a base pair. On the third strand, a deletion has occurred, removing a base pair. On the fourth strand, an insertion has occurred so there is an extra base pair in the sequence. These mutations can cause changes in amino acid sequences.*

Consequences of gene mutations:

This depends on the gene in which the mutation has occurred. Some mutations are silent and have no consequences; others affect the gene so that the plan can no longer be used to make a functional protein. In the Wheaten this could be the effect the mutated gene has on kidney formation, the consequence being Renal Dysplasia (RD). Once a mutation has occurred within a gene, it is fixed and cannot be reversed. The dog carrying the mutation will pass this mutant gene onto its offspring, if the consequence of the mutation is a disease state, like RD, then this is an inherited disease.

**Note:** Not all mutations are bad (deleterious), occasionally, some mutations can be beneficial. This is how evolution has progressed to make the individual fitter and enabling them to have the advantage in their environment.

There are two types of mutation that can occur in genes and the different effects are determined by the fact that dogs have two copies of every gene.

If a recessive mutation occurs in a gene the effect is not initially noticed because the second, normal copy of the gene masks the presence of the recessive mutant gene. A disease caused by a recessive mutant will only be seen in a dog that has two copies of the recessive mutant.

If a dominant mutation occurs the consequences will be felt despite the fact that there will also be a normal gene present. An animal that inherits a dominant mutation will be affected.
Inheritance and genetic mutations:

**Autosomal Dominant Trait:**
Both parents do not have to have the gene for the disorder to cause the trait to occur. However, since the trait is expressed in the heterozygous state, one parent must show the trait in order for it to occur among the offspring. There are few exceptions to this rule. At the present time it is not known why a dominant gene masks or hides the recessive alleles and it may be that the concept of dominance is operational and may not reflect any intrinsic property of the gene. Nevertheless, the fact that dominant traits are expressed in certain ratios can be easily demonstrated.

The general characteristics of an autosomal or simple dominant trait follow:
1. The gene is located on any one of the thirty-eight pairs of autosomes.
2. The gene is generally present in the heterozygous state.
3. At least one parent of an affected offspring must show the trait, unless a new mutation is involved.
4. The trait occurs in successive generations (no skipping).
5. About 50% of the offspring of an affected dam or sire will also be affected.
6. On the average males and females are equally affected.
7. Dogs that are phenotypically normal are also genotypically normal.

**Autosomal Recessive Inheritance:**
The general characteristics of an autosomal simple recessive trait follow:

1. The gene is located on any one of the 38 pairs of autosomes.
2. To be expressed (to show the trait) the gene must be present in the homozygous state (both genes must be identical).
3. The trait tends to occur in one generation and then skips one or two generations until carrier descendants are again mated allowing the genes to be expressed.
4. Each of the parents of an affected puppy is a proven carrier (heterozygote) of the abnormal gene but generally show no phenotypic manifestation of the trait.
5. If the given trait is rare in a breed (one affected amongst 2,000 or 3,000 normal dogs) there may be increased inbreeding among the parents (increased consanguinity) of affected dogs.
6. Matings between heterozygotes (carriers), on average, produce 25% affected (homozygous recessive), 50% carriers (heterozygous) and 25% that do not have the mutant gene (homozygous dominant or wild type).
7. On the average males and females are affected equally.

**Polygenic Trait:**
The general characteristic of a polygenic trait follow:

1. As with a recessive trait, both the sire and the dam must contribute one or more of the genes that cause the abnormal phenotype in the offspring.
2. Unlike recessive traits, the contribution from the sire and dam need not be equal.
3. Since we do not know the number or the specific effect the genes involved in polygenic traits have in dogs, no predictable Mendelian ratios are associated with these traits.
4. Both sexes are affected with polygenic traits (excluding sex-limited traits) but not necessarily in equal numbers.
5. The trait may skip generations and may appear to be erratic in occurrence.

"...there is no hope for control without knowledge."
Dr George Padgett DVM, Professor of Pathology at Michigan State University.
INHERITANCE OF AUTOSOMAL MUTATIONS

Dominant Mutation:

Affected

Clear

50% Affected Offspring

50% Clear Offspring

Recessive Mutation:

Carrier

Clear

50% Carrier Offspring

50% Clear Offspring

Carrier

Carrier

25% Affected Offspring

50% Carrier Offspring

25% Clear Offspring
INHERITANCE OF AUTOSOMAL MUTATIONS

Recessive Mutations Cont:

Note: The figures quoted are, in all probability, estimates. Reality can be different. In principle if you flip a coin it has a 50% chance of coming down 'heads' and 50% chance of coming down 'tails'. So the proportion of offspring in individual litters can differ from the expected outcomes given above.
Glossary of Genetic Terms:

**Alleles** – One of two or more alternative versions of the same gene.

**Amino acids** – One of the chemical compounds that are the building blocks of proteins.

**Autosomes** – The name given to all chromosomes other than the two involved in determining the sex of an individual (the X and Y chromosomes). The dog has 38 pairs of autosomes and one pair of sex chromosomes.

**Bases** - There are four bases which join together to form DNA, Adenine, Guanine, Thymine and Cytosine, identified by their initials A, G, T and C. The bases join end to end to give a molecule of DNA. These bases join in a specific sequence and it is this base sequence that represents the genetic plan.

**Candidate gene** – A gene involved in a particular inherited disease in the dog which has been identified because the same gene is known to be the cause of a similar disease in man or other animals.

**Carrier** – With regard to hereditary disease this is a dog that carries a recessive, mutant allele that is matched by the presence of a normal allele. On average, it will pass on this mutant allele to half of its offspring.

**Cells** – One of the tiny living units from which organisms are made.

**Cell membrane** – The thin protective membrane that surrounds a cell.

**Characteristic** – A feature such as brown or blue eyes.

**Chromosome** – This is the body that carries the DNA within the nucleus. A thread of DNA runs along the length of each chromosome carrying individual genes.

**Code** – Cells use the genetic code to convert the DNA’s sequence of bases into a sequence of amino acids.

**Congenital** – Present at birth. May be inherited, but not necessarily.

**Cytoplasm** – The thick fluid that forms most of the inside of a cell.

**DNA (Deoxyribonucleic Acid)** – The chemical found in the nucleus of a cell that makes up chromosomes and genes. DNA consists of two chemical strands which twist around each other in the form of a helix. Each strand is made up by the joining together of the chemical units called bases.

**DNA Sample** – DNA can be collected in a number of ways. The most common methods used with dogs are by blood sample or a scraping of cheek cells, this is called a buccal sample.

**Dominant Mutation** – A mutation that can express itself when present only as a single copy, even in the presence of a normal allele.

**Effective Population Size** – The number of breeding animals in a hypothetical population that would deliver the same rate of inbreeding as the population in question.
**Enzyme** – A type of protein found in the body that greatly speeds up the rate of chemical reactions inside and outside cells.

**Gamete** – A reproductive cell. At fertilisation, the male gamete (the sperm) and female gamete (the egg), unite and the genetic material combines.

**Gene** – A part of the DNA which controls the hereditary characteristics of an organism. Individual genes consist of a unique sequence of about 2000 bases which permits the cell to make a particular protein. Each individual has two sets of genes (one set from each parent) and passes this on to each of its offspring.

**Genetic** – Describes something to do with genes and inheritance.

**Gene pool** – All of the genes that exist within an inbreeding population.

**Genome** – A complete set of chromosomes, i.e. genes within a living organism.

**Genotype** – The genes found in the cells on an individual. The genetic makeup of an individual will influence the appearance of phenotype of the individual.

**Heritability** – The transmission, or passing on, of features controlled by genes from both parents to their offspring. The proportion of phenotypic variation that is due to genetic variation.

**Heterozygous** – An individual that has two different alleles of a gene for a particular characteristic. If one allele is recessive and the other dominant, then the effect caused by the dominant allele will be apparent.

**Homozygous** – An individual that has identical alleles for a particular characteristic. Recessive characteristics will only show if an individual is homozygous for that characteristic.

**Inbreeding** – The breeding of individuals more closely related than average in the population.

**Locus** – Position on matching maternal and paternal chromosomes at which alleles of the same gene are found.

**Marker** – A component of a genetic map which uniquely identifies a locus.

**Maternal** – Something belonging to, or coming from, the mother (dam)

**Microsatellite** – A region of DNA which possesses an unusual base sequence where, two, three or four bases are continually repeated.

**Monogenic** – A characteristic controlled by a single gene.

**Mutation** – A change in the base sequence of DNA caused by an error in copying or some other factor. A mutation may be passed onto offspring.

**Nucleus** – The control centre of the cell which contains the chromosomes.

**Paternal** – Describes something belonging to, or coming from the father (sire).

**Phenotype** – The phenotype is the physical expression of an individual’s genotype. Observable, or measurable, properties of an organism, e.g. hip score, weight.
**Polygenic** – Descriptive of a trait which is under the control of many genes.

**Protein** – One of a group of chemical substances that build and run cells. Proteins are built of amino acids using instructions encoded in genes.

**Recessive Mutation** – A mutation that is masked by the presence of a normal counterpart. These are only expressed when there are two copies of the mutation.

**Selection** – The process of varying relative individual reproductive success in propagation of a population.

**Sex chromosomes** – Chromosomes involved in determining the sex of the animal, i.e. females have two X chromosomes and males possess one X and one Y chromosome.

**Sex linked inheritance** – Inheritance of characteristics that are determined by genes present on either the X or Y chromosome.

**Somatic** – All cells in the body apart from the reproductive cells (gametes).
OTHER CONDITIONS

This section of the Health Handbook describes conditions that can affect dogs in general. Some of these conditions have very occasionally been diagnosed in the Wheaten Terrier but are not thought to be hereditary.

TOPICS:

- Cushing’s Disease
- Deafness
- Degenerative Myelopathy/Failing Back Legs
- Ectopic Ureter & Vulvovaginal Stenosis
- Eyes
- ‘Gulpies’
- Hips
- Luxating Patella
- Skin Conditions
Endocrine System:

Include the pancreas, thyroid gland, parathyroid glands and adrenal glands. Diseases of the endocrine system may lead to the production of too much or too little hormone.

Adrenal Glands:
The adrenal glands are in close proximity to the kidneys. The outer portion of the adrenal glands are located on top of each kidney, this is called the adrenal cortex.

The adrenal cortex produces, among other things, steroid hormones which regulate carbohydrate and fat.

Cushing’s Disease:
Cushing’s disease is the common name for Hyperadrenocorticism, (Addison’s Disease is Hypoadrenocorticism).

Cushing’s Disease is caused by a hyperactive adrenal gland that secretes too many glucocorticoids, (steroids), into the bloodstream. The adrenal gland produces a wide range of hormones and Cushing's can cause the overproduction of any one or more of them. The symptoms of the disease vary widely and because of this it is difficult to detect, however this is a treatable disease.

There are three basic causes of Cushing’s disease:

1. A tumour in the adrenal gland
2. A tumour in the pituitary gland
3. Medically induced by administration of long term cortisone drugs. These medications are used to treat a variety of illnesses in dogs.

About 85% of dogs with Cushing’s have an overactive pituitary gland which is a small pea sized gland in the brain producing an excessive secretion of the hormone ACTH. This in turn over stimulates the adrenal glands and produces an excess of cortisol. The majority of the remaining cases result from adrenal tumours. Approximately 50% of these adrenal tumours are benign.

Signs & Symptoms:

- Increased/excessive water consumption (polydipsia)
- Increased/excessive urination (polyuria)
- Urinary accidents in previously housetrained dogs
- Increased/excessive appetite (polyphagia)
- Appearance of food stealing/guarding, begging & scavenging
- Sagging, bloated, pot-bellied appearance
- Weight gain or its appearance, due to fat redistribution
- Loss of muscle mass, giving an appearance of weight loss
- Bony, skull-like appearance of the head
- Exercise intolerance, lethargy, general hind leg weakness
- Reluctance to jump on furniture or people
- Excessive panting, seeking cool surfaces to rest on
- Symmetrically thinning hair or baldness (alopecia) on the body
- Dullness and dryness to coat
• Slow re-growth of hair
• Thin, wrinkled, fragile and/or darkly pigmented skin
• Easily damaged/bruised skin that heals slowly
• Hard calcified lumps in the skin
• Susceptibility of infections (especially skin or urinary)
• Diabetes, pancreatitis, seizures

**Diagnosis:**
Cushing’s disease is difficult to diagnose, there is no single test to identify it. Vets generally undertake several blood and urine tests to compare the results to normal levels. They may follow up with x-rays and/or ultrasound to reveal the presence or absence of a tumour.

**Treatment:**
This depends how severe the symptoms are and on the general health of the animal. It can be treated both surgically and medically. These two options are, surgically removing the tumour (if one is present), and the prescribing of medications that slow down the adrenal gland. The majority of dogs are treated medically.

**Deafness:**
In 1997 and 1998 a very small number of Wheatens were identified with varying degrees of deafness.

Out of 81 Wheatens tested:
• 5 had a hearing defect
• 8 had an ear infection

This was confined to a group of dogs who had a common male ancestor, Harwelden Casey No, on both sides of their pedigree. Geneticist Dr Bruce Cattenach felt it was possibly hereditary and the mode of inheritance likely to be an autosomal recessive gene.

As a result, the Committee of the SCWT Club of GB obtained professional guidance from Celia Cox, BvetMed, Cert VR, FRCVS who carried out the BAER (Brainstem Auditory Evoked Response) test on the hearing impaired dogs and their close relatives. It is still encouraged to have puppies of approximately 6 weeks of age BAER hearing tested if the pedigree has this male ancestor on both sides.

*However, the last reported cases of deafness or hearing impairment to the SCWT Club of GB were in 1998.*
Degenerative Myelopathy (DM)/Failing Back Legs:

Degenerative Myelopathy (DM) is a disease that causes progressive deterioration of the spinal cord in older dogs, eventually resulting in total rear end paralysis.

Symptoms:

- Loss of coordination (ataxia) in the hind limbs
- Wobbling when walking, rear feet dragging and ‘knuckling’ of toes, worn nails
- Hind end weakness (failing back legs), tremors of rear legs
- Difficulty rising, walking up steps, getting in the car, or squatting to defecate
- Weakness can initially occur in one hind limb but both will become affected
- Loss of urinary and faecal continence
- Weakness to front legs

This condition is associated with a number of breeds and Wheatens can also be affected with this condition.

It is thought to be genetic in nature, being caused by a gene mutation, and a DNA test is now available to identify this gene following research carried out by the Missouri College of Veterinary Medicine by Dr Gary Johnson and others.

In Spring 2009 an article published in “Wheaten Health News” by The Soft Coated Wheaten Terrier Club of America (SCWTCA), stated that as part of the DM research at Missouri, DNA samples from 29 SCWT’s were tested with none affected. i.e. carrying two copies of the mutated gene. Of the sample, 5 (17%) had one copy and may be considered to be carriers. The remaining 24 Wheatens tested as normal, with no mutated copies of the gene present.

Because this test has not been validated for Wheaten Terriers only further genetic testing of affected dogs will help to verify the validity of it.

The article also indicated that research was continuing in an attempt to determine if environmental or other factors may also be involved in the development of DM.

The full article and updates can be found at: http://www.scwtca.org/health/healthnews.htm

2017 News University of Missouri clinical trial for CDM:
http://www.veterinarypracticenews.com/canine-degenerative-myelopathy-test-moves-toward-trial/

If your dog is diagnosed with DM or ‘Failing Back Legs’, then please inform your Breeder and Club so that records may be kept to monitor this condition.

Further reading on this condition:

http://www.fitzpatrickreferrals.co.uk/neurology/canine-degenerative-myelopathy/
http://www.laboklin.co.uk/laboklin/showGeneticTest.jsp?testID=8158D
**Ectopic Ureter:**

A small number of Wheatens have been born with congenital Ectopic Ureter. This condition can affect one or both Ureters. An Ectopic Ureter bypasses the bladder and can open into the urethra, vagina or uterus. Any of these malformations result in the puppy constantly dribbling urine.

This condition is present from birth so the problem may not be noticed at first as the mother constantly cleans the puppy. Many puppies have problems house training and can have bladder infections.

**Signs of an Ectopic Ureter are:**

- Almost exclusively diagnosed in females.
- Incontinence i.e. urine leaking or dribbling at times but normal urination at other times.
- Frequent urination.
- If infection is present blood tinged urine can sometimes be seen.
- Excessive licking of the genital area. Often the urine leakage will cause a rash in this area.

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**Normal Anatomy**

The kidney is made up of very small filters called nephrons, these filter blood and the final waste result is urine. The tube that carries urine from the kidney to the bladder is called a Ureter.

The urine is stored in the bladder until the dog needs to expel it to the outside via the urethra.

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**Anatomy showing Ectopic Ureter**

This diagram shows the right Ureter opening into the bladder as normal but the left Ureter is totally bypassing the bladder and entering via the Urethra.
Anatomy showing Ectopic Ureter

This diagram shows the abnormal Ureter separated from the bladder and only connecting at the Urethra

At the neck of the bladder is a valve called the urinary sphincter, which controls the release of urine. Dogs that have Ectopic ureters can also have weak urinary sphincters.

Although surgery is possible for ectopic ureters, in many cases it is not successful and the puppy continues to leak urine.

Sadly, in extreme cases this condition usually results in the euthanasia of the puppy.

Vulvovaginal Stenosis:
A less common cause of incontinence in female dogs is call vulvovaginal stenosis. It is a condition in which the vagina at the level where the urethra ends is narrowed. Occasionally when the bitch urinates, some urine will be trapped in the vagina in front of this narrowed area. When the dog rises from lying down the urine seeps out. This condition can be diagnosed by veterinary examination. In some dogs the narrowing can be stretched under anaesthesia. The incontinence may or may not resolve as sometimes other defects are also present.
Eyes:

Eye testing is recommended; especially for breeding stock. Breeders usually test their litter of puppies at about 6 to 8 weeks old. An eye certificate is current for one year. Ophthalmic Vets perform this test; and you can find one in your area by contacting the BVA (whose details are at the back of this Handbook).

The SCWT Club of GB hold BVA eye testing sessions at some Fun Days, check their web site for details. Testing sessions are also advertised by Dog Societies and Clubs in the weekly Dog Press.

The following conditions have occasionally been found in Wheaten Terriers, therefore, breeders and owners should be constantly vigilant.

**Retinal Folds:** The small 'folds' are found on the retina at about 5 weeks of age to approximately 4 months. It is important for breeders to eye test their puppies between the ages of 6 to 8 weeks. The folds can 'flatten out' and may not be detected later than this. It is recommended by the SCWT Club of GB that any puppy diagnosed as having 'folds' should not be bred from.

**Persistent Pupillary Membranes – PPM:** These are remnants of a foetal structure called the pupillary membrane. This membrane covers the pupil before the puppy is born. Normally the pupillary membrane is partially present and continues to disappear as the puppy develops.

Absorption may not be complete when the puppy’s eyes first open at about 10-14 days old and a small web like structure can be seen across the pupil. This usually disappears by the time the puppy is 4-5 weeks of age. In some breeds these strands never disappear and become PPM. PPM’s seem to be insignificant in the Wheaten and do not appear to affect their eye sight.

**Progressive Retinal Atrophy (PRA)** is the name given to a group of hereditary retinal diseases in dogs. There are several classifications of the disease according to the age of onset of the diseases and the types of retinal pathology which occur. PRA is not painful but the loss of sight is permanent.

Wheaten’s have occasionally been reported with PRA. In breeds that have been investigated in sufficient detail, the mode of inheritance appears to be simple autosomal recessive.

PRA affects the retina (the ‘film’ in the camera). It occurs in both eyes simultaneously and results in the degeneration of the rod and cone cells in the retina.

Owners may notice their dog bump into objects, especially in a dimly lit room. This progresses to night blindness and usually within months, with a loss of daylight vision as well. Night blindness is first noticed because the rods (the cells which allow vision in reduced light) degenerate before the cones (the cells which allow vision in bright light). The dog will frequently have dilated pupils and the owner may notice increased shininess at the back of the eye.

Dogs with PRA can develop cataracts later as the disease progresses.

Most dogs adjust well to vision loss, they are usually happy as long as their routine is stable. It is more difficult for them if their surroundings become unfamiliar.
Microphthalmia - This condition is apparent in pups once their eyes have opened. It can be mild or severe. A defect early in development results in the smaller than normal eye (microphthalmia). Affected dogs have prominent third eyelids and small eyes which appear recessed in the eye socket (enophthalmos). This is often associated with other eye abnormalities, including defects of the cornea, anterior chamber, lens and/or retina. At worse puppies can be blind, or may have cataracts which may be progressive, resulting in worsening vision.

Microphthalmia is also seen with coloboma – a cleft in a portion of the eye.

Breeding advice – Parents, normal eyed siblings, and affected dogs should not be bred from.

Research - The Finnish Kerry Blue and Wheaten Terrier Club have published an article written by Veterinary Ophthalmologist, Marjukka Sarkanen, who has given permission to reproduce part of the text (see below). The article is in Finnish but there are images of Wheaten puppies with this condition. There is a link to the document on the WHI Web site: http://www.wheatenhealthinitiative.com/Pages/healtheyes.html

This following translation has the second and third pages omitted, since they mainly contain specific information on how Finnish breeders should act if they should have a litter with this problem.

From the Breeding Committee of the Finland Kerry Blue and Soft Coated Wheaten Terrier Club: http://www.kerryvehna.net/

“…. Ocular anomalies and Microphthalmia found and reported in a Finnish SCWT litter. The puppies’ eyes seemed abnormal and the eyeballs small (see pictures). They were checked by an eye-specialist, who diagnosed the puppies with various ocular anomalies, e.g. microphthalmia, coloboma and PPM’s (Persistent Pupillary Membrane).

The puppies were practically blind, and had to be put to sleep. The breeder of the litter passed the information to the Breeding Committee. Blood samples taken from the sick puppies, their siblings and parents were sent to the Canine Genetic Studies group led by Prof. Hannes Lohi (www.koirangeenit.fi).

There have been rumours of similar litters in Canada, Sweden and The Netherlands. In Finland, this was the first litter brought to the attention of the Breeding Committee. In 1995, a research article published in The Netherlands reported a similar syndrome in two closely related SCWT litters (Van der Woerdt, A. Stades, F.C. Linde-Sipman, J.S van der Boeve, M.H. 1995. “Multiple ocular anomalies in two related litters of Soft-Coated Wheaten Terriers” Veterinary and Comparative Ophthalmology.

The Finnish Breeding Committee strongly recommends that all puppies should have their eyes examined, even if there are no abnormalities visible…”

(The rest of the document mainly gives instructions how to proceed if something is indeed wrong).

Your Vet may recommend visiting a Specialist Veterinary Ophthalmologist.

Further Reading about Microphthalmia:
http://discoveryspace.upei.ca/cidd/disorder/microphthalmia-ocular-dysgenesis
‘Gulpies’:

This is a digestive condition which can occur in any breed of dog and is commonly referred to as ‘Gulpies’.

**What are ‘Gulpies’?**

Dogs with ‘Gulpies’ have uncontrolled licking of their lips and also gulp in air, they may also vomit and can be quite distressed. When the dog has one of these episodes it wants to eat anything; grass, leaves, twigs, paper, carpet, dog blankets/beds etc., in a frenzied manner.

It is **important** you **stop** your dog ingesting any of these or similar items!

It appears to happen more frequently after food; it could be Gastro Intestinal (GI) a build-up of ‘gas’ and/or acid. The medical term for gas build-up is Aerophagia. For some dogs the cause may be an allergy to certain foods.

**Some suggested treatments:**

- Slippery Elm Capsules - dosage 2 capsules.
- Slippery Elm Powder – ½ teaspoon per 10 pounds of body weight.
- Homeopathic Nux Vomica - Three of the Nux 30C, (do not touch with your hand), crush between two spoons and put into the pouch at the side of the mouth. No food or drink 10 minutes before or after. Repeat if needed
- Pepto Bismol
- Sulcrate
- Marshmallow root

It also may help by lightly rubbing the throat and tummy.

**Articles** – Two informative articles are:


**Videos** - YouTube has quite a number of videos showing dogs with ‘gulpies’:

[http://www.youtube.com/watch?v=tpYAC9IfZjo](http://www.youtube.com/watch?v=tpYAC9IfZjo)

**Facebook Support Group** – ‘Dog Gulping Disorder Awareness & Owner Support’ [https://www.facebook.com/gulpydogs/about/](https://www.facebook.com/gulpydogs/about/)
Hips:

Hip Dysplasia is a term which describes developmental and other abnormalities involving the hip joint.

Genetically it is complex, and it can also be caused by environmental factors; an injury, or if a puppy is exercised too much, too soon, and allowed to run up and down stairs and jump of beds and/or furniture etc.

To perform a hip X-ray a Vet usually anaesthetises the dog so that there is no movement during the procedure. Some Vets are now performing hip scoring using sedation rather than full anaesthetic.

In the UK, the X-ray is sent to the British Veterinary Association (BVA), where specialists examine each hip and give it a number (score). The panel meet regularly, but the score result can take up to 8-12 weeks before notification is returned to the submitting Vet.

Please note the following:

- Hip scoring is only required once in a dog’s lifetime.
- Hip scoring should only be undertaken on dogs over the age of 12 months, there is no upper age limit.
- Hip scoring should be undertaken if you are using your Wheaten for breeding.
- To check if the dog is in good health, the Vet may also undertake a blood and urine test prior to this procedure.
- If a bitch is to be scored, it is thought by many that this is best undertaken as near to the mid-point between her seasons, otherwise the change in hormone levels could possibly result in a higher score.

The minimum best score per hip is zero, the maximum is 53, and this gives a total range of 0-106. The SCWT breed mean score in the UK is about 13.

BVA - full and up to date information and documents available on the BVA website whose details are at the back of this Health Handbook.

Information for Other countries:

There are different methods for scoring hips, so you need to check with your own Breed Club and/or Kennel Club for full details.

The Table below gives an approximate correlation between different schemes:

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<td>0</td>
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<td>Good</td>
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<td>A2</td>
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<td>Borderline</td>
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<td>B2</td>
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Luxating Patella:

Luxating Patella is a condition which can affect dogs in general, in particular Toy Breeds.

Very occasionally a Wheaten has received this diagnosis, but it is not thought to be hereditary.

It can be caused by a congenital abnormality but some cases of the condition can be the result of environmental causes; such as, an accident, or over exercising a young puppy!

It is therefore important that young puppies are not allowed to run up and down stairs, and jump off furniture. Wheaten puppies should be exercised for no more than 10 minutes, twice daily until 6 months of age, and 20 minutes, twice daily, up to 12 months. After this, the bones and joints should have matured enough to take normal exercise.

Clinical signs:

- Lameness
- A skipping gait
- Pain
- Stiffness of the hind limb
- Some dogs show only a single sign, whereas others show many signs of the condition
- Failure to treat the condition could lead to progressive debilitating arthritis of the joint

The patella (commonly known as the kneecap) becomes displaced from its normal position which is over the centre of the lower part of the thighbone. The patella slides up and down in a groove in the femur, or thigh bone, when the knee bends or extends. If the patella is not positioned correctly, the leg cannot function properly.

The problem is caused by an abnormal development of the bones and joints when the dog is growing, especially an abnormal position of the tibia. This leads to a more than normal wear and tear of the joint and will always lead to arthritic changes in the long term. It can also cause other problems of the knee joint, such as torn cruciate ligaments.

Treatment:

An operation will be required to correct the position of the patella. Dogs usually recover quickly and can bear weight on the leg within 2 weeks. Arthritis will develop in the long term whether surgery is performed or not.
Skin Allergies:

Like any dog, Wheaten can be prone to allergies, itchy skin, in particular, excessive biting, licking and nibbling of the paws.

**Atopic Dermatitis** – an allergic skin disease of dogs, known as canine atopic dermatitis, is caused by the dog’s immune system hypersensitivity to common substances in the environment such as dust mites or moulds.

The signs of atopic dermatitis usually appear within the first two years of a dog’s life. If the dog begins to groom excessively, with licking or chewing of the paws, abdomen and hindquarters, then it may suffer from atopic dermatitis. Another indicator is the ears are reddened and hot to the touch.

A hidden sign that a dog is atopic is in the armpits, groin, or between the toes of the paws. Check to see if there is saliva staining. In light coloured dogs it appears as a red-brown staining. In chronic cases the skin, mostly in the abdomen, may change colour from a pinkish, to angry red, to black mottling.

Food allergy and parasitic infestations may mimic the symptoms of atopic dermatitis making it difficult to diagnose. Once fleas, foods, and parasitic infestations are eliminated, then an allergy skin testing for dust mites, pollens, and moulds may be done to determine what causes the dog’s atopic dermatitis.

Flea treatments can also cause allergic reactions.

**Inhalant Allergy** – just like humans, canine inhalant allergies are caused by pollens (tree, grass and weed), dust mites, moulds and chemicals. Any dogs can acquire inhalant allergies the most common breeds that are affected include terriers.

The symptoms of an inhalant allergy include sneezing, runny nose and eyes, scratching, biting, chewing at feet and constant licking. The itching may be most severe on feet, flanks, groin and armpits. Inhalant allergies are often the reason for recurrent ear infections in your dog.

Aerosols, ‘plug ins’ and powders used to make a room or furniture/carpets smell good can cause inhalant and skin allergies also be careful of products used to wash bedding and floors.

**Food Allergy** – dogs can become allergic to a food they have eaten for years this means many people overlook the possibility of a food allergy. Food allergies only account for approximately 10 per cent of allergy problems in dogs.

Food sensitivities in a dog may manifest as itchy skin, scratching at ears, shaking of the head, licking and biting at the hind quarters or feet, rubbing faces on carpeting, ear inflammations, coughing, diarrhoea, flatulence, sneezing, asthma like symptoms, behavioural changes, seizures, gagging, ‘gulps’ and vomiting.

If food contains chicken this could well be the cause. Some foods are ‘sprayed’ with hydrolysed chicken fat which makes it more palatable to dogs, but they can still be allergic to this. Also take care with feeding wheat/gluten and other grains.

**Allergy Testing** – Your Vet may recommend testing for allergies. One such facility is Hemopet (Dr Jean Dodds): [http://www.nutriscan.org/](http://www.nutriscan.org/)

Hemopet on Facebook: [https://www.facebook.com/DrJeanDoddsHemopetNutriScan/?fref=ts](https://www.facebook.com/DrJeanDoddsHemopetNutriScan/?fref=ts)
Links – others are available - just google!

Coconut Oil:  
http://www.dogsnaturallymagazine.com/?post_type=post&s=coconut+oil


Turmeric Paste:  
http://www.dogsnaturallymagazine.com/?post_type=post&s=tumeric

http://www.dogsnaturallymagazine.com/turmeric-dogs/

12 Benefits of Turmeric for dogs:  http://keepthetailwagging.com/a-dog-supplement-turmeric/

Dr Karin Becker discusses turmeric on YouTube:  
https://www.youtube.com/watch?v=rkCz2MR-k_Q
References & Acknowledgements:
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Special thanks go to Dr Meryl Littman (retired), for her dedication and work on behalf of the SCWT.

Contacts & Web sites:
WHI Web site: www.wheatenhealthinitiative.com
SCWT Club of America – Web site: www.scwtca.org
SCWT Club of GB – Web site: www.wheaten.org.uk

United Kingdom (UK)
Vet Referral Only

Animal Health Trust - Diagnostic Laboratory Services
Post Mortem & blood/urinalysis
Lanwades Park
Kentford
Newmarket
Suffolk, CB8 7UU
Telephone: 01638 552993
Fax: 01638 555643
Email: diagnostics@aht.org.uk Web site: www.aht.org.uk

DNA Storage at the AHT - http://www.aht.org.uk/cms-display/genetics_why_dna_test.html
Swabs for DNA Storage - contact the Health Team at the SCWT Club of GB: www.wheaten.org.uk

Post Mortem RVC:
Professor Ken Smith, BVM&S, PhD, FHEA, FRCPath, FRCVS
Professor of Companion Animal Pathology, Royal Veterinary College
Telephone: 01707 666208 Fax: 01707 661464
Email: ksmith@rvc.ac.uk Web site: www.rvc.ac.uk

USA
Your vet should be able to find a Veterinary Internist in your area who is familiar with the hereditary diseases which can affect the SCWT.

Organisations - UK:
British Veterinary Association (BVA) (For information on Eye and Hip Schemes)
7 Mansfield Street
London W1G 9NQ Web site: www.bva.co.uk

BVA Canine Health Schemes:
http://www.bva.co.uk/canine_health_schemes/Canine_Health_Schemes.aspx

The Kennel Club
1 Clarges Street
Piccadilly
London, W1J 8AB
Telephone: 0870 606 6750 Web site: www.the-kennel-club.org.uk
Organisations – USA:
American Kennel Club - http://www.akc.org/

Canine Health: http://www.akc.org/dog-breeders/breeder-education/canine-health/

CHIC: http://www.caninehealthinfo.org/chicinfo.html

Orthopaedic Foundation for Animals (OFA) - http://www.ofa.org/

Worldwide:
Information – Genetic Testing for PLN-Associated Variant Genes
http://www.scwtca.org/health/dnatest.htm

UK – Swabs for the PLN-Associated Variant Gene Test contact: The Health Team at the SCWT Club of GB: www.wheaten.org.uk

Grooming your Wheaten:
It is very beneficial to your dog’s health to avoid the coat becoming knotty, tangled and too dirty. Grooming enables you to inspect your dog’s ears, eyes, mouth and skin and pick up on any changes.

The WHI website has information on grooming equipment and a number of videos which will assist you to learn how to care for your Wheaten’s coat.
Go to: www.wheatenhealthinitiative.com and click on the grooming section.

SCWT Club of GB also run Grooming Workshops visit their website for details of one near you: www.wheaten.org.uk

SCWT Club of America also has detailed information on grooming: http://www.scwtca.org/groom/index.htm

Facebook Group ‘Wheaten Grooming Matters’ – Professional Groomer and Wheaten Breeder Lisa Lopez offers videos and helpful tuition for bathing, drying and trimming etc: https://www.facebook.com/groups/472338763131723/