Familial nephropathy in Wheaten Terriers was originally described as a dysplastic disorder of renal maturation. Dogs as young as few months old were affected by poor weight gain, anorexia, vomiting and weight loss. The microscopic features of dysplastic kidneys in Wheaten Terriers included immature glomeruli, tubular dilation, thickened basement membrane, tubular loss, and proliferation of connective tissue. A subsequent report documented protein-losing nephropathy characterized by similar clinical signs, but histologically as membranoproliferative glomerulonephritis. Some of the dogs in the latter study were also diagnosed with protein-losing enteropathy. The goal of this study was to characterize blood and urine results in Wheaten Terriers evaluated for renal disease.

**SCREENING PROTOCOL**

Between September 1992 and November 1998, Wheaten Terriers were evaluated for renal disease every 6 months. No dog exhibited weight loss, anorexia, vomiting, or polyuria. Serum concentrations of creatinine, urea nitrogen and albumin were performed on every dog. Urine specific gravity, pH, and protein (dipstick) were determined on voided urine samples or samples collected by cystocentesis. Urine samples with qualitative protein determinations of 3+ or greater were further evaluated by urine protein creatinine ratios.

Dogs were categorized based on the following definitions. Dogs with serum creatinine concentrations of 1.7mg/dl or greater had azotemia. Azotemic dogs with a urine specific gravity of 1.030 or less had renal failure. Dogs with urine protein creatinine ratios greater than 1 had abnormal proteinuria. Hypoalbuminemia was defined in any dog with a serum albumin concentration of 2.5 g/dl or less.

**RESULTS**

Six hundred and sixty three samples were evaluated from 342 apparently healthy Wheaten Terriers. Most dogs were evaluated serially; however, 112 dogs were evaluated only once. Dogs ranged in age form 3.3 months to 13 years.

Azotemia was diagnosed in 6.8% of dogs. Six azotemic dogs had inappropriately low urine specific gravity consistent with renal failure. These dogs ranged in age from 8 months to 12 years. Of these 6 dogs with renal failure, 4 dogs had excessive proteinuria consistent with a diagnosis of protein-losing glomerulonephropathy. The remaining 2 dogs had urine protein creatinine ratios less than 0.5.

Seventeen dogs with azotemia had urine specific gravity values greater than 1.035. Twelve dogs were evaluated one or more times following the initial detection of azotemia. In 6 dogs, azotemia was transient. In 6 dogs, azotemia persisted. Two dogs with persistent azotemia subsequently developed inappropriately low urine specific gravity consistent with a diagnosis of renal failure.

Proteinuria was diagnosed in 2.3% of dogs evaluated. Dogs with proteinuria ranged in age from 3 to 12 years. At the time proteinuria was diagnosed, 3 dogs also were azotemic. All 3 azotemic dogs had inappropriately low urine concentrating ability consistent with renal failure. Another dog subsequently developed renal failure a year later. Of these 8 proteinuric dogs only 1 was hypoalbuminemic; this dog also had renal failure.

Hypoalbuminemia was diagnosed in 1.5% of dogs. Azotemia and proteinuria were diagnosed in 1 hypoalbuminemic dog. Another dog developed weight loss and diarrhea; microscopic evaluation of intestinal biopsies were consistent with a diagnosis of lymphocytic-plasmocytic enteritis.

**DISCUSSION**
In a population of apparently healthy Wheaten Terriers, 3% were diagnosed with renal failure or protein-losing nephropathy. Proteinuria was one of the earliest abnormalities detected, occurring prior to the development of azotemia. Although, a diagnosis of primary renal failure (rather than pre-renal or post-renal azotemia) is usually confirmed by detection of azotemia and impaired urine concentrating ability (urine specific gravity below 1.030), this definition may not be accurate for all forms of renal failure. The ability of dogs to concentrate urine to a specific gravity of 1.030 or greater is generally accepted as evidence of adequate renal function to prevent clinical signs of primary renal failure. The observation that 6 dogs with persistent azotemia had urine specific gravities greater than 1.035 and that 2 dogs later developed reduced urine specific gravity consistent with renal failure questions the validity of the urine specific gravity cutoff of 1.030. A higher value may be a more suitable endpoint of an adequate population of functioning nephrons to prevent the clinical signs associated with renal failure.

Based on results of this study, the following guidelines are recommended for identifying renal disease in Wheaten Terriers:

1. Serum concentrations of creatinine and albumin, and urine determinations of specific gravity, protein, and protein creatinine ratio are adequate to detect primary renal failure (azotemia and inappropriately low urine specific gravity) and protein-losing nephropathy (abnormal proteinuria).

2. With the goal of identifying disease early, dogs should be screened even though they may not have clinical signs of disease.

3. Immature dogs with clinical signs consistent with renal failure (e.g. poor weight gain, anorexia, and polyuria) should be tested.

4. Once abnormal laboratory values are detected, they should be repeated to verify persistence of disease.

5. Persistent azotemia with adequate urine concentration may be an early indication of primary renal failure.

6. Consider protein-losing enteropathy as a potential cause of hypoalbuminemia in dogs with proteinuria.