Persistent proteinuria of renal origin is an important marker of chronic kidney disease (CKD) in dogs and cats. Unfortunately, because of the high incidence of false-positive results for proteinuria on the urine dipstick screening test and proteinuria associated with lower urinary tract inflammation in dogs and cats, positive reactions for urine protein are quite common, and therefore often disregarded. Ruling out false-positive proteinuria and identifying proteinuria of renal origin are necessary first steps when evaluating the results of tests for proteinuria. In the case of CKD, albumin is usually the primary component of renal proteinuria. In addition to being a diagnostic marker for CKD, the potential for renal proteinuria/albuminuria to be a mediator of CKD progression also exists. The recent development of species-specific albumin ELISA technology that enables detection of low concentrations of canine and feline albuminuria has stimulated discussion about what level of proteinuria/albuminuria is normal and what levels may be associated with renal disease progression. For these reasons, detection and monitoring of renal proteinuria in dogs and cats have recently received renewed interest. Perhaps somewhat similar to our changing definition and treatment guidelines for systemic hypertension, the need to recognize, monitor, and potentially treat renal proteinuria, which may have been considered normal not long ago, is increasing.

NORMAL PHYSIOLOGY

The urine of healthy dogs and cats contains only a small amount of albumin and other proteins. The selective permeability of the glomerular capillary wall restricts the filtration of most plasma proteins, primarily on the basis of protein weight and, to a lesser extent, on the basis of protein charge size, and sterical configuration. Small and electrically neutral or positively charged proteins are more readily filtered than are large and negatively charged proteins.
proteins. For example, normal glomerular filtrate usually contains little protein with a molecular weight the size of albumin (69,000 d) or greater.

The glomerular capillary wall has three primary components: the endothelial cells that line the capillary lumen, the basement membrane, and the epithelial cells that line the visceral surface of the capillary wall (Fig. 1). The endothelial cells are highly fenestrated and provide part of the electrostatic barrier for negatively charged proteins. The basement membrane is composed of hydrated and tightly cross-linked type IV collagen, laminin, nidogen, and proteoglycans. Glomerular epithelial cells, also known as podocytes, form extensions (foot processes) that interdigitate on the visceral surface of the basement membrane. These podocyte foot processes are covered by a proteinaceous structure known as the slit diaphragm. The glomerular basement membrane and the slit diaphragm are thought to provide most of the size- and charge-selective permeability of the glomerular capillary wall.

The glomerular filtrate of healthy dogs and cats contains only 2 to 3 mg/dL of albumin compared with the 4 g/dL of albumin found in the plasma. Smaller molecular-weight proteins as well as those positively charged larger proteins that do pass through the glomerular capillary wall are almost completely reabsorbed by tubular epithelial cells by an active process termed pinocytosis. Such reabsorbed proteins may be broken down and used by the epithelial cells or returned to the bloodstream. This protein reabsorption occurs primarily in the proximal convoluted tubule and reduces the concentration of albumin in normal distal tubular fluid to less than 1 mg/dL. This reabsorptive process has a transport maximum, however. Tubular proteinuria may occur if that maximum is exceeded (e.g., excessive production of small-molecular-weight proteins like Bence-Jones proteins) or if damage to the tubular epithelial cells

![Fig. 1. Transmission electron micrograph of the glomerular capillary wall from a normal dog. BM, basement membrane; CL, capillary lumen; E, endothelial cell (note fenestrations); FP, podocyte foot processes; MFP, major foot process; US, urinary space.](image-url)
(eg, nephrotoxic damage, chronic tubulointerstitial disease) decreases their reabsorptive capacity.

Protein present in normal urine may also result from the secretion of enzymes, mucoproteins, and immunoglobulins by tubular and lower urinary and genital tract epithelial cells. These secreted proteins may account for as much as 50% of the proteins that are present in the urine of healthy animals.

SCREENING TESTS FOR PROTEINURIA

Proteinuria is routinely detected by semiquantitative screening methods, such as the conventional dipstick colorimetric test (common) and the sulfosalicylic acid (SSA) turbidimetric test (less common). The dipstick test is inexpensive and easy to use (Fig. 2). This test primarily measures albumin; however, the sensitivity and specificity for albumin are relatively low with the dipstick methodology. False-negative results (decreased sensitivity) may occur in the setting of Bence-Jones proteinuria, low concentrations of urine albumin, or dilute or acidic urine. The conventional dipstick test has a sensitivity level of greater than 30 mg/dL. False-positive results (decreased specificity) may be obtained if the urine is alkaline or highly concentrated, the urine sediment is active (pyuria, hematuria, or bacteriuria), or the dipstick is left in contact with the urine long enough to leach out the citrate buffer that is incorporated in the filter paper pad. False-positive results with the dipstick method occur more frequently in cats compared with dogs but are common in both species. For example, when 298 canine and feline urine samples were analyzed by a conventional urine protein dipstick method (Multistix Reagent Sticks; Bayer Corporation, Elkhart, Indiana) and a canine or feline albumin-specific quantitative ELISA (Heska Corporation, Fort Collins, Colorado), there were disparate results [1]. The sensitivity for the conventional urine protein dipstick test for albuminuria in canine and feline urine was 54% and 60%, respectively, and the urine

Fig. 2. Standard screening with dipstick methodology for assessment of proteinuria.
protein dipstick specificity for canine and feline albuminuria was 69% and 31%, respectively. If urine samples with an alkaline pH (≥7.5) or hematuria (≥10 red blood cells [RBCs] per high-power field [hpf]), pyuria (≥5 white blood cells [WBCs]/hpf), or bacteriuria were excluded, the dipstick specificity for canine and feline albuminuria increased to 84% and 55%, respectively. These data demonstrate that conventional urine protein dipstick tests have a high percentage of false-negative and false-positive results for detection of albuminuria in canine and feline urine when compared with an albumin-specific ELISA. Urine protein dipstick false-positive results in both species can be decreased by excluding alkaline urine and urine with hematuria, pyuria, or bacteriuria from analysis.

The SSA test is performed by mixing equal quantities of urine supernatant and 5% SSA in a glass test tube and grading the turbidity that results from precipitation of protein on a scale from 0 to 4+ (Fig. 3). In addition to albumin, the SSA test can detect globulins and Bence-Jones proteins. False-positive results may occur if the urine contains radiographic contrast agents, penicillin, cephalosporins, sulfisoxazole, or the urine preservative thymol. The protein content may also be overestimated with the SSA test if uncentrifuged turbid urine is analyzed. False-negative results are less common in comparison with the conventional dipstick test because of the increased sensitivity of the SSA test for protein (>5 mg/dL). Because of the relatively poor specificity of the conventional dipstick analysis, many reference laboratories confirm a positive dipstick test result for proteinuria with the SSA test. Grading of the color change on the dipstick test and the turbidity on the SSA test is subjective; therefore, results can vary between individuals and laboratories.

Proteinuria detected by these semiquantitative screening methods has historically been interpreted in light of the urine specific gravity and urine sediment. For example, a positive dipstick reading of trace or 1+ proteinuria in hypersthenuric urine has often been attributed to urine concentration rather than

Fig. 3. SSA standards demonstrate the increasing turbidity that occurs with increasing proteinuria when 5% SSA is mixed with an equal volume of urine.
to abnormal proteinuria. In addition, a positive dipstick reading for protein in the presence of microscopic hematuria or pyuria has often been attributed to urinary tract hemorrhage or inflammation. In both examples, the interpretation may not be correct. Given the limits of the conventional dipstick test sensitivity, any positive result for protein, regardless of urine concentration, may be abnormal (except in the case of false-positive results). Likewise, hematuria and pyuria have an inconsistent effect on urine albumin concentrations; not all dogs with hematuria and pyuria have albuminuria [2].

**LOCALIZATION OF PROTEINURIA**

When proteinuria is detected by screening tests, it is important to try to identify its source. Proteinuria may be caused by physiologic or pathologic conditions (Table 1). Physiologic or benign proteinuria is often transient and abates when the underlying cause is corrected. Strenuous exercise, seizures, fever, exposure to extreme heat or cold, and stress are examples of conditions that may cause physiologic proteinuria. The mechanism of physiologic proteinuria is not completely understood; however, transient renal vasoconstriction, ischemia, and congestion are thought to be involved. Decreased physical activity may also affect urine protein excretion in dogs; one study showed that urinary protein loss was higher in dogs confined to cages than in dogs with normal activity levels [3].

Pathologic proteinuria may be caused by urinary or nonurinary abnormalities. Nonurinary disorders associated with proteinuria often involve the production of small-molecular-weight proteins (dysproteinemias) that are filtered by the glomeruli and subsequently overwhelm the reabsorptive capacity of the proximal tubule. An example of this “prerenal” proteinuria is the production of immunoglobulin light chains (Bence-Jones proteins) by neoplastic plasma cells. Genital tract inflammation (eg, prostatitis, metritis) can also result in pathologic nonurinary proteinuria. Obtaining urine samples by means of cystocentesis reduces the potential for urine contamination with protein from the lower urinary tract.

Pathologic urinary proteinuria may be renal or nonrenal in origin. Nonrenal proteinuria most frequently occurs in association with lower urinary tract inflammation or hemorrhage (also referred to as postrenal proteinuria). Changes observed in the urine sediment are usually compatible with the underlying inflammation (eg, pyuria, hematuria, bacteriuria, increased numbers of transitional epithelial cells). Conversely, renal proteinuria is most often caused by increased glomerular filtration of plasma proteins associated with intraglomerular hypertension or the presence of immune complexes, structural damage, or vascular inflammation in the glomerular capillaries. Renal proteinuria may also be caused by decreased reabsorption of filtered plasma proteins attributable to tubulointerstitial disease. In some cases, tubulointerstitial proteinuria may be accompanied by normoglycemic glucosuria and increased excretion of electrolytes (eg, Fanconi syndrome, acute tubular damage). Glomerular lesions usually result in higher magnitude proteinuria compared with proteinuria
associated with tubulointerstitial lesions. Renal proteinuria caused by glomerular and tubular disease is most frequently accompanied by an inactive urine sediment, with the exception being the presence of hyaline casts. In addition to glomerular and tubulointerstitial disease, renal proteinuria may be caused by inflammatory or infiltrative disorders of the kidney (eg, neoplasia, pyelonephritis, leptospirosis), which are often accompanied by an active urine sediment.

Table 1
Localization of proteinuria

<table>
<thead>
<tr>
<th>Type of proteinuria</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physiologic/benign proteinuria</td>
<td>UP/C usually &lt;0.5</td>
</tr>
<tr>
<td>Examples include</td>
<td>Compatible history</td>
</tr>
<tr>
<td></td>
<td>Intermittent/transient</td>
</tr>
<tr>
<td>Pathologic proteinuria</td>
<td></td>
</tr>
<tr>
<td>Nonurinary</td>
<td></td>
</tr>
<tr>
<td>Examples include</td>
<td></td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>Variable UP/C</td>
</tr>
<tr>
<td>Hemoglobinuria/myoglobinuria</td>
<td>History/PE/echocardiogram</td>
</tr>
<tr>
<td>Dysproteinemia/dysproteinuria</td>
<td>Urine remains red after centrifugation</td>
</tr>
<tr>
<td>Genital tract inflammation/hemorrhage</td>
<td>Serum/urine electrophoresis</td>
</tr>
<tr>
<td>Urinary, nonrenal</td>
<td></td>
</tr>
<tr>
<td>Examples include</td>
<td></td>
</tr>
<tr>
<td>Lower urinary tract</td>
<td>UP/C not indicated</td>
</tr>
<tr>
<td>inflammation (eg, bacterial</td>
<td>History/PE</td>
</tr>
<tr>
<td>cystitis, cystoliths, polyps</td>
<td>Urine sediment</td>
</tr>
<tr>
<td>neoplasia)</td>
<td>Imaging</td>
</tr>
<tr>
<td>Urinary, renal</td>
<td></td>
</tr>
<tr>
<td>Examples include</td>
<td></td>
</tr>
<tr>
<td>Renal parenchymal</td>
<td>Variable UP/C</td>
</tr>
<tr>
<td>inflammation (eg, pyelonephritis,</td>
<td>Urine sediment</td>
</tr>
<tr>
<td>renoliths, neoplasia)</td>
<td>Imaging</td>
</tr>
<tr>
<td>Tubular proteinuria</td>
<td>UP/C usually 0.5–1.0</td>
</tr>
<tr>
<td></td>
<td>Can be associated with normoglycemic glucosuria and excessive urinary loss of electrolytes</td>
</tr>
<tr>
<td>Glomerular proteinuria</td>
<td>Persistent UP/C &gt;1.0</td>
</tr>
<tr>
<td></td>
<td>Inactive urine sediment with the exception of possible hyaline casts</td>
</tr>
</tbody>
</table>

Abbreviations: PE, physical exam; UP/C, urine protein/creatinine ratio.
DETECTION OF ALBUMINURIA/MICROALBUMINURIA

Albuminuria can be measured by point-of-care semiquantitative tests (eg, E.R.D.-HealthScreen Urine test; Heska Corporation) and quantitative immunoassays at reference laboratories. Like proteinuria, albuminuria can be caused by pre- and postrenal disorders; therefore, it is important to localize the source of albuminuria as discussed previously. Microalbuminuria (MA) is defined as concentrations of albumin in the urine that are greater than normal but less than the limit of detection using conventional dipstick urine protein screening methodology (ie, ≤30 mg/dL). Urine albumin concentrations greater than 30 mg/dL are referred to as overt albuminuria and can often be detected using the urine protein/creatinine ratio (UP/C) (see section on quantitation of proteinuria). The lower end of the MA range has been less easily defined because of the requirement that this concentration be greater than “normal” and the necessity that this concentration be reliably detected. In the dog and cat, the lower limit was defined based on the log mean plus 2 standard deviations of populations of apparently healthy dogs and cats as greater than 1 mg/dL. Urine albumin concentrations can be adjusted for differences in urine concentration by dividing by urine creatinine concentrations. For example, a urine albumin/creatinine ratio greater than 0.03 is considered abnormal in people. Alternatively, urine can be diluted to a standard concentration, such as 1.010, before assay. In one study of dogs, normalizing urine albumin concentrations to a 1.010 specific gravity yielded similar results to the urine albumin/creatinine ratio [4].

Indications for the use of MA tests [5] include the following: (1) when conventional screening tests for proteinuria produce equivocal or conflicting results or false-positive results are suspected, (2) when conventional screening tests for proteinuria are negative in apparently healthy older dogs and cats and a more sensitive screening test is desired, (3) when conventional screening tests for proteinuria are negative in apparently healthy young dogs and cats with a familial risk for developing proteinuric renal disease and a more sensitive screening test is desired, (4) when conventional screening test results for proteinuria are negative in dogs and cats with chronic illnesses that are associated with proteinuria renal disease and a more sensitive screening test is desired, 5) when a previous MA test result(s) was positive and monitoring for persistence or progression of the MA is desired.

CAUSES OF MICROALBUMINURIA

MA reflects the presence of intraglomerular hypertension or generalized vascular damage and endothelial cell dysfunction in human beings [6]. It is interesting to note that the presence of MA has been shown to be an accurate predictor of subsequent renal disease in human beings with systemic hypertension and diabetes mellitus, and it has also been observed in human beings with systemic diseases that are associated with glomerulopathy [7–11]. Importantly, early detection of albuminuria and institution of appropriate treatment have slowed the progression of kidney disease in people [12].
Based on recent studies, MA seems to be a good indicator of early renal disease in dogs, particularly those diseases that involve the glomerulus [4,13,14]. Albuminuria was evaluated in 36 male dogs with X-linked hereditary nephropathy, a rapidly progressive glomerular disease that is secondary to a defect in type IV collagen, a structural component of the glomerular basement membrane [4]. In these dogs, lesions in the glomerular basement membrane become apparent by 8 weeks of age. Persistent MA was detected between 8 and 23 weeks of age, 0 to 16 weeks before the onset of overt proteinuria, which occurred at 14 to 30 weeks of age. It was concluded that MA was a reliable early marker of developing nephropathy.

In 12 healthy dogs that were experimentally infected with *Dirofilaria immitis* L3 larvae and longitudinally evaluated, all the dogs developed MA, with 82% of all samples collected over the 14- to 23-month postinfection period of study being positive for MA [13]. The onset of MA corresponded to the onset of antigenemia. The magnitude of MA increased over time, and MA preceded the development of overt proteinuria, as measured by the UP/C. At the end of the study, the dogs had histologic evidence of glomerular disease by light (n = 11) or electron (n = 12) microscopy [13].

Finally, the prevalence of MA in 20 Soft-Coated Wheaten Terriers that were genetically at risk for the development of protein-losing enteropathy and nephropathy was 76% [14]. The magnitude of MA increased over time, and 43% of the dogs with MA eventually developed abnormal UP/Cs. Of interest is the observation that persistent MA develops in dogs with this type of protein-losing nephropathy at approximately the same time that mesangial hypercellularity and segmental glomerular sclerosis occur. Concurrent inflammatory bowel disease may account for MA in some of the dogs that have not progressed to overt proteinuria.

Other conditions have been reported in dogs with MA, including infectious, inflammatory, neoplastic, metabolic, and cardiovascular disease [15,16]. Results of a study of MA in dogs with lymphosarcoma and osteosarcoma demonstrated that urine albumin concentrations were significantly increased in dogs with these tumors, even though the UP/Cs may not be increased to greater than the reference range [17]. Urine albumin concentrations did not, however, consistently decrease with decreased tumor burden.

The prevalence of MA in dogs admitted to intensive care unit (ICU) is higher than in other reported patient populations and seems to vary with different classifications of disease [15,16]. As reported in people with acute inflammatory conditions, transient MA occurred in some of these dogs. A large percentage of patients that were euthanized or died had MA, suggesting that, as in people, the presence of MA may be a negative prognostic indicator.

Although amoxicillin and clavulanic acid and carprofen do not seem to affect albuminuria, corticosteroid administration does increase albuminuria. Short-term prednisone administration has been shown to cause a substantial but reversible increase in the magnitude of proteinuria in heterozygous, or carrier, female dogs with X-linked hereditary nephropathy [18]. Finally,
a moderate amount of exercise (treadmill work for 20 minutes) did not affect albuminuria in dogs [19].

It is important to note that the sensitivity of MA assays makes it likely that some positive results are caused by benign or physiologic proteinuria. In these cases, follow-up assays should be negative, confirming that the MA was transient. Transient MA is likely to be of little or no consequence.

**QUANTITATION OF PROTEINURIA**

If the results of the screening tests suggest the presence of renal proteinuria/albuminuria, urine protein excretion should be quantified. This helps to evaluate the severity of renal lesions and to assess the response to treatment or the progression of disease. Methods used to quantitate proteinuria include the UP/C and immunoassays for albuminuria, the results of which are expressed as urine albumin/creatinine ratios or in milligrams per deciliter in urine samples that have been diluted to a standard urine specific gravity (eg, 1.010). Albumin greater than or equal to 30 mg/dL in urine that has been diluted to a specific gravity of 1.010 usually results in UP/Cs greater than the normal range in cats and dogs. Urine that contains enough albumin to register greater than a medium reaction on the early renal damage (ERD) test also often has a UP/C greater than the normal range. The UP/C and urine albumin/creatinine ratio from spot urine samples have been shown to reflect the quantity of protein/albumin excreted in the urine over a 24-hour period accurately. Because of the difficulty of 24-hour urine collection, this methodology has greatly facilitated the diagnosis of proteinuric renal disease in veterinary medicine. Most studies have shown that normal urine protein excretion in dogs and cats is 10 to 30 mg/kg or less over 24 hours and that normal UP/Cs are 0.2 to 0.3 or less [20–22]. Initially recommended normal values for canine UP/Cs of less than 1.0 were likely conservative and have more recently been lowered. Today, UP/Cs less than 0.5 and less than 0.4 are considered to be normal for dogs and cats, respectively [5]. Persistent proteinuria that results in UP/Cs greater than 0.4 and greater than 0.5 in cats and dogs, respectively, in which pre- and postrenal proteinuria has been ruled out, are consistent with glomerular or tubulointerstitial CKD. UP/Cs greater than 2.0 are strongly suggestive of glomerular disease. The definition of normal may continue to change with additional research. For example, even the ultralow-level single-nephron proteinuria that can arise secondary to intraglomerular hypertension in hypertrophied nephrons in CKD is abnormal in the face of what may be considered normal whole-body or whole-kidney proteinuria.

**MONITORING RENAL PROTEINURIA**

Transient renal proteinuria/albuminuria is likely of little consequence and does not warrant treatment. Conversely, persistent proteinuria/albuminuria indicates the presence of CKD. Persistent proteinuria/albuminuria of renal origin can be defined as positive test results on three or more occasions 2 weeks or longer apart. Because persistent proteinuria/albuminuria can be constant or increase or
decrease in magnitude over time, monitoring should use quantitative methods to determine disease trends or response to treatment. Changes in the magnitude of proteinuria should always be interpreted in light of the patient’s serum creatinine concentration because proteinuria may decrease in progressive renal disease as the number of functional nephrons decreases. Decreasing proteinuria in the face of stable serum creatinine suggests improving renal function, whereas decreasing proteinuria in the face of increasing serum creatinine suggests disease progression.

**IMPLICATIONS OF PROTEINURIA/ALBUMINURIA**

In addition to the classic complications of moderate to heavy proteinuria (hypoalbuminemia, edema, ascites, hypercholesterolemia, hypertension, and hypercoagulability), there is increasing evidence in laboratory animals and human beings that proteinuria can cause glomerular and tubulointerstitial damage and result in progressive nephron loss. Proteinuria can arise secondary to immune-mediated, vascular inflammatory, or structural damage to the glomerular capillary wall or as a consequence of intraglomerular hypertension. Plasma proteins that have crossed the glomerular capillary wall can accumulate within the glomerular tuft and stimulate mesangial cell proliferation and increased production of mesangial matrix in human beings [23]. In addition, excessive amounts of protein in the glomerular filtrate can be toxic to human tubular epithelial cells and can lead to interstitial inflammation, fibrosis, and cell death by several mechanisms [24–26]. These mechanisms include tubular obstruction, lysosomal rupture, and complement-mediated and peroxidative damage as well as increased production of cytokines and growth factors.

Several studies in human patients with proteinuric renal disease suggest that proteinuria is associated with renal disease progression. In a study of people with chronic glomerulonephritis, the decrease in proteinuria associated with several different treatments predicted the change in the slope of the reciprocal value of serum creatinine over 6 months [27]. In a 3-year study of 583 human beings with various renal diseases, the angiotensin-converting enzyme (ACE) inhibitor benazepril reduced proteinuria and systemic blood pressure and slowed the decline in glomerular filtration rate (GFR) when compared with placebo treatment [28]. The protective effect of benazepril on renal function was greatest in those patients with substantial proteinuria (>3 g over 24 hours) even after adjustments were made for changes in diastolic blood pressure or urinary protein loss over time [28]. Finally, in a study of 7728 nondiabetic people, overt albuminuria was independently associated with decreased GFR [11].

Evidence linking proteinuria to progression of renal disease in dogs and cats is also beginning to accumulate. In cats with naturally occurring CKD, relatively mild proteinuria (UP/C >0.43) seemed to be negative predictors of survival [29]. In cats with the remnant kidney model of chronic renal failure, proteinuria was associated with nephron hypertrophy, increasing intraglomerular pressures, and hyperfiltration [30]. Interestingly, proteinuria has also been associated with an increased risk of mortality attributable to all causes in cats.
that have normal renal function when their proteinuria is first detected [31]. In dogs with naturally occurring CKD, the relative risk of uremic crises and mortality was approximately three times greater in dogs with UP/Cs greater than 1.0 (n = 25) compared with dogs with UP/Cs less than 1.0 (n = 20) [32]. In this study, the risk of an adverse outcome was approximately 1.5 times greater for every single-unit increase in UP/C and the decline in renal function was greater in dogs with higher UP/Cs [32]. Individual nephron hyperfiltration and proteinuria have been documented in dogs with the remnant kidney model of renal failure [33]; however, treatments that have slowed the functional decline or histologic changes associated with this model have had variable effects on proteinuria. ACE inhibition and ω-3 fatty acid supplementation have decreased proteinuria and slowed progression [34–36]; however, calcium blockade treatment resulted in increased mesangial cell proliferation despite decreasing proteinuria [34]. Other treatments, such as reduction of dietary phosphorus, decreased renal disease progression in remnant kidney dogs but had no effect on proteinuria. In dogs with experimentally induced immune complex glomerulonephritis, treatment with a thromboxane synthetase inhibitor decreased proteinuria and attenuated the development of glomerular lesions but had no effect on established lesions [37,38]. Reduction of proteinuria by means of an ACE inhibitor (enalapril) was also associated with slowed progression of renal disease in dogs with two different types of naturally occurring glomerulopathies [39,40].

SUMMARY
Proteinuria is a common disorder in dogs and cats that can indicate the presence of CKD before the onset of azotemia or the presence of more severe CKD after the onset of azotemia. Although a direct pathogenetic link between glomerular disease, proteinuria, and progressive renal damage has not been established, attenuation of proteinuria has been associated with decreased renal functional decline in several studies. There is a need to continue to increase our understanding of the effects of proteinuria on the glomerulus, the tubule, and the interstitium in dogs and cats. In addition to being a diagnostic marker of renal disease, proteinuria may also contribute to the progressive nature of canine and feline renal disease. Proteinuria is commonly associated with primary glomerular diseases; however, the loss of renal autoregulation that occurs secondary to nephron loss attributable to any cause (eg, vascular, tubular, interstitial, glomerular) can also result in intraglomerular hypertension and proteinuria. In addition, renal proteinuria can be associated with decreased tubular reabsorption secondary to tubulointerstitial disease.

References


