Canine & Feline Urinary Tract Infections and Pyelonephritis
Richard E. Goldstein, DVM, DACVIM, DECVIM-CA
The Animal Medical Center, New York, NY
Urinary tract infections (UTIs) are the most common cause of lower urinary tract signs in dogs. Although rare in young cats, UTIs are being diagnosed with a much higher frequency today in older cats and cats with underlying urinary and non-urinary diseases. The incidence of pyelonephritis is unknown in companion animals but is thought to be a common cause of acute renal failure and of deterioration of more stable chronic renal disease. This talk will provide an overview of UTIs in dogs including causes of UTIs, and the common bacteria associated with UTIs. Emphasis will be placed on the pathogenesis of E. Coli UTIs, as well as less common bacterial and fungal organisms, and how lower urinary tract infections induce pyelonephritis and in many cases permanent renal damage. Treatment of acute, chronic and recurrent UTIs will be discussed. Feline UTIs and pyelonephritis will be discussed including the results of a large study assessing UTIs in cats with a variety of underlying medical conditions.

DEFENSE MECHANISMS
Urinary tract infections do not occur with even higher frequency due to the normal defense mechanisms of the canine and feline lower urinary tract. These include:

1. The antibacterial properties urine including those of urea of urea as well as those of increased urine osmolality and urine acidity (likely important especially in cats).

The normal host defenses of the bladder and urethra include complete and normal voiding or hydrokinetic washout, inherent antimicrobial properties of the normal urothelium and a high pressure zone in the mid urethra. This area is more effective in males than females. Local secretion of IgA provides humoral immunity.

The unidirectional flow of urine in the ureters may play a role in preventing ascending bacterial infections. There are no ureteral valves in dogs and cats, like in human ureters. Vesicoureteral reflux can be normal in dogs.

If bacteria do arrive at the renal pelvis there are poorly understood last ditch defense mechanisms to prevent nephritis.

Anatomical abnormalities, urolithiasis, incontinence, low urine osmolality, increased urinary glucose or decreased immunity would increase the likelihood of a UTI. Urinary catheterization also thought to increase the likelihood of a UTI. A recent study in the ICU unit at UC Davis, though, showed that when managed properly indwelling catheters only caused UTIs in approximately 5% of the cases if left in for 1 day and close to 40% if left in for 4 days. Interestingly, the positive predictive value for catheter tip culture in that study was only 25%. Much has been published about underlying disease as a predisposing factor for UTIs and pyelonephritis in dogs. Little though is known about cats. In a recent retrospective study we determined the incidence of UTIs in cats with chronic renal disease (22%), diabetes mellitus (12%) and hyperthyroidism (12%) all much higher than cats presenting with otherwise unexplained signs of lower urinary tract disease (3%). Although bacteria and white blood cells were recognized in the urine of the majority of cats with positive urine cultures, there were quite a few cats with UTIs despite an inactive sediment. This observation is true in dogs as well and therefore justifies culturing the urine of any dog or cat thought to be at risk for a UTI even if the sediment is benign.

Bacterial virulence factors have been studied extensively over the last 5–10 years including many studies involving common bacteria of the canine (very little feline) urinary tract. Most of the studies are on factors effecting adhesion, penetration and virulence of E. coli, the most common canine urogenital pathogen. Uropathogenic virulence Factors (UVFs) have been identified that appear to differentiate uropathogenic E. coli from commensal intestinal E. coli. These strains are more likely to possess operons for pap (encoding for P fimbriae), fim (type 1 fimbriae), sfa (S fimbriae), hlyA (α-hemolysin), and cnf1 (cytotoxic necrotizing factor 1).

The mechanisms of renal damage caused by ascending UTIs have not been well studied in dogs or cats. In humans it has been shown that 55–75% of children that develop febrile UTIs have concurrent renal
parenchymal damage, and 20–40% of these children will suffer permanent renal cell damage. The possible pathogeneses of renal damage from UTIs that has been studied in humans include:

**Vesicoureteral reflux**

Direct effects of uropathogenic bacteria on renal cells via above mentioned virulence factors (fimbriae, LPS, HlyA) causing:
- Local inflammatory response (cytokine and chemokine production)
- Infiltration of inflammatory cells
- Liberation of proteolytic enzymes and free radicals

Eventually leading to cell death, and if sub-lethal then ultimately to fibrosis and renal scarring. It is unknown how similar the processes are in companion animal medicine.

Bacterial resistance represents a constant challenge to the veterinary practitioner or urologist assisting in the treatment of dogs and cats with complicated UTIs. This is especially challenging when dealing with gram-negative bacteria that may contain plasmids encoding for multi resistance genes. Some of these genes encode for resistance to 3rd or 4th tier antibiotics and may even be exchanged between human and canine strains of bacterium. Nosocomial resistant bacteria and resistance to 2nd and 3rd tier antibiotics like fluoroquinolones are becoming more and more common and present a major concern in large veterinary referral centers and teaching hospitals. Examples would be fluoroquinolone resistant *E. coli*, *Pseudomonas* or *Enterococcus faecium*. Often aminoglycosides remain the only antibiotic available for those cases, obviously a worrisome choice for many renally impaired patients. The following table represents the historical susceptibility patterns for the commonly used antibiotics. Remember that today this is much less predictable and culture and sensitivities are warranted even for the most apparently simple UTI.

<table>
<thead>
<tr>
<th>Agent</th>
<th><em>E. coli</em></th>
<th>Coag + Staph</th>
<th>Proteus mirabilis</th>
<th>Kleb</th>
<th>Pseudomonas</th>
<th>Strep viridans</th>
<th>Strep canis</th>
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<tbody>
<tr>
<td>Amoxi</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<td>+</td>
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<tr>
<td>Amoxi + Clav</td>
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<td>+</td>
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<tr>
<td>Ampicillin</td>
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<td>Cephalexin</td>
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<td>+</td>
<td>+</td>
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<tr>
<td>Chloro</td>
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<td>Enro</td>
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<tr>
<td>Gentamicin</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+ (&gt; 89%)</td>
<td>+</td>
<td>+</td>
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<tr>
<td>TMP-Sulfa</td>
<td>+</td>
<td>+</td>
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**REFERENCES**


