Infections of the urinary tract vary in severity. The most frequent form is asymptomatic bacteriuria (ABU) which occurs in about 1% of young girls, 2% of pregnant women and 15-20% of elderly individuals. The frequency estimates for acute pyelonephritis and acute cystitis are less consistent. Both are common conditions in all age groups. Acute pyelonephritis is distinguished from acute cystitis by the involvement of the kidneys and systemic sites. Acute pyelonephritis is commonly diagnosed as significant bacteriuria accompanied by fever, loin pain, elevated acute-phase reactants and reduced renal concentrating capacity (1).

Acute pyelonephritis may be defined as a bacterial infection involving the kidneys. Despite this apparently simple definition, acute pyelonephritis is a heterogeneous disease entity, varying in severity as well as consequences. Prior to the advent of antibiotics, the mortality rate was 15-20%, and recurrent episodes of acute pyelonephritis and progressive renal scarring were a major cause of end stage renal diseases. Today, acute pyelonephritis remains a serious condition, and, despite adequate treatment, renal scarring still occurs in a subgroup of the patients with acute pyelonephritis (1).

The aims of this review are: [1] to summarize the information on bacterial virulence and kidney tropism; [2] to comment on host response mechanisms which are involved in the pathogenesis of acute pyelonephritis; [3] to discuss host and bacterial determinants of renal scarring

**BACTERIAL VIRULENCE**

Escherichia coli is the most frequent cause of acute pyelonephritis and renal scarring (1). This review of bacterial virulence will therefore be limited to Escherichia coli. This
Gram-negative bacterium is an inhabitant of the large intestine both in man and animals (2). Most Escherichia coli strains are members of the normal fecal flora in healthy individuals. A subset of Escherichia coli cause infection both in the intestine and at extraintestinal sites e.g. diarrhoea, urinary tract infection (UTI), neonatal meningitis and septicemia.

This difference in virulence between Escherichia coli strains was recognized as early as the turn of the century (3, 4). Several investigators subsequently tried to classify Escherichia coli hoping to find markers which would discriminate between the more or less virulent strains. Systematic studies of Escherichia coli virulence were made possible by the development of serotyping (5-7). Isolates which had particular pathogenicity in man were found to belong to certain O antigen types and to be hemolytic more often than other strains (8, 9).

The clonal structure of Escherichia coli populations

The extended use of serotyping techniques revealed that certain O antigens occurred in combination with a limited set of capsular (K) antigens and flagellar (H) antigens (7). Furthermore, strains of a given O:K:H antigen combination (serotype) resembled each other in the biochemical reactions used for biotyping.

These findings led to the concept that natural populations of Escherichia coli occur as lineages or clones (10). Orskov (10) used the word "clone" to denote "bacterial cultures isolated independently from different sources in different locations and at different times but sharing so many identical phenotypic and genotypic traits that the most likely explanation for this identity is a common origin". More recent work has shown that Escherichia coli strains of a specific O:K:H antigen combination share a number of independent phenotypic characteristics including outer membrane protein patterns (OMP), electrophoretic types of cytoplasmic enzymes (ET) and adherence properties (11-15). The identification of Escherichia coli isolates as members of the same clone should ideally be based on DNA sequence homology. For practical purposes the identification of clones among clinical isolates is mainly based on phenotypic characteristics such as O:K:H serotype, biotype and ET.

The multilocus enzyme electrophoresis technique deserves some special comments. Intracellular enzymes are separated according to their mobility in an electric field. Enzymatically active proteins are detected by their ability to convert a substrate to a colored end product. Isoenzymes of the same substrate specificity but with different mobilities are identified and designated as electromorphs. Since the difference in mobility