Characteristics

- Gram -ve, rod shaped
- oxidase -ve (lack cytochrome oxidase)
- Non-capsulated **except** *Klebsiella*, some strains of *E.coli*
- Most are motile by a flagella (peritrichous) **except** *Shigella* and *Klebsiella*
- Most reduce nitrate to nitrite via nitrate reductase
- Most are normal part of the gut flora in intestines of human and other animals other found in H_2O and soil

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1. Lactose fermenters
   - *Escherichia coli*
   - *Klebsiella spp.*
   - *Citrobacter spp.*
   - *Enterobacter spp.*

2. Non lactose fermenters
   - *Salmonella spp.*
   - *Shigella spp*
   - *Proteus spp*
   - *Yersinia spp*

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LF organism appears as pink colonies on MacConkey (e.g. *E. coli*)
NLF organism appears as colorless colonies on MacConkey (e.g. *Shigella*)

NLF

LF

Can you see? No?
Neither can I

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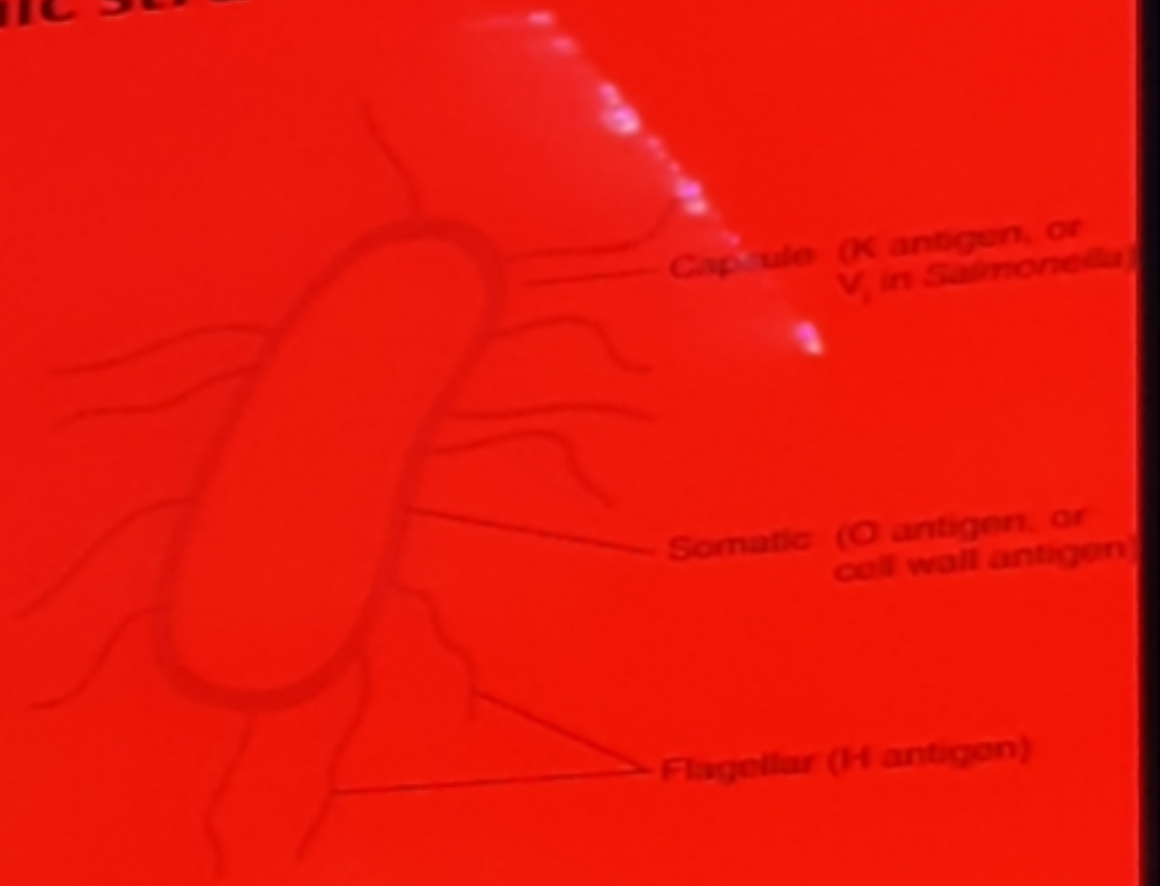
Antigenic structure

- Cell wall/ Somatic or O antigen-a single organism may carry several O antigens
- Capsular/K antigen- Prevent O agglutination, Not all strains have a capsule
- Flagella /H antigen-Not all are motile



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Escherichia coli

- Normal inhabitant of the G.I.T
- Produce infections in Humans and Animals
- Some strains cause various forms of gastroenteritis.
- Is a major cause of urinary tract infection and neonatal meningitis and septicemia.
- Detection of *E.coli* in water indicates pollution and contamination.

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Characteristics

- Gram negative rods
- ± capsule
- Motile by peritrichious flagella.
- Lactose fermenter
- pathogenic strains are haemolytic on blood agar
- Indole and methy lred positive

Virulence factors

1. Surface antigens and Toxins
   - –O antigen – LPS-has endotoxic activity,
   - –K antigen –protects against phagocytosis. E.g K1 has a strong association with virulence, particularly meningitis in neonates
2. Fimbriae attaches to uroepithelial cells - important in UTI
3. Exotoxins– haemolysins and enterotoxins

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Pathogenesis

1. Gastrointestinal – caused by
   - ETEC (enterotoxigenic *E.coli*)
   - EPEC (enteropathogenic *E.coli*)
   - EIEC (enteroinvasive *E.coli*)
   - EHEC (enterohaemorrhagic *E.coli*)
   - EAggEC (enteroaggregative *E.coli*)
2. Extra-intestinal
   - Urinary tract infections- uropathogenic *E.coli (UPEC)*
   - Pyogenic infections
   - Septicaemia

Uropathogenic *E.coli* (UPEC)

- The E.coli serotypes commonly responsible for UTI are those normally found in the feces
- Urinary tract infection can be:
  - Ascending infection- via urethra
    - Fecal bacteria, spread up tract to bladder, as well as kidneys, or prostate
  - Descending infection-haematogenous route
    - Rare
    - *E.coli* enter upper urinary tract organs from bloodstream

UPEC

- Virulence factors
  - –Adhesins: fimbriae or pili
  - –Biofilm formation
  - –LPS
  - –Iron acquisition mechanisms: siderophores contribute to survival and persistence in the urinary tract
  - –Immune evasion mechanisms

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Neonatal meningitis

- second most common bacterial cause of neonatal meningitis
- Babies are exposed to the bacteria during delivery
- Main viruence factor is capsular antigen

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Laboratory diagnosis

- Specimens- (List)
- Gram stain (Direct)– Gram negative rods, ± pus cells
- Culture – in air, 35-37°C, 18-24hrs
  - –BA – some strains β-haemolytic
  - –MacConkey- pink lactose fermenters colonies
  - –CLED – yellow colonies - lactose fermenters

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Lab

- Gram stain of colonies – gram negative rods
- Biochemical tests
  - –Indole test - positive
  - –Methyl red test- positive
  - –Voges-Proskauer test–negative
  - –Citrate utilization test–negative

  (IMViC tests ++––)

Treatment

- Should be guided by in vitro susceptibility tests
  - Uncomplicated UTI – Trimethoprim-Sulphamethoxazole / Nitrofurantoin
  - Nosocomial infections
    - Cephalosporins/ Carbapenems/ Fluoroquinolones/ Aminoglycosides, Piperacillin-Tazobactam

Genus Klebsiella

General Characteristics

- Gram -ve bacilli
- Large polysaccharide capsule - colonies with mucoid appearance
- Non-motile

Klebsiella species

- *Klebsiella pneumoniae*
- *K. oxytoca*
- *K. ozaenae*
- *K. rhinoscleromatis*
- *K.granulomatis*

Note: K. pneumoniae is mostly commonly isolated species

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K. pneumoniae

- Most *K. pneumoniae* infections originates from an individual's microbiota.
- *K. pneumoniae* is the causative agent of pneumonia, sepsis, UTI, bacteremia, meningitis, and pyogenic liver abscesses.
- Responsible for community-acquired pneumonias (CAPs) and hospital-acquired pneumonias (HAPs).

Pathogenesis

Virulence factors

- Capsular k antigen
- synthesise siderophores- taking up iron bound to host proteins
- fimbriae- adherence to respiratory and urinary epithelia
- Endotoxin
- Efflux pump

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Clinical presentation

- Pulmonary infections - pneumonia lobar;
- Bronchitis, bronchopneumonia
- Extrapulmonary infections
  - Meningitis and enteritis in infants
  - UTI
  - Septicemia
  - Cholecystitis, cholangitis, otitis, peritonitis, wound infections

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Laboratory diagnosis

1. Specimen: urine, pus, blood etc. depending on the site of infection.
2. Gram stain -Gram -ve rods
3. Culture: It grows on MacConkey , CLED or Blood agar media
   - Growth conditions in air, 35-37°C, 18-24hrs.
   - Colonies appear mucoid and pink on MacConkey agar media
   - CLED- yellow colonies
4. Gran stain- Gram -ve rods
5. Biochemical:
   - IMViC = - - + +

Klebsiella on Nutrient agar and Blood agar

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Treatment

- Based on antibiotics susceptibility tests
  - Beta lactam + beta lactamase inhibitior combination
  - 3rd generation of cephalosporins e.g cefataxime, ceftriaxone
  - Carbapenems/ fluoroquinolones/ aminoglycosides

Genus Proteus

- Non Lactose fermenters.
- Urease +ve
- Non-capsulated
- Highly motile- swarms on culture media
- Contain O (somatic antigen) and H (flagellar antigen).

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Pathogenesis

- Habitat - human colon, soil, H_2O
- Highly motile may contribute to ability to invade urinary tract
- Has fimbriae- for uroepithelial colonization
- Urease hydrolyzes urea in urine into ammonium hydroxide increasing urinary pH- Encourage calculus(stone) formation which;
  - Obstruct urine flow

Pathogenesis

- Alkaline urine favors growth of the organism and causes more extensive renal damage
- Bacteria in the calculus not reached by drugs; site for re-infections

Clinical syndrome

1. Urinary tract infection
2. Wound infections (including pressure sores, burns, damaged tissues)
3. Septicemia
4. Ear infections
5. Brain abscesses

Laboratory diagnosis

1. Specimens – (List)
2. Direct G/stain - Gram negative bacilli
3. Culture
   - Mac - Non-Lactose fermenter (NLF)
   - BA - Swarming, distinct fishy odour
4. Gram stain – Gram negative bacilli
5. Biochemical reactions
   - Urease positive
   - Oxidase negative
   - TSI: Alkaline slant, acid butt, gas, H_2S

characteristic "swarming" motility on BA

urease positive on right

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Treatment

- Based on susceptibility testing
- Combination of Trimethoprim - sulphamethoxazole
- Ampicillin - P. mirabilis
- Cephalosporins- P. mirabilis
  - The rest are resistant to 1st generation cephalosporins
- Fluoroquinolone
- Aminoglycosides

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Citrobacter

- NLF
- Associated with nosocomial UTI and respiratory infections, Endocarditis, Neonatal meningitis & brain abscesses
- Rx – multidrug resistance
  - –Aminoglycosides/ carbapenems/ quinolones/ antipseudomonal penicillins

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Enterobacter species

- Commensals in the human gut
- Cause- wound infections, burn infections, pneumonia, and UTI, Intra-abdominal infections, neonatal meningitis

Treatment:

–carbapenems, 4th generation cephalosporins

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