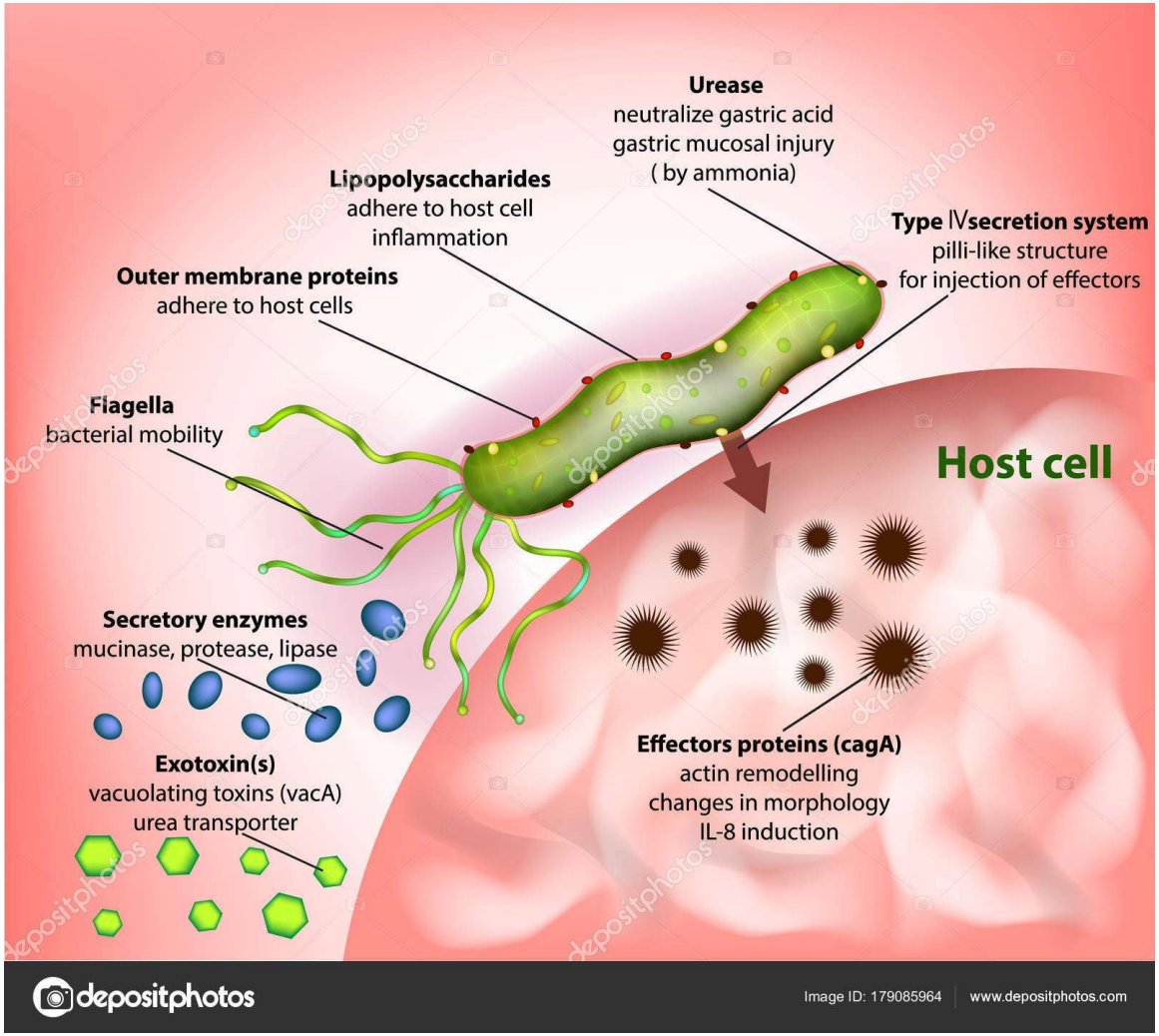
Helicobacter pylori

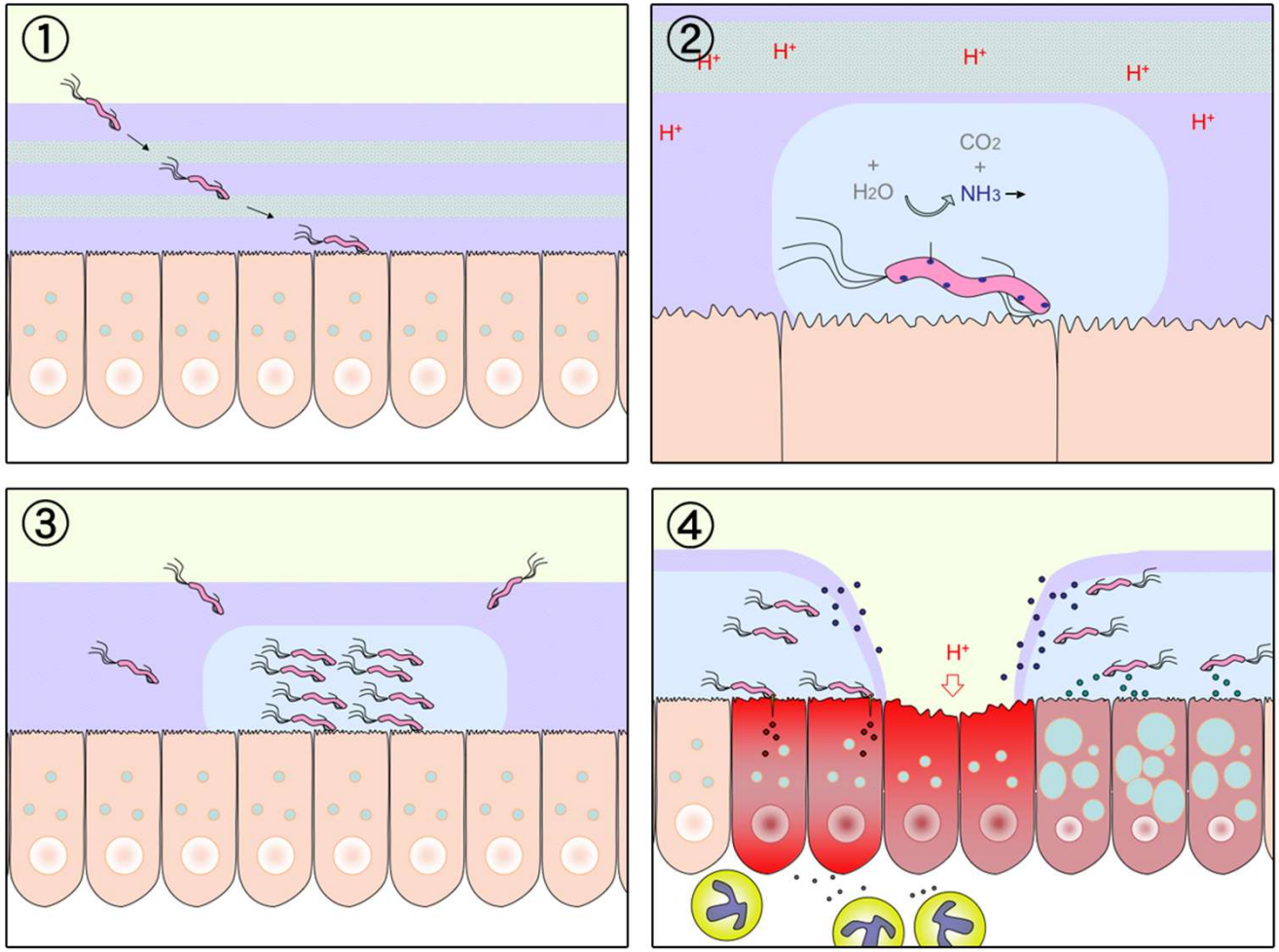
**** Helicobacter pylori*** is a spiral (helical), a microaerophilic, gram-negative bacterium that colonizes the gastric mucosa.
* The **prevalence** of *H. pylori* infection in our population is estimated at **30–55%.** The prevalence increases with the age of the population. *H. pylori* infection is present in 90–95% of patients with duodenal ulcers and in 60–80% of patients with gastric ulcers.

** Helicobacter pylori* is **classified** as a WHO **Group 1 Carcinogen**. However, there is no evidence that its eradication reduces the risk of stomach cancer

Virulence factors



Pathogenesis



See also: https://youtu.be/x3aZDY9Q_Qk

Etiology and epidemiology

* **motile**, curved microbe with **flagellum**
* lives in the mucus **the gastric mucosa**
* high **urease** production
* a purely **human pathogen**, has not been detected in animals or soil

about 50% of the population is estimated to be infected; the incidence is lower in developed countries

* **transmission** – **orofecal** or **oro-oral**; direct and indirect transmission from person to person is possible (e.g. contaminated food, dishes)

most infections are acquired in childhood - the most common is direct transmission from an infected mother; but there are also new infections in adulthood, especially people with impaired immunity

Symptoms

* Helicobacter colonizes mainly the **mucosa** of the **antrum** of the stomach, later the body, but also the cardia. The settlement is focal, not diffuse; therefore, a larger number of endoscopic biopsies is required for capture. Colonization of the **gastroduodenal mucosa** is accompanied by the development of **chronic gastritis**, which represents a **heterogeneous group of inflammatory processes** of various etiologies. Prolonged chronic gastritis caused by *H. pylori* can lead to mucosal atrophy and **intestinal metaplasia**, the most common precursor of intestinal gastric adenocarcinoma.
H. pylori infection has a causal relationship to the **peptic ulcer of the gastroduodenum**.
* **Disruption of the mucosal barrier** defense mechanisms (surfactant, mucus, basement membrane of gastric epithelial cells) by bacteria is followed by the **release of inflammatory metabolites of epithelial cells** and is one of the most important factors in the pathogenesis of chronic gastritis and peptic ulcers. It is not known to what extent the degree of inflammation is affected by the characteristics of the host (genetic factors) or pathogen (phenotype, genotypes).
* Eradication of *H. pylori* infection leads to **healing of the ulcer**.

Helicobacter extraintestinal diseases and syndromes

SIGNIFICANT

*** larynx disorders**

benign: chronic laryngitis (incidence of infection in almost 46% of patients in the research), vocal cord polyps (singing nodule)

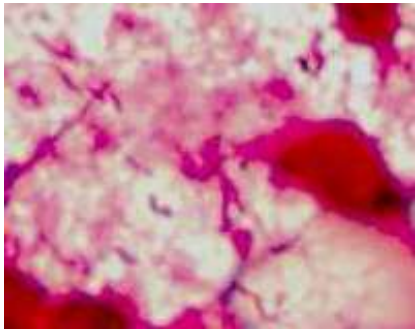
malignant: laryngeal tumors (incidence of infection in 46% of patients in the research)

Also documented

* autoimmune disease (idiopathic trombocytopenic purpura, autoimmune thyreoiditis)
* skin condition (acne, rosacea, idiopathic chronic urticaria)
* endocrine disorders (thyreopatia)
* neurological disorders (migraine)
* hepatobiliary disease
* cardiac (ICHS), vascular (Raynaudova choroba)
* recurrent dyspepsia/discomfort
* iron-deficiency anemia
* growth disorders

Diagnosis

- A stool polymerase chain reaction (**PCR**) test, stool **antigen test**
- Serological examination of *antibodies* against *Helicobacter pylori* by ELISA method (IgG, IgA)
- Endoscopically obtained **biopsy** specimens can be using Gram staining (see also the fig. below) tested by rapid **urease test**, histologically, or by **culture** (Skirowa's soil)
- 13C-labeled urea **breath test**
- Serological detection of preneoplastic markers may be useful in preventing gastric cancer



Gram stained bioptate with Gramnegative spiral (or „flying bird“ shaped) bacteria

Urease test
Positive on the right (red colour)

Treatment

* The gold standard of eradication is a **triple combination of drugs** given for 7 days:

omeprazole + amoxicillin + clarithromycin (or any other macrolide)

* If the patient has a penicillin allergy: omeprazole + metronidazole + clarithromycin

* In recent years, there has been a **decline in the successful eradication** after standard triple combination therapy with **increased resistance to antibiotics**, especially clarithromycin. Where there is increasing resistance to clarithromycin, bismuth quadruterium (bismuth) is recommended. Probiotics can reduce the incidence of side effects of standard eradication. The most successful eradication treatment is now the subject of numerous studies.

Campylobacteriosis

Etiology, epidemiology

- most frequent worldwide diarrheal infection
- **hemorrrhagic enterocolitis – immunocompetent patients**
- extraintestinal – immunocompromised
- **Gramnegative rods**, microaerophilic, special culture media, higher temperature (42C), ***Campylobacter jejuni***, *C. coli* and others
- **zoonosis – commensal in the intestine of wild and domestic animals**, **source** – ingestion of **contaminated food** (e.g. chicken, pork)

Campylobacteriosis

etiology, epidemiology

• incubation period – 2-7 days

• **hemorrrhagic enterocolitis** – immunocompetent patients

• extraintestinal – immunocompromised

* **pathogenesis:** invasive, production of toxins, found – jejunum, ileum, colon

* **symptoms:** from secretory diarrhea to severe illness, most common – **hemorrhagic enterocolitis** – mucuc and blood, high fever, rarely extraintestinal – sepsis or localized (e.g. meningitis)

Campylobacteriosis

Pathogenic Campylobacter species

Worldwide, pathogenic *Campylobacter* species are responsible for the cause of over **400–500 million infections cases each year**. Pathogenic *Campylobacter* species known to be implicated in human infections includes *C. jejuni*, *C. concisus*, *C. rectus*, *C. hyointestinalis*, *C. insulaenigrae*, *C. sputorum*, *C. helveticus*, *C. lari*, *C. fetus*, *C. mucosalis*, *C. coli*, *C. upsaliensis* and *C. ureolyticus*. These pathogenic *Campylobacter* species are grouped into **major human enteric pathogens** (*C. jejuni*, *C. jejuni* subsp. *jejuni (Cjj)*, *C. jejuni* subsp. *doyley* (*Cjd*), *C. coli* and *C. fetus*); **minor pathogens** (*C. concisus*, *C. upsaliensis, C. lari* and *C. hyointestinalis*) and **major veterinary pathogens** (*C. fetus* subsp. *venerealis* (*Cfv*) and *C. fetus* subsp. *fetus* (*Cff*)).

Ref: Igwaran A. Human campylobacteriosis: A public health concern of global importance, *2019*

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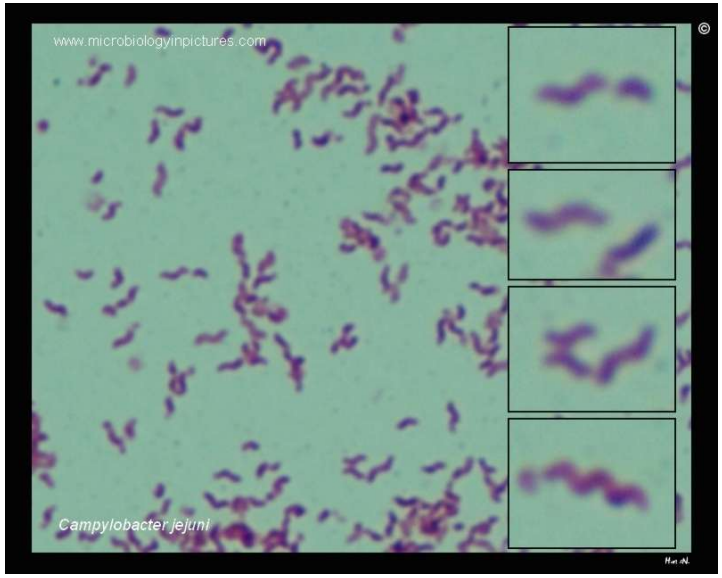
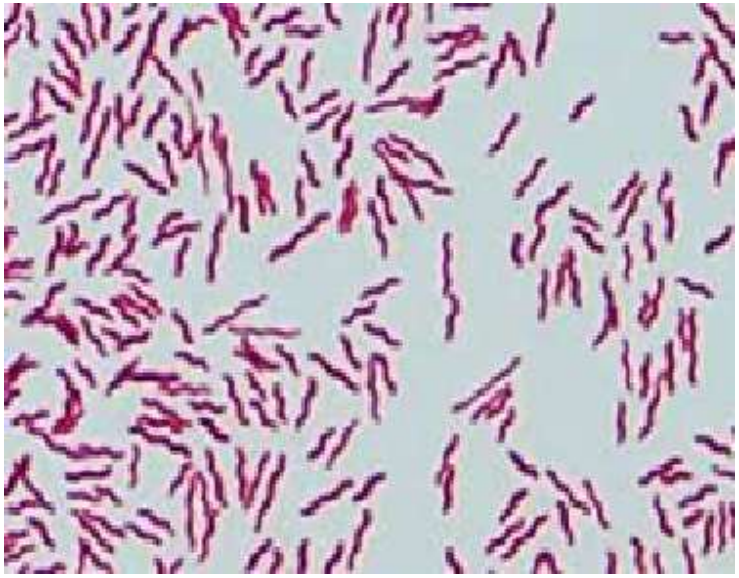
Campylobacteriosis

- ***C. jejuni*** is a **motile, microaerophilic, zoonotic, thermophilic** bacterial considered as the **leading cause of worldwide foodborne bacterial gastroenteritis**.
- It's a member of the genus *Campylobacter* with polar flagella and **helical morphology** that is used for movement through viscous solutions including the mucus layer of the gastrointestinal tract





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Campylobacteriosis

- ***Campylobacter coli*** is an S-shaped curved cell measuring about 0.2–0.5 micrometers long with a single flagellum. It's very similar to *C. jejuni*; and both bacteria cause inflammation of the intestine and diarrhea in humans.
- ***C. fetus*** is a curved cell, fastidious motile bacterial that majorly cause septic abortion in farm animals. *C. fetus* can cause infection in human and its infection can be acquired through direct contact with animals, through consumption of undercooked contaminated meat or through ingesting food or water contaminated by animal faeces. **Reported to be associated with human infection such as bacteremia. It's an opportunistic human pathogen that largely infects immunecompromised patients.** Some of the major reported symptoms of *C. fetus* infections include **endocarditis, meningitis, septicemia, septic arthritis, peritonitis and cellulitis,** but infections are rare.

Pathogenicity of *Campylobacter* species

- *Campylobacter* **pathogenicity is based on the virulence factors** (Larson et al., 2008) and these **virulence factors are multi-factorial** in nature and the ability of these bacteria to survival and resist physiological stress also contributes to its pathogenicity (Casabonne et al., 2016; Ketley, 1995). The various virulence related mechanisms displayed by *Campylobacter* species includes **invasive properties**, oxidative stress defence, **toxin production**, iron acquisition and its ability to remain viable but non-culturable state (Bhavsar and Kapadnis, 2006). *Campylobacter* **invasion**, **adherence** and colonization also add to the pathogenicity of these groups of bacteria (Backert et al., 2013). **Other virulence factors** of *Campylobacter* include; **secretion of some sets of proteins**, **translocation capabilities** and **flagella-mediated motility** (Biswas et al., 2011).

Motility and flagella

- **Motility is important for *Campylobacter* survival under diverse chemotactic conditions it comes across in the gastrointestinal tract** (Jagannathan and Penn, 2005). In some *Campylobacter* species, the motility system with the **flagella involves a chemosensory system** that steers flagella movement depending on the environmental conditions where these bacteria are found. *Campylobacter* **chemotaxis** and **flagellin** are the two important **lead these bacteria to its colonization site** and also **help in invading** the host cell (van Vliet and Ketley, 2001).
- **Chemotaxis -** is a method or system by which **motile bacteria sense and move to the direction of more favourable conditions** and several pathogenic bacteria uses this practice to invade their hosts.

Adhesion

- **Colonisation mediated by some adhesins on the bacterial surface (Jin et al., 2001).** *Campylobacter* adhesion virulence factors includes **outer membrane protein**, ***Campylobacter* adhesion protein A**, **phospholipase A, lipoprotein, periplasmic binding protein, fibronectin-like protein A and Type IV secretion system** (Bolton, 2015). *Campylobacter* adhering to fibronectin F is another important *Campylobacter* **virulence factor that enables these bacteria to bind to fibronectin** which **promotes the bacterium-host cell interactions and colonization** (Konkel et al., 2010). Other virulence genes in *Campylobacter* species reported to be linked with human infections responsible for **expression of colonization and adherence** (Datta et al., 2003).

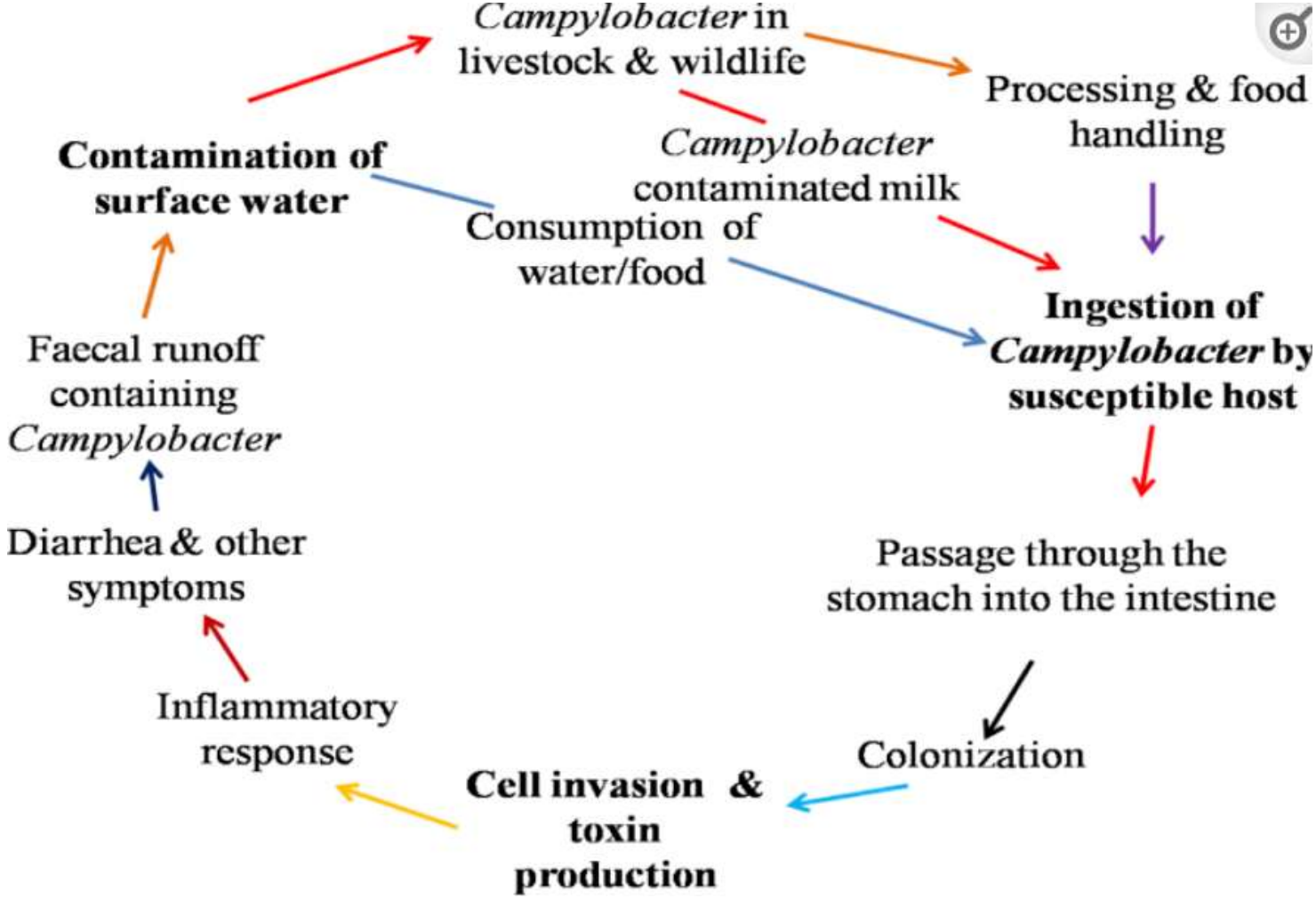
Toxin production

- *Campylobacter* produce different type of **cytotoxins** and **cytolethal distending toxin (CDT)** is one of these toxins **(Schulze et al., 1998).** CDT is a **tripartite toxin that is made up of three subunits encoded. Activity is determined by these three *cdt* cluster genes (Martinez et al., 2006).** The *cdtA* and *C* genes are **heterodimeric toxin subunits** responsible for **toxin binding and internalization of the host cell while *cdtB* is the subunit which encodes for the toxic/active components of the toxin (Abuoun et al., 2005). Cytolethal distending toxins induce diarrhea in both humans and animals by intrusive with the division of cells in the intestinal crypts (Carvalho et al., 2013).**

Invasion

- **Invasion is another virulence mechanism in *Campylobacter* that is carried out by the flagella** which also function as an export apparatus in the secretion of non-flagella proteins during host invasion **(**Poly and Guerry, 2008**). Flagella secretion system is vital for invasion and colonisation (**Konkel et al., 2004**). The secretion of invasion antigens and invasion protein B (*ciaB*) are also important virulence proteins** synthesized by *Campylobacter* species which **help in the epithelial cells invasion and adhesion of the host gastrointestinal tract (**Casabonne et al., 2016**). The periplasmic protein HtrA responsible for full binding to the epithelial cells.**

Campylobacter infections



Extraintestinal infections

* Are infections **outside the intestines** but symptoms are associated with a problem within the intestine (Hernandez and Green, 2006). Extragastrointestinal infections reported to be associated with *Campylobacter* infections includes **reactive arthritis**, **Guillain-Barré syndrome -** GBS (Kuwabara and Yuki, 2013), **bacteremia, septicaemia** (Man, 2011), **septic arthritis, endocarditis, neonatal sepsis, osteomyelitis, and meningitis** (Allos, 2001).

Campylobacteriosis

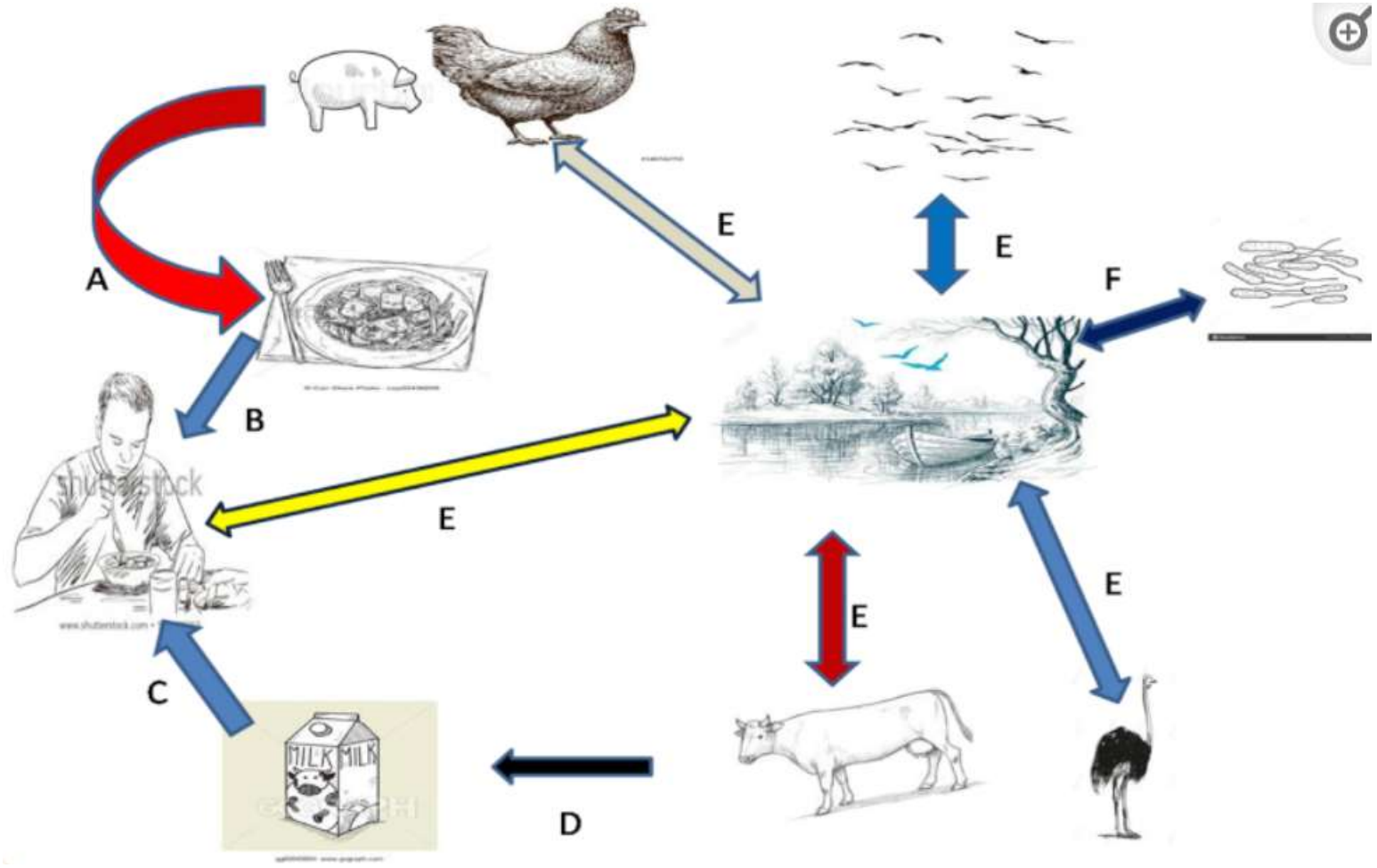
Diagnosis: stool culture, special media and atmosphere, extraintestinal – blood culture and other (CSF…), **PCR gastropanel** (together with patogenic protozoa, viruses and other bactaria)

Treatment: mild or moderate cases – **rehydratation, diet, adsorbent, probiotics**, **severe diarrhea and systemic inf**. – **macrolides,** fluoroquinolones (systemic inf. – weeks depending on susceptibility)

Charcoal-based selective media (CSM)

- Specimen – swab, stool and transport media
- e.g. Charcoal-based selective media (CSM) for isolation Campylobacter spp. from fecal specimes – small whitish or greyish colonies
- microaerophilic atmosphere at 42C, 48h
- microscopy – curved Gramnegative rods
- phenotypical identification – oxidase positive, mass spectrometry (MALDI)

Transmission routes of *Campylobacter* infection



Conclusion

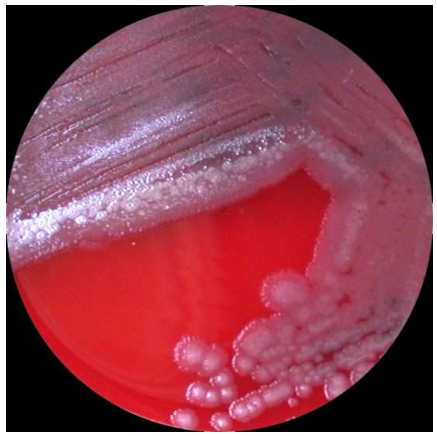
Worldwide, outbreaks of campylobacteriosis have been increasing and the major routes of transmission of these bacteria to human is generally believed to be through consumption of contaminated foods. The development of rapid Kits for *Campylobacter* detection and quantification in foods from animal origin will be essential for the prevention of *Campylobacter* infections. *Campylobacter* infections are majorly treated with antibiotics and the actions of these antibiotics have been compromised and this call for the development of new vaccines that will help to control the regular use of antibiotics in animal husbandry. In addition, regular domestic hygiene will also help to prevent *Campylobacter* infections. The production of new and effective antibiotic for better treatment of campylobacteriosis will as well help in the reduction of antibiotic-resistant *Campylobacter* strain and the spread of antibiotics resistant genes.

Nonfermenters (gramnegative non-fermenting rods)

Characteristics

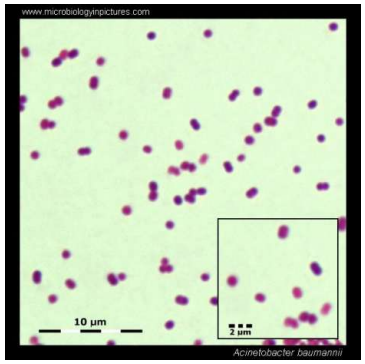
* **gram-negative rods**
* **easy to culture**; room temperature (approx. 30°C) to 37°C is sufficient
* **strictly aerobic**
* oxidize glucose (enterobacteria ferment glucose)
* usually oxidase-positive (enterobacteria are oxidase-negative)

Pseudomonas aeruginosa



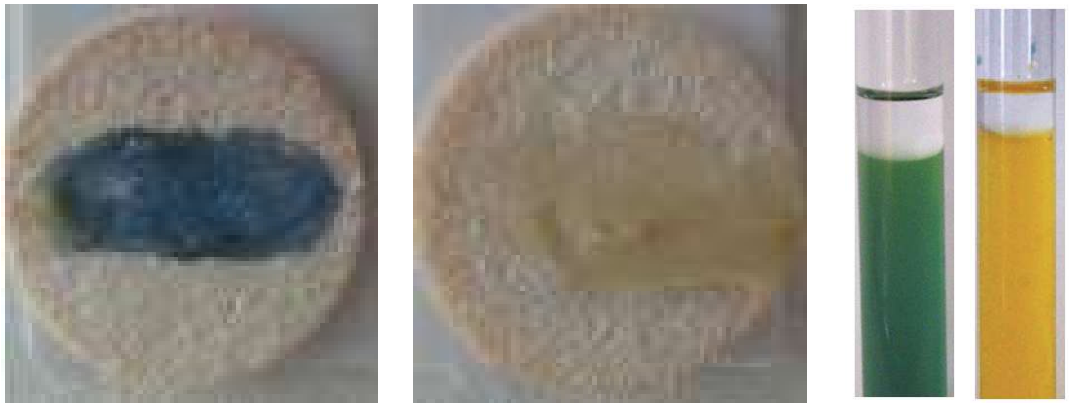
P. aeruginosa on nutrient agar – S phase (left, center), M phase – mucoid colonies (right) – correlate with biofilm formation in vivo; isolated from the same sputum sample from a patient with cystic fibrosis

Gram staining



Pseudomonas aeruginosa (on the left) and *Acinetobacter baumannii* (on the right)

Oxidase detection and glucose oxidation/fermentation tests



A **B** **C** **D**

Pseudomonads and other non-fermenting rods typically produce the enzyme oxidase (A – a blue color in the test indicates oxidase production), in contrast to enterobacteria (B, negative reaction), which are very similar to them in terms of morphology (Gram-negative) and growth. Pseudomonads and other non-fermenting rods do not ferment glucose (C), in contrast to enterobacteria (D). (Media containing glucose are overlaid with paraffin oil.)

Occurence

* **Distribution – ubiquitous** (environment; colonizes plants, animals, and humans)
common in hospital settings – usually strains resistant to 3 or more antibiotics (**multidrug-resistant strains**)
* **the causative agents of most infections are** – *Pseudomonas aeruginosa, Burkholderia cepacia, Stenotrophomonas maltophilia, Acinetobacter baumannii*

Virulence factors

* **have generally not been sufficiently studied** (with the exception of *Pseudomonas aeruginosa*)
* **structural components** (e.g., cell wall—inhibition of neutrophil and lymphocyte activity; fimbriae—adhesion; LPS—endotoxin activity)
* **toxins and enzymes** (e.g., exotoxins—tissue destruction; hemolysins)
* **Resistance to antibiotics and disinfectants**

Treatment and prevention

* **combination antibiotic therapy**; monotherapy is usually ineffective (risk of resistance development) e.g., aminoglycosides and antipseudomonal penicillins (e.g., piperacillin)
* **prevention of contamination of sterile instruments, equipment, and administered medications** (e.g., infusion solutions)
* **prevention of the spread of hospital-acquired infections**

Most significant pathogens

P. aeruginosa
Infections of various organs

MOST COMMONLY
* Primary skin infections
* Ear and eye infections
* Urinary tract infections
* Lower respiratory tract infections

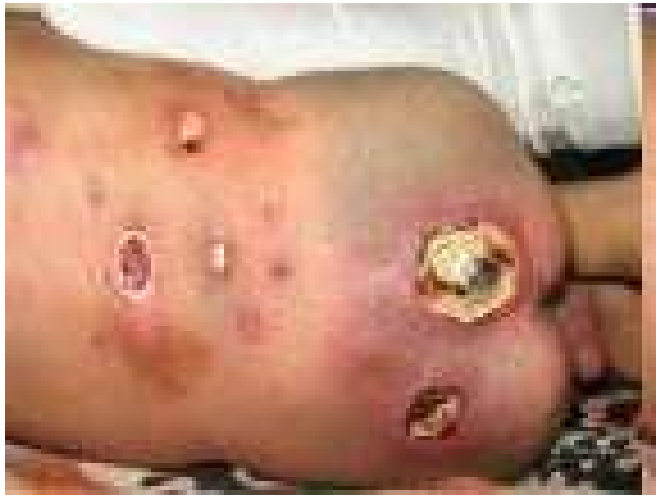
Virulence factors of *P. aeruginosa*

Structural components

* **adhesins** – fimbrial, non-fimbrial
* **polysaccharide capsule** – alginate (binds to epithelial cells, protects against phagocytosis, complement, and antibiotics; a component of the
* **biofilm**
* **endotoxin**
* **pigment production** – e.g., pyocyanin – a blue pigment (generates reactive oxygen species) – tissue damage, inhibition of ciliary cell motility in the respiratory epithelium

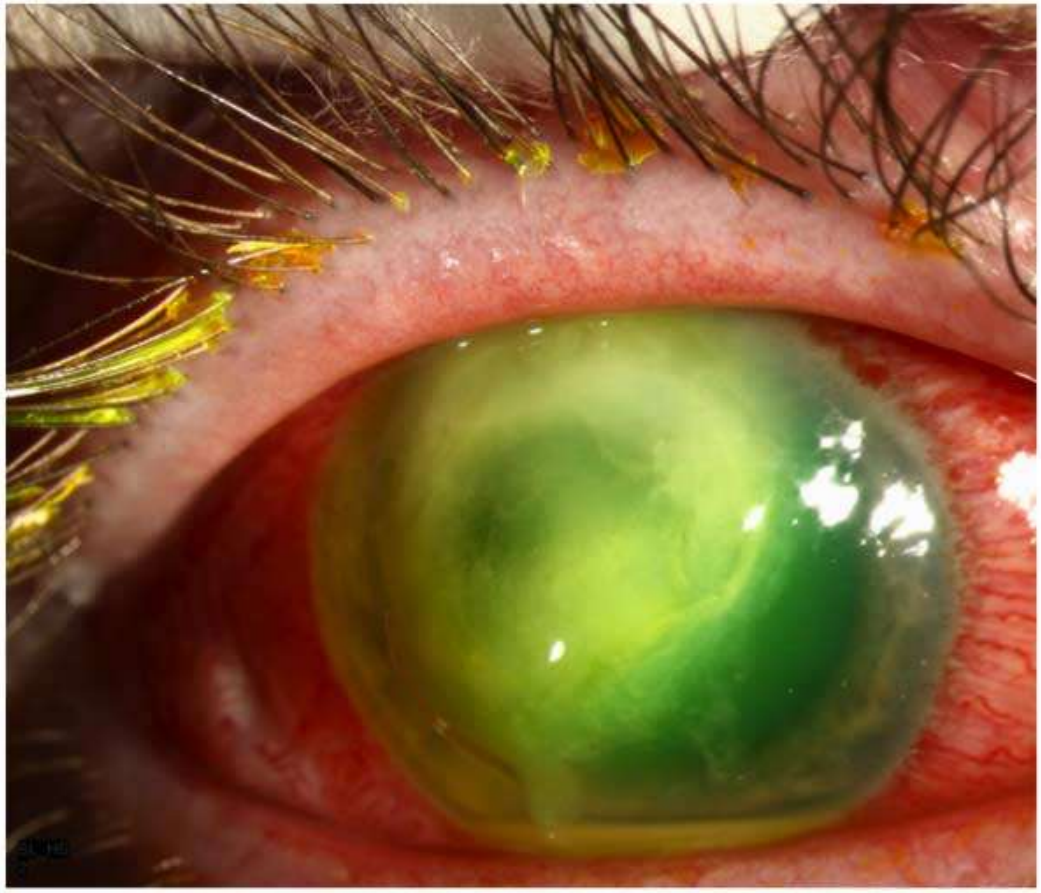
Toxins and enzymes

* **exotoxin A** (inhibition of protein synthesis, tissue damage), **enterotoxin S** (inhibition of protein synthesis, immunosuppression)
* **elastase** (damage to elastin—blood capillaries, lung parenchyma), hemorrhagic lesions (fig.),
* **phospholipase C (hemolysin)—**tissue damage and stimulation of the inflammatory process

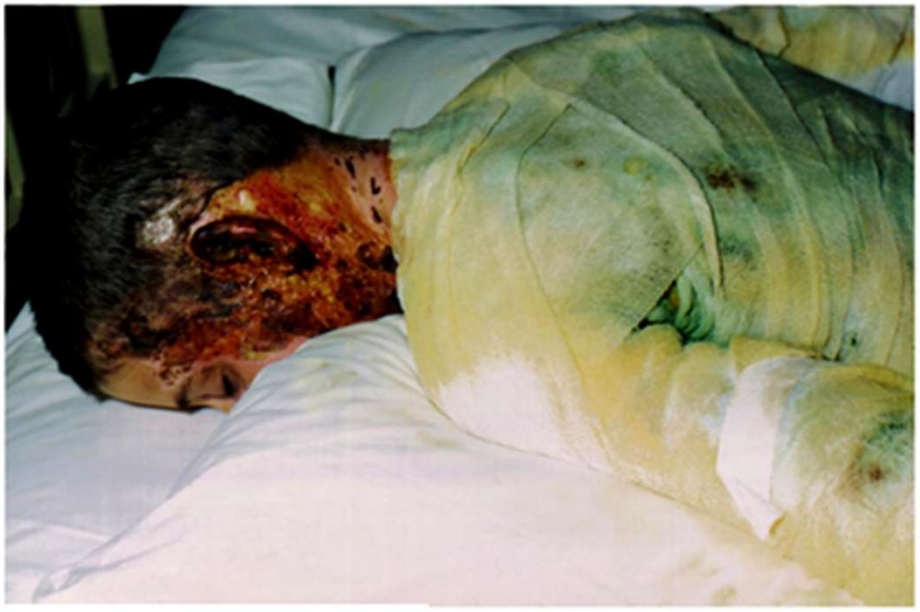


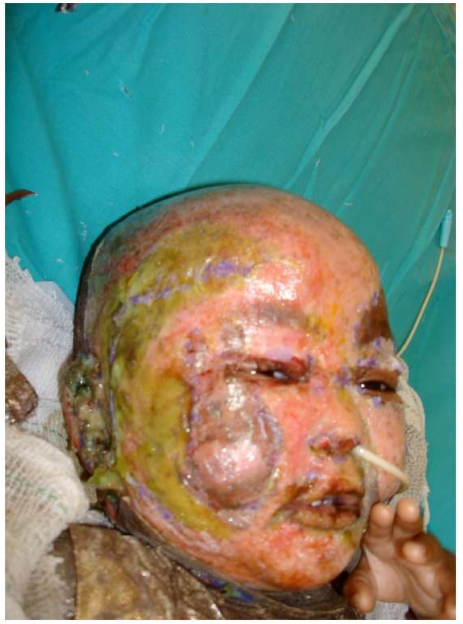
ectyma granulosum

Pseudomonas keratitis



Burn infections caused by *P. aeruginosa*





Drug of choice

Drugs of choice: The primary drug of choice for ***Pseudomonas aeruginosa*** is typically a beta-lactam with antipseudomonal activity, most commonly **cefepime, ceftazidime**, or **piperacillin-tazobactam**. For **MDR** infections, **ceftolozane-tazobactam** or **ceftazidime-avibactam** are preferred, **with aminoglycosides** (e.g., tobramycin) or **fluoroquinolones** (e.g., ciprofloxacin) used in combination.

Other Nonfermenters

Acinetobacter baumannii – a causative agent of serious **hospital-acquired infections** (e.g., ventilator-associated pneumonia or urinary tract infections), often **multidrug-resistant**

Drug of choice

Drugs of choice: For carbapenem-susceptible ***Acinetobacter baumannii***, **carbapenems** (meropenem, imipenem) are generally considered the drug of choice. For **multidrug-resistant (MDR)** or carbapenem-resistant strains (CRAB), **colistin** (polymyxin E) or minocycline typically show the highest susceptibility, with sulbactam-durlobactam or **tigecycline** used as alternatives.

Other Nonfermenters

Burkholderia cepacia

* **Chronic lung infections**: most commonly in patients with cystic fibrosis; complications **include sepsis (Cepacia syndrome)**
* **Hospital-acquired infections:** urinary or systemic infections in patients with indwelling catheters
* **Occurrence of pan-resistant strains** (resistant to most antibiotics)

Drug of choice

Drugs of choice: The first-line drug for the treatment of *Burkholderia cepacia* complex (Bcc) infections is **co-trimoxazole** (trimethoprim/sulfamethoxazole). Given the high **multidrug resistance** of these bacteria, **combination therapy** is often used, with other effective antibiotics including **ceftazidime, meropenem, minocycline**, and **levofloxacin**.

Other Nonfermenters

Stenotrophomonas maltophilia

* **hospital-acquired pathogen**: causative agent of various infections (primarily of the respiratory and urinary tracts) in immunocompromised patients, particularly those who have been treated with broad-spectrum antibiotics

Drug of choice

Drugs of choice: Trimethoprim-sulfamethoxazole (TMP-SMX) is generally recognized as the drug of choice for *Stenotrophomonas maltophilia* infections. Due to high rates of **intrinsic resistance to many antibiotics**, this pathogen has limited treatment options. When TMP-SMX cannot be used due to allergies, resistance, or toxicity, preferred alternatives include **minocycline**, **levofloxacin**, or **cefiderocol**.

*** Healthcare-associated infections (HAI)**

* Nosocomial infections (NI)

* Hospital aquired infection

Nosocomial infections

- Definition
- Predisposition
- Transmission
- Prevention
- Epidemiology

Definition of nosocomial infections

- infections which are a **result of treatment in a hospital or a healthcare service unit**, but secondary to the patient's original condition

- infections are considered nosocomial **if they first appear 48 hours or more after hospital admission or usually within 30 days after discharge**

- **etymology** - nosocomial comes from the Greek word **nosokomeion** meaning (*nosos* - **disease**, ***komeo*** - **to take care of**)

Predisposition to nosocomial infections

* patients **already in a poor state of health**, impairing their defense against bacteria – advanced age or premature birth along with **immunodeficiency** (due to drugs, illness, or irradiation) present a **general risk**, while other diseases can present **specific risks** - for instance chronic obstructive pulmonary disease

* **invasive devices**, for instance **intubation tubes, catheters, surgical drains and tracheostomy tubes** all **bypass the body's natural lines of defence** against pathogens and provide an **easy route for infection**. Patients already colonised on admission are instantly put at greater risk when they undergo an invasive procedure.

* **a patient's treatment itself can leave them vulnerable** to infection – immunosuppression and antacid treatment undermine the body's defences, while antimicrobial therapy (removing competitive flora and only leaving resistant organisms) and recurrent blood transfusions have also been identified as risk factors.

Transmission of nosocomial agents

- microorganisms are transmitted in hospitals **by several routes**
- the same microorganism **may be transmitted by more than one route**
- main routes of transmission:

- contact (direct, indirect)
- droplet
- airborne
- common vehicle
- vectorborne

Contact transmission

The **most important and frequent mode of transmission**

Direct-contact transmission

involves **a direct body surface-to-body surface contact** and physical transfer of microorganisms between a susceptible host and an infected or colonized person, such as occurs when a person turns a patient, gives a patient a bath, or performs other **patient-care activities** that require direct personal contact. Direct-contact transmission **also can occur between two patients**

Indirect-contact transmission

involves **contact of a susceptible host with a contaminated intermediate object**, usually inanimate, such as contaminated instruments, needles, or dressings, or contaminated gloves that are not changed between patients.

Other ways of trasmission

- **Droplet transmission** occurs **when droplets are generated** from the source person mainly during coughing, sneezing, and talking, and during the performance of certain procedures such as bronchoscopy
- **Airborne transmission** occurs by dissemination of **either airborne droplet nuclei** (small-particle residue {5 µm or smaller in size} of evaporated droplets containing microorganisms that remain suspended in the air for long periods of time) or **dust particles** containing the infectious agent, may become inhaled by a susceptible host (e.g. Legionella, Mycobacterium tuberculosis and the rubeola and varicella viruses)
- **Common vehicle transmission** applies to microorganisms transmitted to the host **by contaminated items** such as **food, water, medications, devices, and equipment**.
- **Vector borne transmission** occurs when **vectors** such as mosquitoes, flies, rats, and other vermin **transmit**

Categorization and prevention of NI

central line-associated bloodstream infections (CLABSIs)

before insertion – e.g. educated healthcare personel) insertion, care, maintenance)

at insertion – e.g. hand infection, sterile barrier preacutions, chlorhexidin-based antiseptic for skin preparation, all-inclusive catheter kit, avoid using the femoral vein for central acces in adults

after insertion - e.g. remove nonessential catheters, change catheters and gauze dressing permanently,

in the following days – e.g. bathe ICU patients older>2 months daily with chlorhexidine containing sponge, use

Categorization and prevention of NI

ventilator-associated pneumonia (VAP)

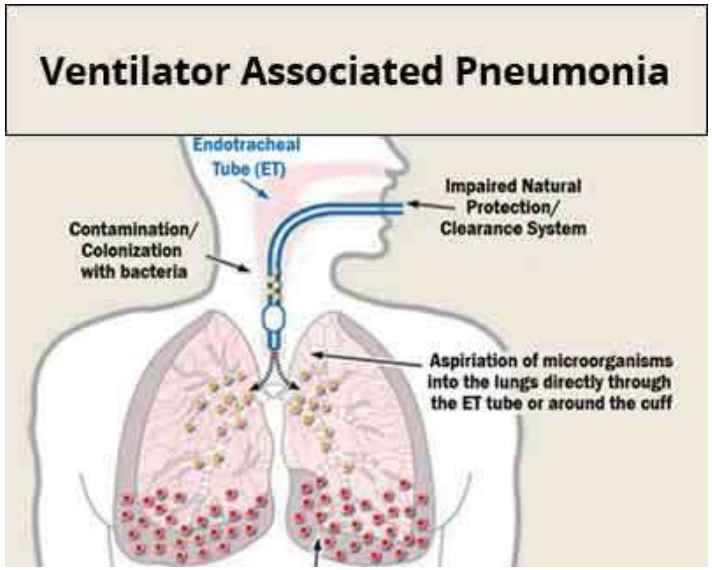
education – e.g. educate healtcare personnel (local epidemiology, risk factors)

surveillance – e.g. active surveillance of VAP in units

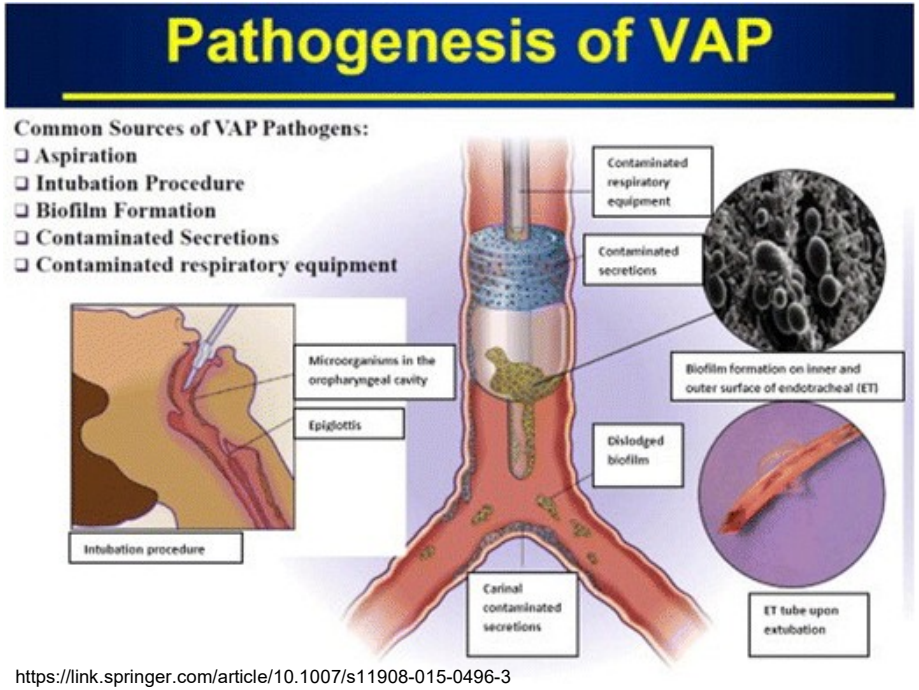
practice – e.g. implement policies and practices for disinfection, sterilization, and maintenance of respiratory equipment, patients must be maintained in a semirecumbet position, antiseptic oral care

other – e.g. use endotracheal tube with suction

Risk factors and pathogenesis of VAP



https://speciality.medicaldialogues.in/preventing-ventilator-associated-pneumonia-in-hospitals-goi-guidelines/



https://link.springer.com/article/10.1007/s11908-015-0496-3

Risk factors and pathogenesis of VAP

Traditionally, the clinical diagnosis of VAP has included a **combination of the following: clinical symptoms/signs, chest radiography, and microbiological data**.

Clinical symptoms and signs include **changes in sputum or tracheal secretions** in terms of purulence, colour and/or increasing production; cough**; temperature >38 or <36 °C**; rales or bronchial breath sounds on examination and worsening oxygenation.

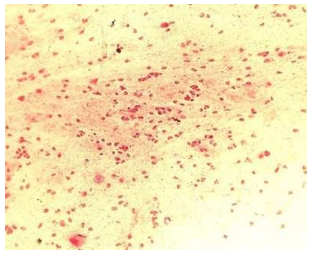
Laboratory findings include non-specific indicators of infection including leukocytosis (>12 × 10^9 WBC/L) or leukopenia (<4.0 × 10^9 WBC/L). Findings on **chest radiography (**CXR) include the development of new infiltrates or the presence of persistent and/or worsening infiltrates. Published case definitions for VAP have included a variety of combinations of these.
There is no reference standard for the diagnosis of VAP, and clinical criteria plus microbiological sampling techniques lack specificity and sensitivity when compared to the demonstration of pneumonia on histological samples obtained by either biopsy or necropsy. For example, **clinical criteria alone** have been reported to have a **sensitivity and specificity of 91 and 15 %**.

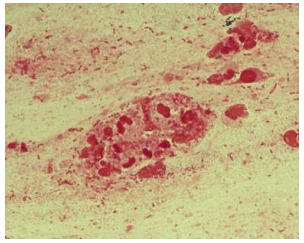
Respiratory tract sampling should be routinely conducted when there is a clinical suspicion of VAP. This can be done via **non-bronchoscopic** or **bronchoscopic techniques**. Bronchoscopic sampling includes **bronchoalveolar lavage** (BAL) or protected specimen brush (PSB), while non-bronchoscopic techniques include **endotracheal aspirates** and mini-BAL. Bacterial growth in semi-quantitative cultures is usually reported as heavy, moderate, light or no growth. Typically, **quantitative cultures** are done on BAL or PSB specimens, while semi-quantitative cultures are done on other samples such as endotracheal aspirates. If quantitative cultures are done, thresholds have been ascribed to the presence of infection as 10^4 colony forming units/mL (cfu/mL) for BAL and 10^3 cfu/mL for PSB. Although quantitative cultures are touted as being more specific for infection, a recent Cochrane analysis that included five randomized control trials (RCTs) (n = 1240 patients) found no change in mortality, days on mechanical ventilation, number of days in the ICU, or antibiotic utilization when compared to semi-quantitative cultures. In the absence of demonstrated superiority of one technique over another, the relative invasiveness of bronchoscopy and its requirement for specialized expertise and equipment, endotracheal aspirates are the preferred method of respiratory tract sampling for microbiology. There may be other indications for bronchoscopy such tracheobronchial toileting, but there is little rationale for its routine utilization for the diagnosis of VAP.

https://link.springer.com/article/10.1007/s11908-015-0496-3

Algorithm of VAP diagnosis

Small magnification (validity of sputum)





large magnification (bacterial morphology)

microscopy: Gram (G- rods and PMN)

sputum

culture: enriched culture media

Identification (MALDI)

antibiogram

Specific treatment

Categorization and prevention of NI

<u>catheter-associated urinary tract infections (CAUTIS)</u>

***infrastructure** – e.g.written guidelines for use, insertion & maintenance (aseptic techniques, records –date…), trained personnel,

***surveillance** – e.g. ID groups of patients with high risk, dg UTI

***education and training** – e.g.procedures for insertion, management and removal

***catheter insertion** – e.g. only when necessary, as long as indication persist, hand hygiene before and after manipulation of the catheter

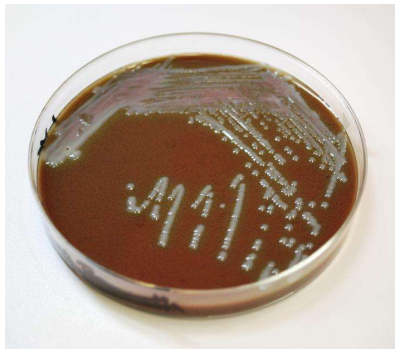
***management of indwelling catheters** – e.g. prevent movement after insertion, maintain asterile, continuosly closed drainage system, replacing by aseptic teqniques,

***prevention** – e.g. implement organization-wide program to identify and remove catheters that are no longer necesary, intermittent catheters…

Algorithm of nocomial UTI

? microscopy: Gram (PMN….), biochemistry

sputum



Identification (MALDI)

culture: enriched culture media

antibiogram

Specific treatment

Categorization and prevention of NI

surgical site infections (SSIs)

***diagnosis – swabs, aspiration – microscopy, culture, PCR**

***surveillance** – e.g. feedback on surveillance measures

***practice** – e.g. antimicrobial prophylactis in accordance with standards and guidelines, dont remove hair from operative sites unless the hair will interfere with the operation

***education** – e.g. educate surgeons and patients about SSI prevention, dont routinely use vancomycin fro prophylaxis