

**Institute for Microbiology, Medical Faculty of Masaryk University  
and St. Anna Faculty Hospital in Brno**

# **Agents of digestive system infections**

# Digestive system

- Its both ends are the „buggiest“ parts of the body
- **Normal colonic flora: 99 % anaerobes** (*Bacteroides, Fusobacterium, Clostridium, Peptostreptococcus*), **only 1 % enteric bacteria** (mostly *E. coli*) & *enterococci*

# Mouth cavity – I

## Normal flora:

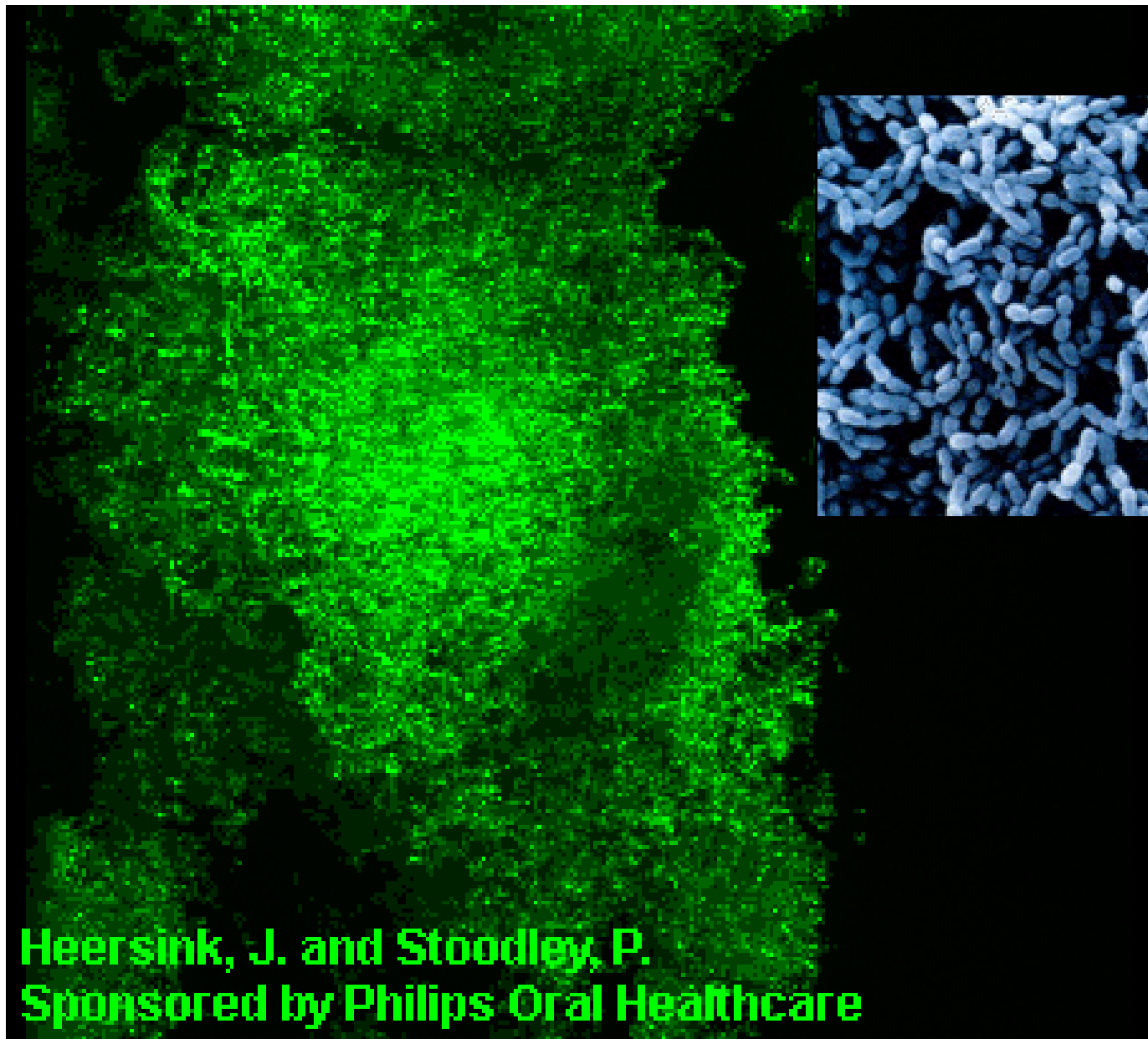
- viridans (=  $\alpha$ -haemolytic) streptococci (e.g. *Streptococcus salivarius*)
- oral neisseriae (e.g. *Neisseria subflava*)
- haemophilli of low pathogenity (e.g. *Haemophilus parainfluenzae*)

**Dental plaque:** adherent microbial layer made up from living and dead bacteria and their products together with components from the saliva

In essence, **dental plaque is a biofilm**

It cannot be washed off, only mechanically removed.

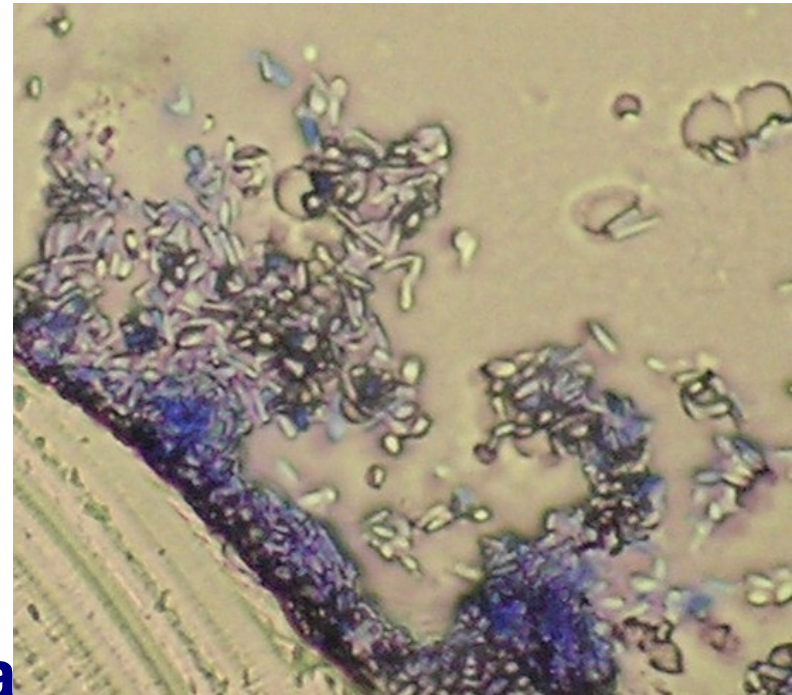




**Heersink, J. and Stoodley, P.  
Sponsored by Philips Oral Healthcare**

# Biofilm

- **Bacteria regulates the quantity of their population by regulative compounds**
- **Process – quorum sensing**
- **More resistant to**
  - **desinfectants**
  - **antibiotics**
  - **immune reaction**
- **The product of normal flora (which is positive) and pathogens as well**



# Mouth cavity – II

**Dental caries:** chronic infections caused by normal oral flora → localized destruction of tooth tissue

**Etiology:** mouth microbes (mostly *Strept. mutans*) making acids from sucrose in food

**Thrush** (in Latin soor): *Candida albicans*  
It occurs mostly in newborns

**Herpetic stomatitis:** primary infection with **HSV 1**

**Ludwig s angina:** polymicrobial **anaerobic** infection of sublingual and submandibular spaces (*Porphyromonas, Prevotella* etc.)

# Herpetic stomatitis





# Thrush



[http://www.mydochub.com/images/oral\\_thrush.jpg](http://www.mydochub.com/images/oral_thrush.jpg)

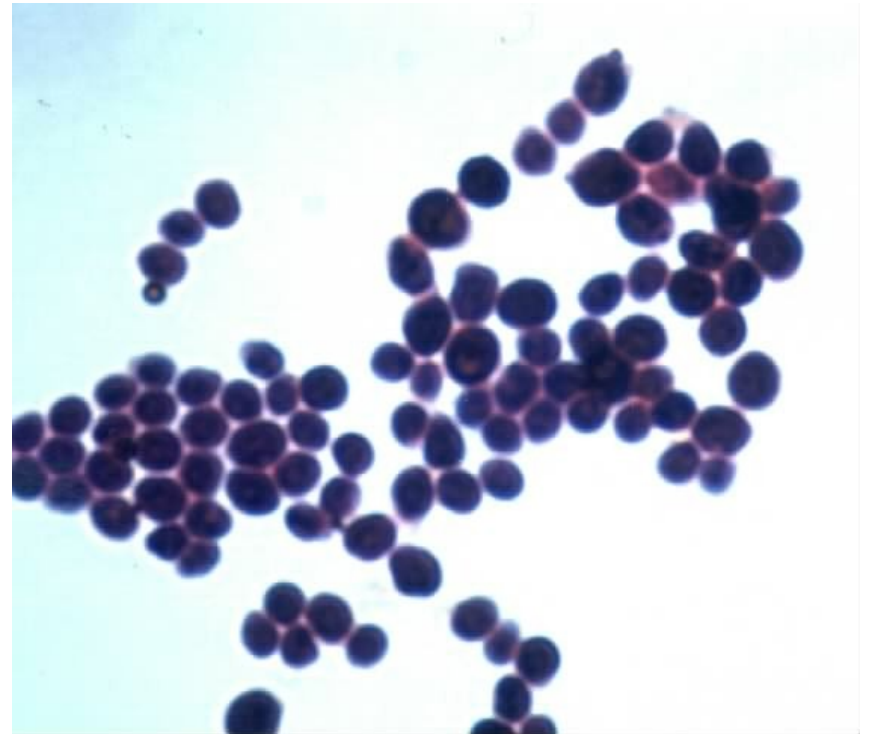
Oral thrush



 ADAM.

<http://www.clarian.org/ADAM/doc/graphics/images/en/17284.jpg>

# *C.albicans*



[www.medmicro.info](http://www.medmicro.info)

# Oesophagus

Infections **never** in previously healthy individuals

**Only** in severely immunocompromised persons (AIDS):

- *Candida albicans*
- Cytomegalovirus (CMV)

# Stomach

**Stomach = sterile, killing by means of HCl most of swallowed microbes**

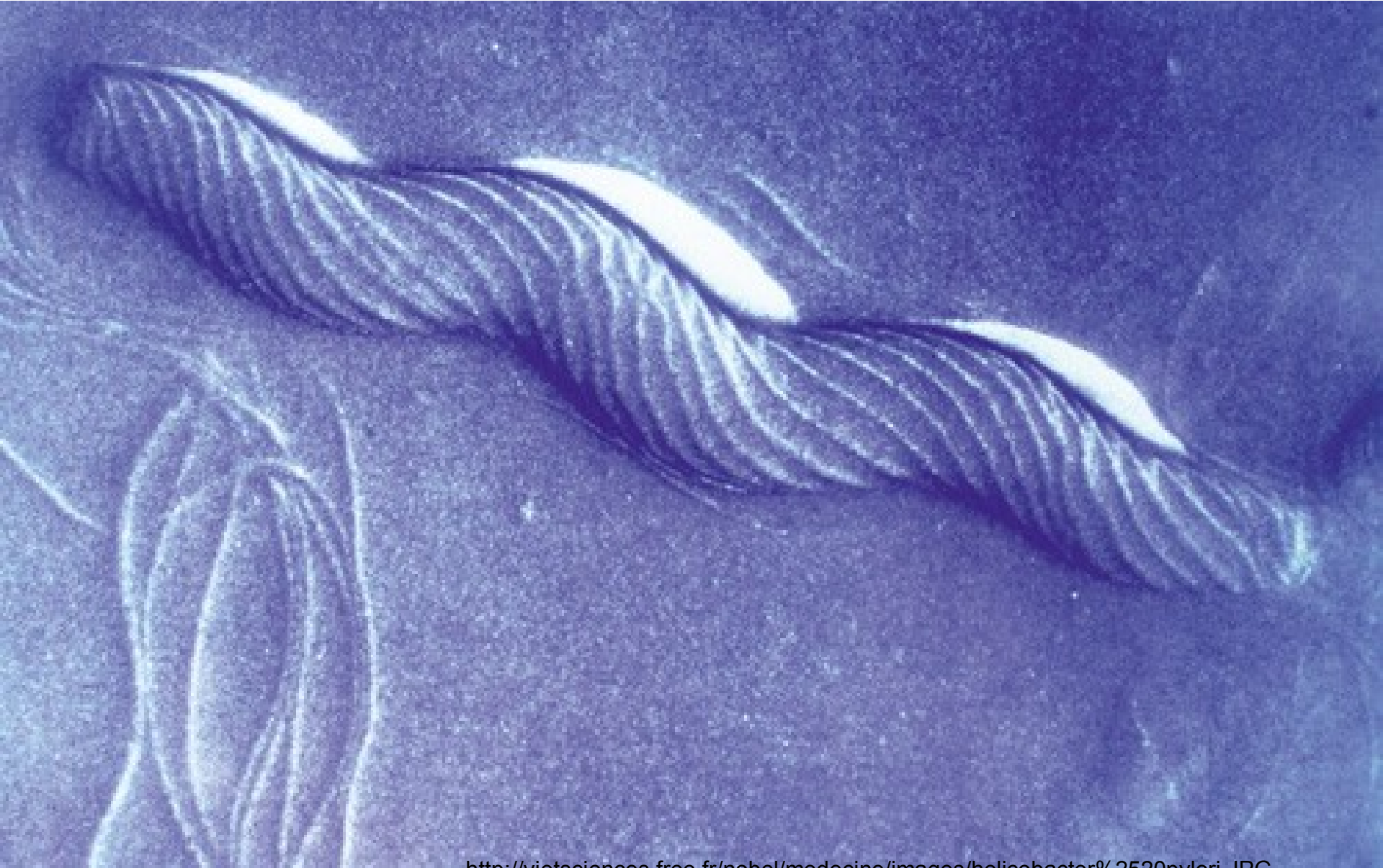
## ***Helicobacter pylori***

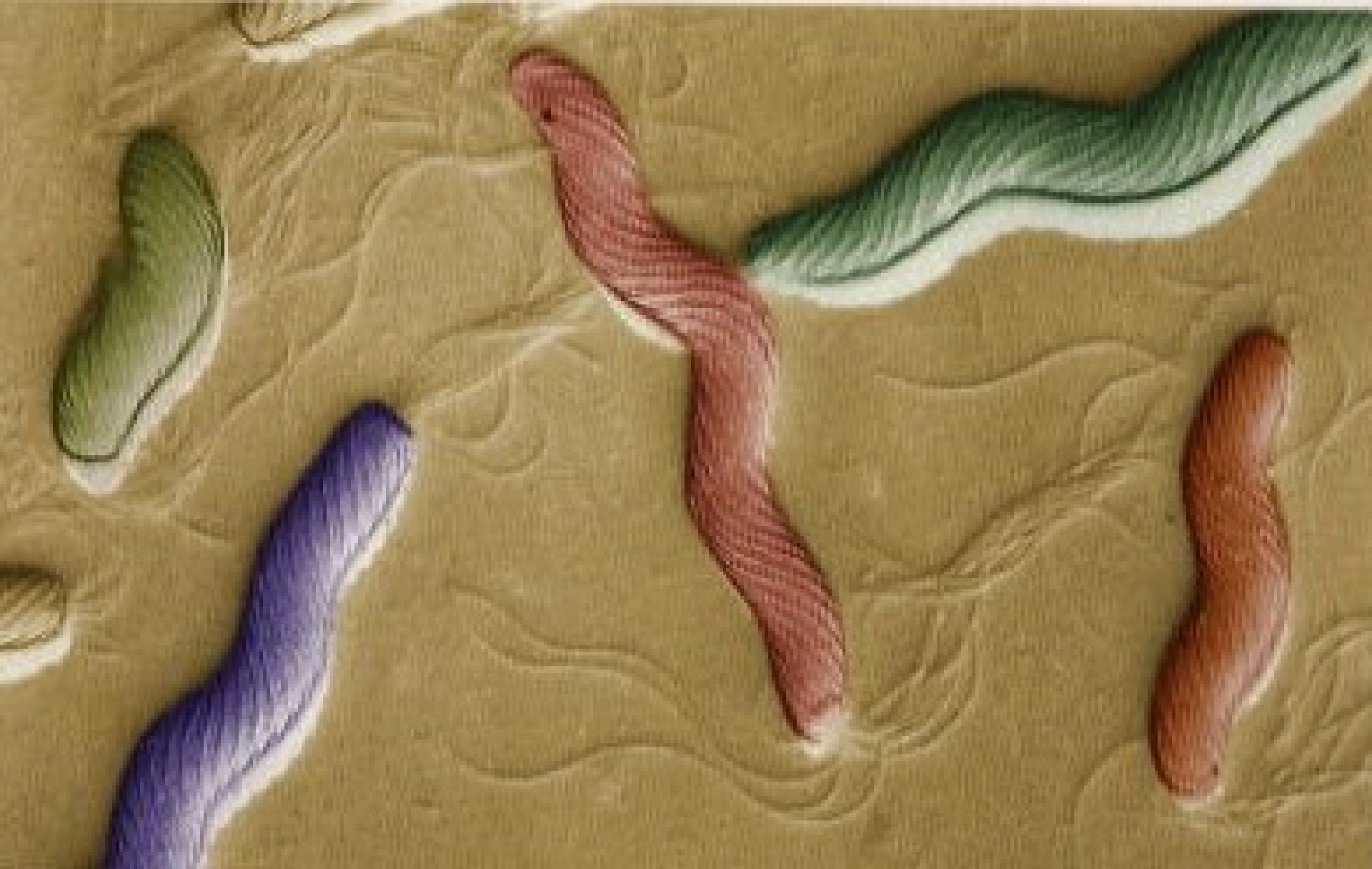
**produces a potent urease and by splitting tissue urea it increases pH around itself (1 molecule of urea  $\rightarrow$  1 CO<sub>2</sub> + 2 NH<sub>3</sub>)**

***H. pylori* causes**

- chronic gastritis**
- peptic ulcers**

# *Helicobacter pylori*





[www.univie.ac.at/hygiene-aktuell/helicobacter.jpg](http://www.univie.ac.at/hygiene-aktuell/helicobacter.jpg)

# Biliary tree & the liver

**Acute cholecystitis** (colic, jaundice, fever): obstruction due to gallstones

Etiology: intestinal bacteria (*E. coli* etc.)

Complication: **ascending cholangitis**

**Chronic cholecystitis**: the most important is *Salmonella Typhi* (carriers of typhoid fever)

**Granulomatous hepatitis**: Q fever, tbc, brucellosis

**Parasitic infections of the liver**: **amoebiasis** (*Entamoeba histolytica*: liver abscess), **malaria** (the very first, clinically silent part of the plasmodial life cycle), **leishmaniasis** (*Leishmania donovani*: kala-azar), **schistosomiasis** (eggs of *Schistosoma japonicum*)

# Systemic infections which start in the digestive tract

**Enteric fever** (typhoid fever and  
paratyphoid fever): *Salmonella* Typhi,  
*Salmonella* Paratyphi A, B and C

**Listeriosis:** *Listeria monocytogenes*

**Peritonitis:** colonic flora

**Viral hepatitis:** HAV, HBV, HCV, HDV, HEV



# Bacterial agents of diarrhea – I

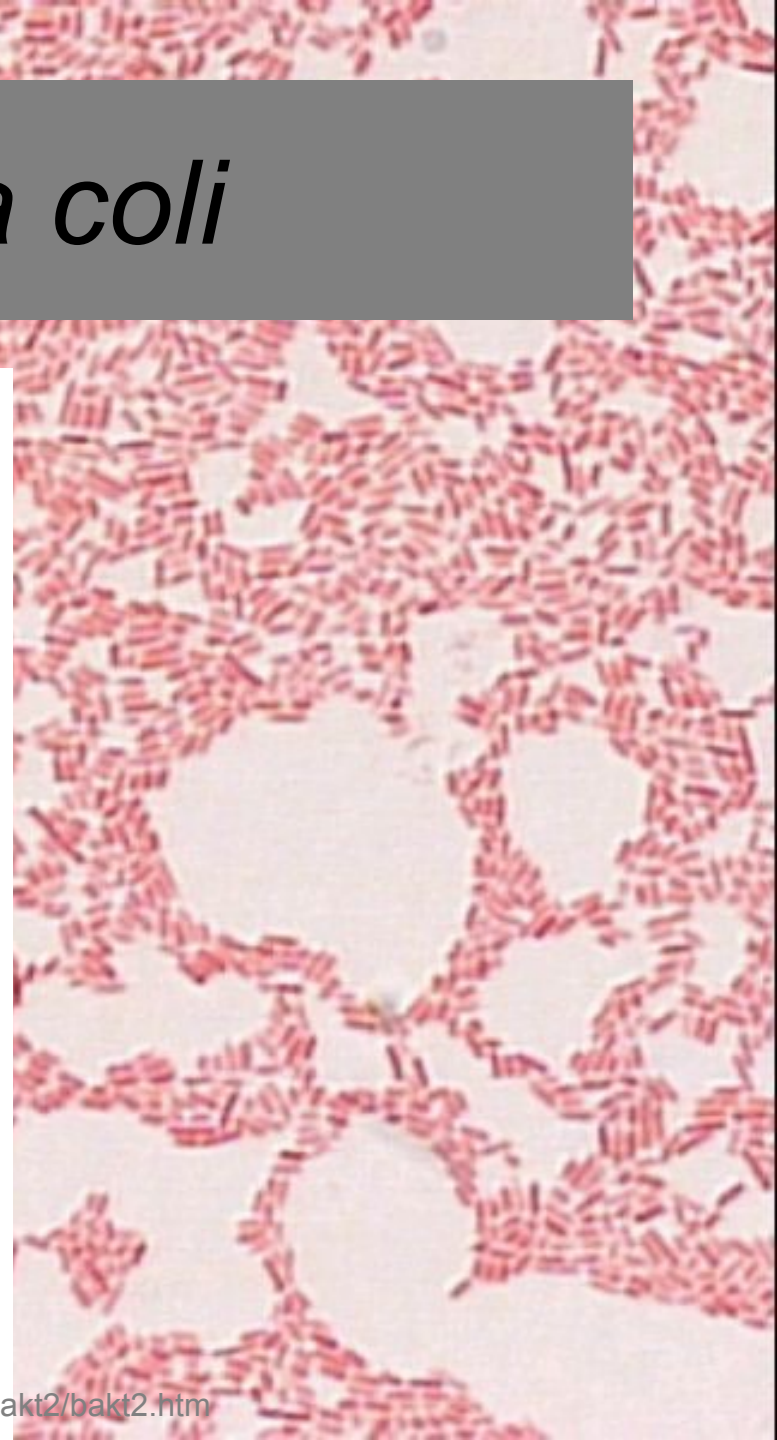
## *Escherichia coli*

Most *E. coli* strains (approx. 1 %) normal intestinal flora

- beneficial
- non-pathogenic in the intestine

Some *E. coli* strains pathogenic in GIT

# *Escherichia coli*



# Bacterial agents of diarrhea – II

## *Escherichia coli* strains causing diarrhea:

- **ETEC** (enterotoxigenic *E. coli*): children in developing countries, traveller's diarrhea, toxins
- **EPEC** (enteropathogenic *E. coli*): O55, O111; infants; disruption of microvillus structure
- **EIEC** (enteroinvasive *E. coli*): invasion of colonic cells
- **EHEC** (enterohaemorrhagic *E. coli*): O157:H7; 2 cytotoxic Shigatoxins, hemorrhagic colitis & hemolytic-uremic syndrome

# Salmonella - MAL agar

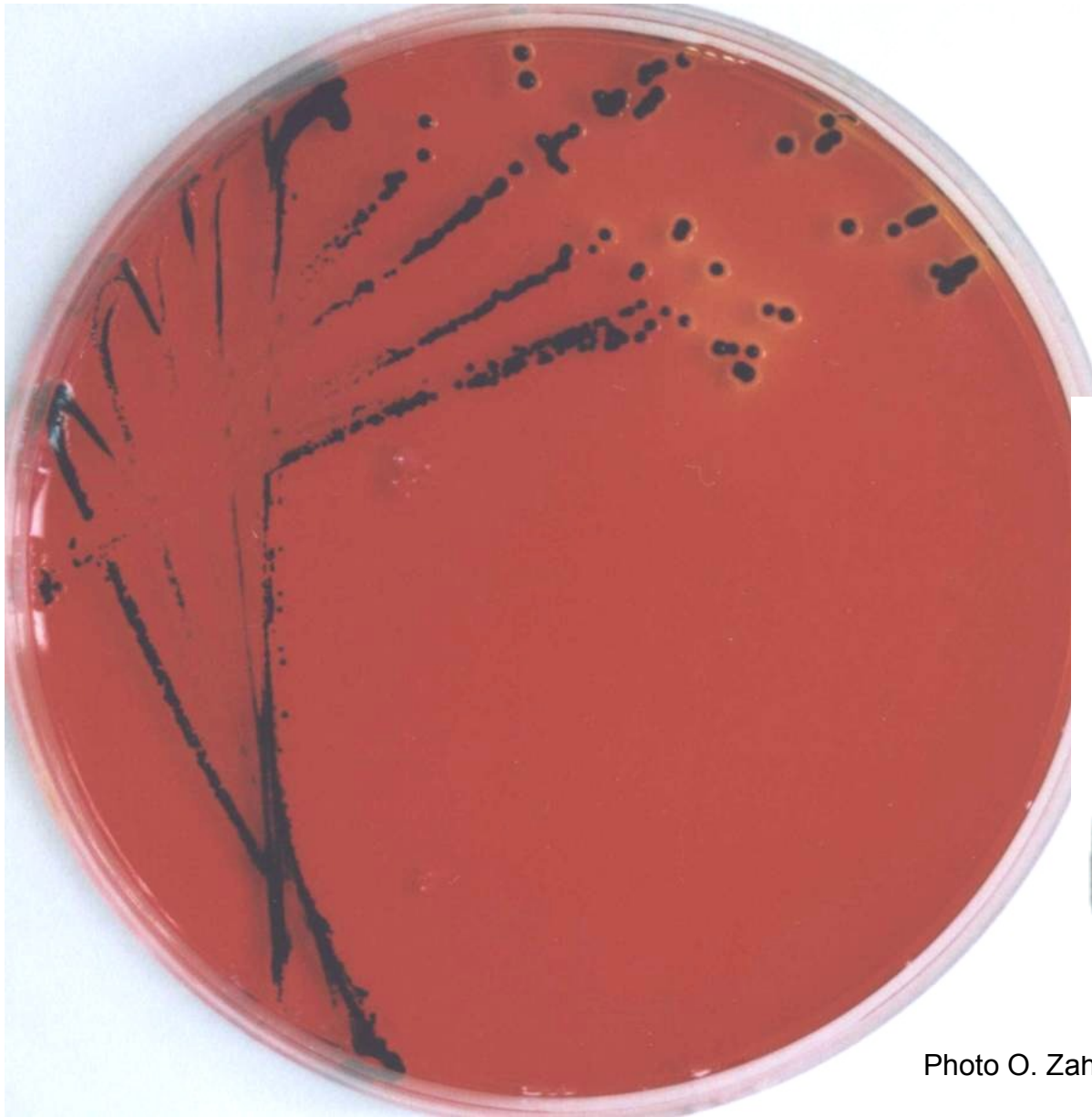


Photo O. Zahradníček.

# Bacterial agents of diarrhea – III

## A) **Salmonella systemic infections (enteric fever):**

S. Typhi, S. Paratyphi A – C (humans)

Gut invasion and **infection becomes generalized** → no diarrhea, pronounced **fever**, detection in **blood, urine and stool**, in susp. carriers in duodenal fluid, antibiotics

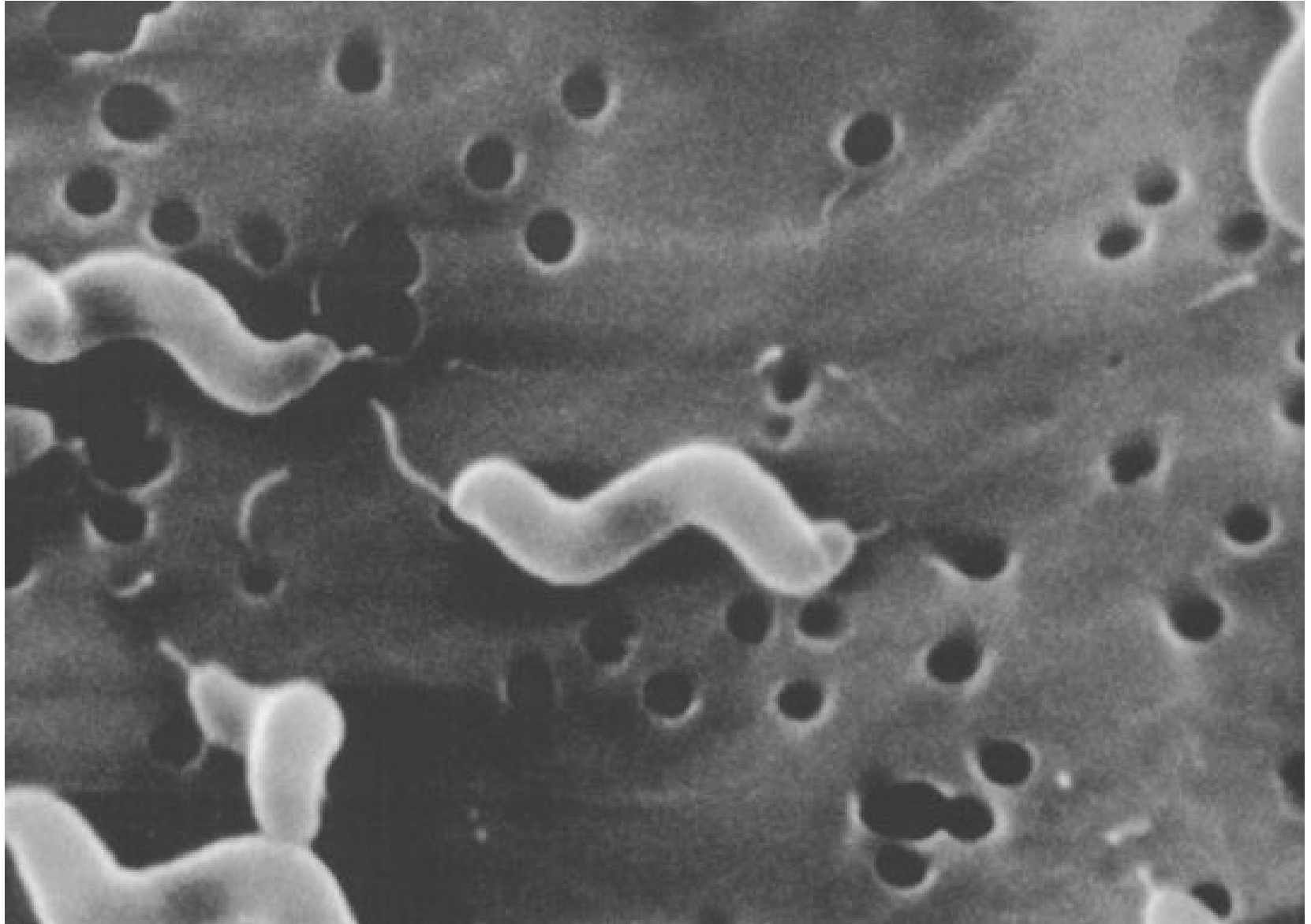
## B) **Salmonella gastroenteritis (salmonellosis, reservoir: poultry & animals):**

>4.000 serotypes – e.g. S. Enteritidis

**Localized** in ileocaecal region → **diarrhea**, nausea & vomiting, abdominal pain, temperature, examination of **stool only**

Treatment: symptomatic, **no antibiotics**

# *Campylobacter jejuni*



# Bacterial agents of diarrhea – IV

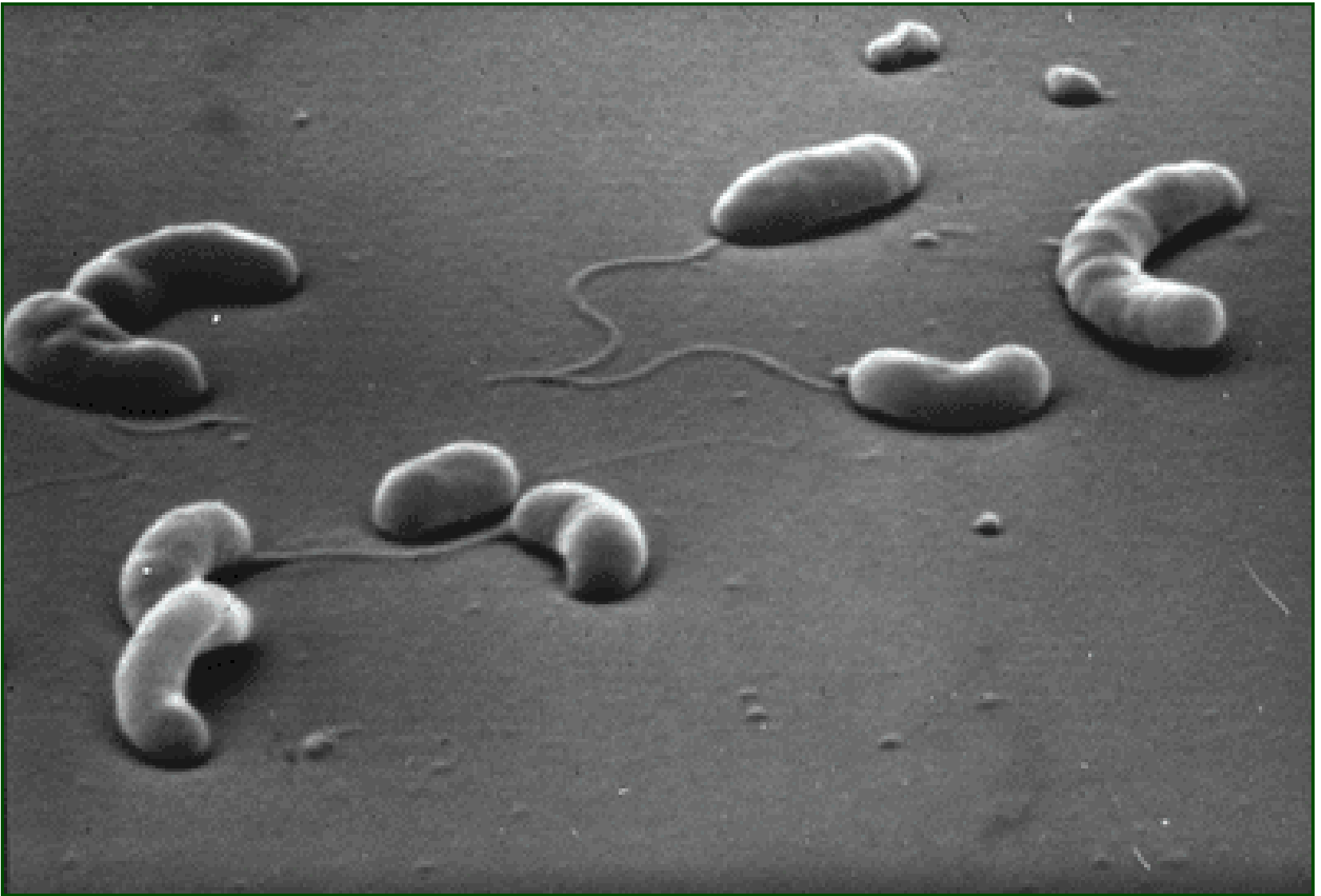
## *Campylobacter jejuni*

invades jejunal epithelium, reservoir: poultry,  
cultured on a special medium, in reduced oxygen,  
at 42 C

## *Shigella sonnei, S.flexneri, S.boydii, S.dysenteriae*

- very low infectious dose → epidemic outbreaks
- transmitted only among human beings
- invasion - cells of colon and rectum
- **bacterial dysentery**

# *Vibrio cholerae*





# Bacterial agents of diarrhea – V

## *Yersinia enterocolitica*

- gastroenteritis, in children also mesenteric lymphadenitis (mimicking acute appendicitis)
- vector: contaminated food, multiplies at 4 °C

## *Vibrio cholerae*

Cholera toxin activates adenylate cyclase → hypersecretion of water & electrolytes → death by dehydration/electrolyte abnormalities

*V. cholerae* flourishes in water & causes epidemics

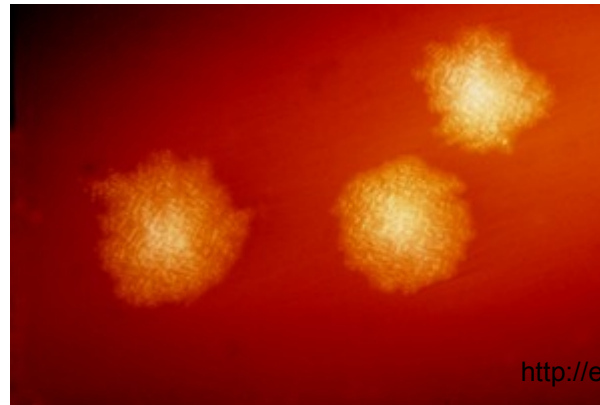
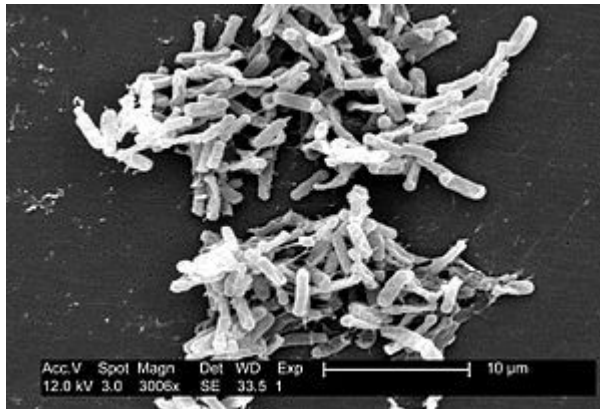
# Diarrhoea during antibiotic therapy

## *Clostridium difficile*:

**pseudomembranous colitis** frequently after **clindamycin, cephalosporines** (virtually after every ATB), hypervirulent serotype O27

Patients contaminate the hospital environment with resistant spores.

Treated with **metronidazol** or **vancomycin**

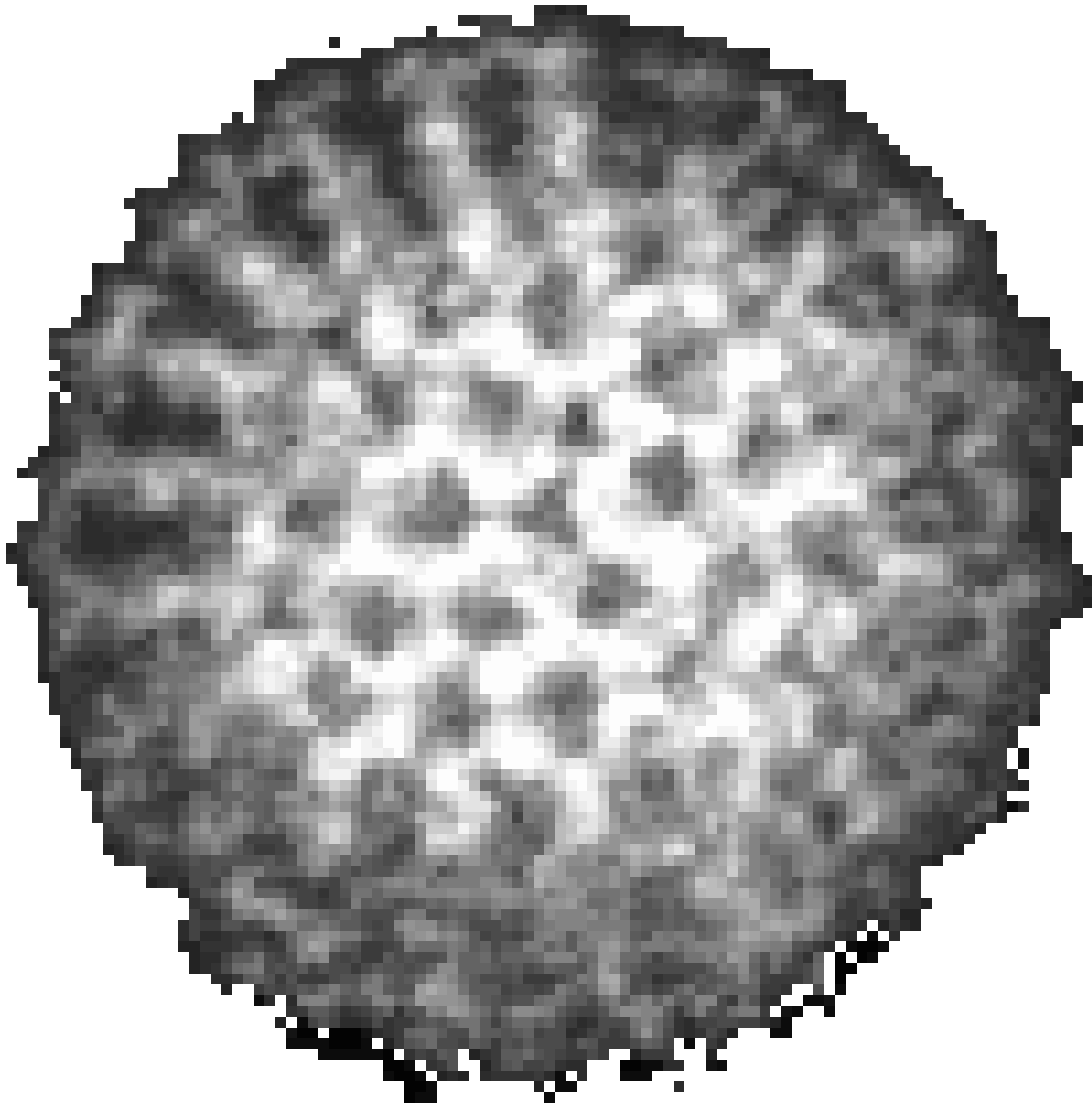


Direct proof of the **Cdiff toxins essential**, *C. difficile* can be found in healthy people



Proof of the toxin A in *C. difficile*. Photo: MÚ archive

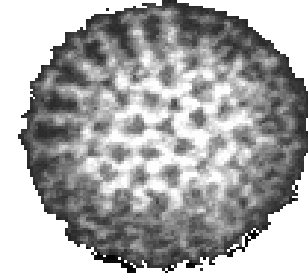
# Rotavirus



[http://web.uct.ac.za/depts/mmi/s\\_tannard/emimages.html](http://web.uct.ac.za/depts/mmi/s_tannard/emimages.html)

# Viral agents of diarrhea

Generally: small, acid- and bile-resistant non-enveloped viruses



Rotaviruses (*Reoviridae* family)

serious diarrhea of young children, epidemics in winter, vaccination

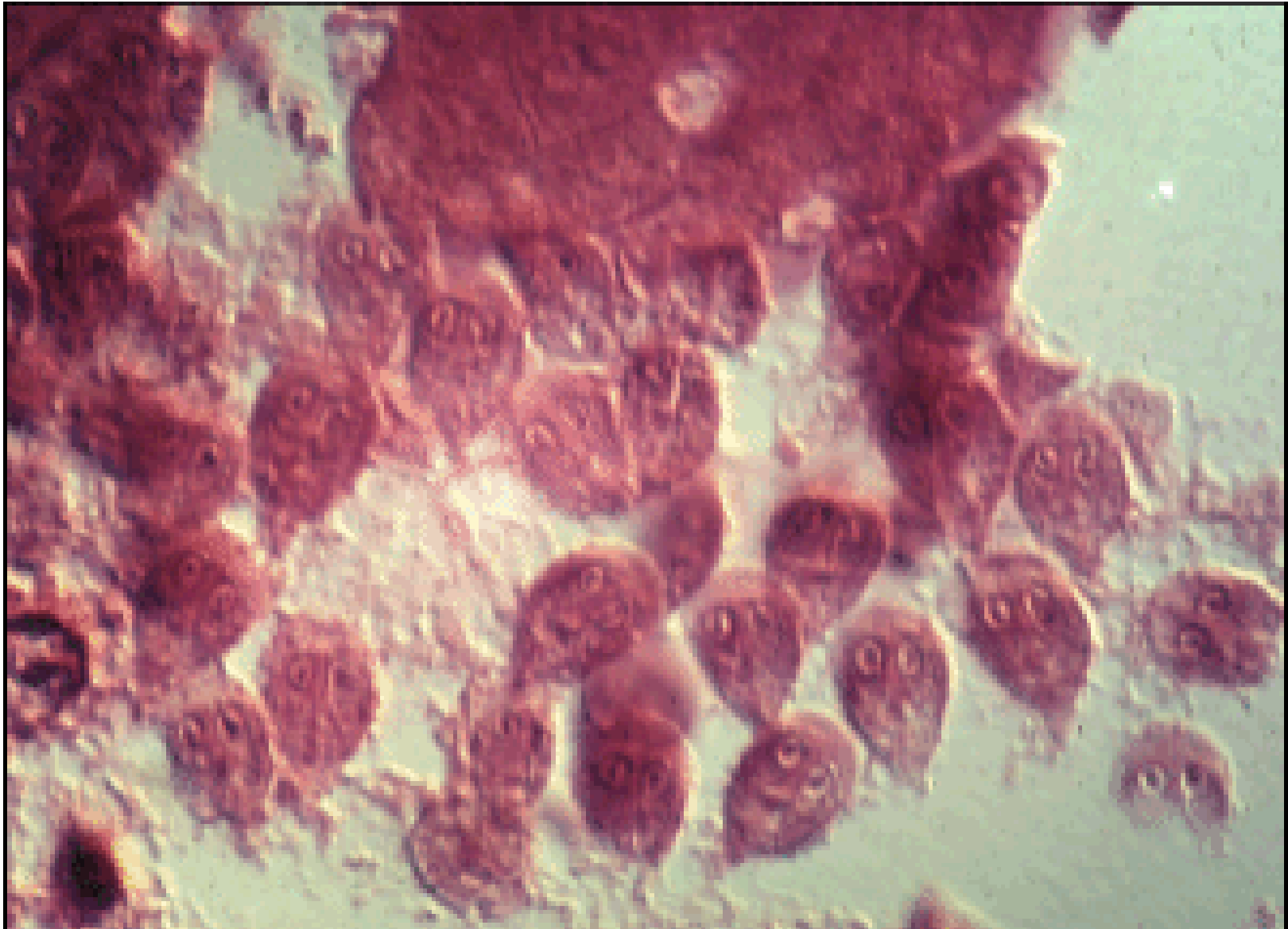
Noroviruses and **sapoviruses** (formerly agents Norwalk and Sapporo, *Caliciviridae* family)

epidemics in children and adults, in hospitals

**Astroviruses** (star-shaped virions)

**Adenoviruses** type 40 and 41

# Lamblia



# Ascaris lumbricoides egg

Egg



Fertile egg (wet mount 400X)

# Parasitic agents of diarrhea

## Protozoa:

*Entamoeba histolytica*: amoebic dysentery

*Giardia lamblia*: giardiasis

*Cryptosporidium parvum*: cryptosporidiosis

## Helminths in the small intestine:

*Ascaris lumbricoides* (human roundworm)

*Strongyloides stercoralis* (threadworm)

*Taenia saginata* (beef tapeworm), *T.solium* (pork tapeworm)

*Hymenolepis nana* (dwarf tapeworm)

## .....in the large intestine:

*Enterobius vermicularis* (pinworm)

*Trichuris trichiura* (whipworm)



# Food poisoning

Intoxication due to a toxin preformed in the food

*Staphylococcus aureus*: heat-stable enterotoxin

*Clostridium perfringens*: heat-labile enterotoxin

*Bacillus cereus*: heat-stable enterotoxin and vomiting toxin (mostly in rice)

*Clostridium botulinum*: heat-labile neurotoxin



**Harmenszoon Rembrandt van Rijn (1606-1669)  
Anatomy Lecture of Doctor Tulp (1632)**

