# THE MODIFICATIONS OF THE BACTERIAL HUMAN ENDOGENOUS FLORA DURING HOSPITALISATION

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***Cuvinte cheie:*** *flora bacteriană, colonizare bacteriană, mediu spitalicesc*

***Rezumat:*** *Microbii se află peste tot în biosferă, iar prezența lor invariabil afectează mediul în care se dezvoltă. Fiecare astfel de organism trebuie să găsească în mediul său toate substanțele necesare pentru a produce energie şi biosinteză celulară, iar celula gazdă trebuie să furnizeze în final nutrienții necesari pentru aceste bacterii. Bacteriile care formează flora normală interacționează simbiotic cu gazda lor, existând mai multe celule bacteriene pe suprafețele corpului uman (inclusiv cele din tractul gastrointestinal) decât celulele umane care alcătuiesc acest organism. Flora normală, ca şi bacteriile "contaminante" din mediu se găsesc pe suprafețele organismului, sângele şi țesuturile interne fiind sterile. Dacă o bacterie depăşeşte aceste suprafețe, poate să apară o infecție, dar aceasta nu conduce automat la o boală infecțioasă; alți factori, cum sunt calea de intrare, numărul de microorganisme infectante şi (cel mai important) statusul mecanismelor de apărare ale gazdei, joacă un rol important în evoluția unei infecții.*

***Keywords:*** *bacterial flora, bacterial colonization, hospital environment*

***Abstract:*** *The microbes are everywhere in the biosphere and their presence invariably affects the environment that they are growing in. Every organism must find in its environment al of the substances required for energy generation and cellular biosynthesis and the host cell must ultimately provide the nutritional requirements of its resident.Bacteria that form the normal flora have a full range of symbiotic interactions with their host and there are many more bacterial cells on the surface of a human body (including the gastrointestinal tract) than there are human cells that make up the body.The normal flora, as well as any "contaminating" bacteria from the environment, is all found on the body surfaces; the blood and internal tissues are sterile. If a bacteria breaches one of these surfaces, an infection may occur, but infection does not necessarily lead to infectious disease; other factors, such as the route of entry, the number of infectious bacteria, and (most importantly) the status of the host defences, play a role in determining the outcome of infection.*

**SCIENTIFICAL ARTICLE PREDOMINANTLY THEORETICALLY**

**Normal Flora in Humans**

Bacteria are the most numerous micro-organisms that compose the normal flora in humans. (1) (Table no.1).

**Bacterial Flora in Hospital Conditions**

The normal flora undergoes major changes in terms of hospitalization, under the influence of multiple factors, especially the environment of the patient and the presence or not of antibiotic therapy which, was previously made. Moreover, the hospital environment is colonized by a number of micro-organisms present in water or even in sterile liquids (Pseudomonas, Acinetobacter, Mycobacterium) on linen, equipment, food, different surfaces or in air (2).

The hospital germs presence is essential for the development of the nosocomial infections. The virulence of these bacteria varies from one medical service to another, but all are characterized by the existence of specific antibiotic resistance phenotypes, which we do not meet at the bacteria that cause communitarian infections (3). The antibiotic resistance is specific to each medical or surgical service, depending largely on the type and duration of the use of certain antibiotics during the service.

**Oro-pharyngeal flora**: in the first five days of hospitalization, the saprophytic flora of oro-pharynges is replaced by a flora characterized by the presence of a specie or the predominance of several bacterial species*: Klebsiella, E.coli, Proteus, Enterobacter, Serratia, Acinetobacter, Pseudomonas, more rarely fungs.* The oro-pharyngeal colonization is rapid: 22% of patients are colonized in the first day, 40% by 5$^{th}$ day (3).

The risk factors for colonization are several, the main illness and its seriousness having importance, the presence of chronic alcoholism, the pre-existing diabetes, the age, the presence of surgery, the antibiotic therapy, which itself is a risk factor in the selection of the resistant strains.

**Intestinal flora**: its modifications are usually the result of the underlying disease (obstruction, peristalsis changes, hypo or achlorhydria, gastric bleeding), which causes the upstream flora to become a *faecal flora*. The exogenous factors have also contributed: prolonged fasting and exclusive nutrition, antibiotics therapy. The rupture of the balance between dominant and dominated bacteria causes the cancellation of the barrier effect. This allows the rapid deployment and proliferation of the potentially pathogenic species, which are responsible for the appearance of the diarrhoeal diseases until the sepsis syndrome.

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Article received on 12.07.2010 and accepted for publication on 18.10.2010
ACTA MEDICA TRANSILVANICA December 2010; 2(4) 305-307

**Table no. 1. Bacteria which are normally found on different areas of the body**

| Bacterium | Skin | Connective Tissue | Nose | Pharynx | Mouth | Lower gastrointestinal tract | Urethra | Vagina |
|---|---|---|---|---|---|---|---|---|
| Staphylococcus epidermidis | ++ | + | ++ | ++ | ++ | + | ++ | ++ |
| Staphylococcus aureus* | + | +/- | + | + | + | ++ | +/- | + |
| Streptococcus mitis | | | | + | ++ | +/- | + | + |
| Streptococcus salivarius | | | | ++ | ++ | | | |
| Streptococcus mutans* | | | | + | ++ | | | |
| Enterococcus faecalis* | | | | +/- | + | ++ | + | + |
| Streptococcus pneumoniae* | | +/- | +/- | + | + | | | +/- |
| Streptococcus pyogenes* | +/- | +/- | | + | + | +/- | | +/- |
| Neisseria sp. | | + | + | ++ | + | | + | + |
| Neisseria meningitidis* | | | + | ++ | + | | | + |
| Enterobacteriaceae*(Escherichia coli) | | +/- | +/- | +/- | + | ++ | + | + |
| Proteus sp. | | +/- | + | + | + | + | + | + |
| Pseudomonas aeruginosa* | | | | +/- | +/- | + | +/- | |
| Haemophilus influenzae* | | +/- | + | + | + | | | |
| Bacteroides sp.* | | | | | | ++ | + | +/- |
| Bifidobacterium bifidum | | | | | | ++ | | |
| Lactobacillus sp. | | | | + | ++ | ++ | | ++ |
| Clostridium sp.* | | | | | +/- | ++ | | |
| Clostridium tetani | | | | | | +/- | | |
| Corynebacteria | ++ | + | ++ | + | + | + | + | + |
| Mycobacteria | + | | +/- | +/- | | + | + | |
| Actinomycetes | | | | + | + | | | |
| Spirochetes | | | | + | ++ | ++ | | |
| Mycoplasmas | | | | + | + | + | +/- | + |

++ =almost 100%  + = about 25%  +/- = seldom (under5%)  * = potentially pathogenic

*After Todar Kenneth, Textbook of Bacteriology (1)*

**Table no. 2. Intestinal contaminant flora**

| | Flora in pathological circumstances | Potentially dangerous germs |
|---|---|---|
| Oesophagus | Mechanical or functional obstruction:<br>- anaerobic streptococci<br>- bactericides<br>- faecal flora | Ø Salivary oral flora<br>Ø Anaerobic streptococci<br>Ø Oral bactericides<br>Ø Faecal anaerobes |
| Stomach | Decrease of gastric acidity: resident oral flora<br>Gastrointestinal colic fistula:resident faecal flora | Ø Oral flora |
| Duodenum<br>Proximal intestine | Interventions for ulcer: resident oral flora<br>Mechanical obstructions: oral and resident faecal flora | Ø Oral flora<br>Ø Faecal flora |
| Distal ileum<br>Appendicitis<br>Colon<br>Rectum | Ulcerative colitis:<br>- resident aerobic flora (e.g. coli, lactobacilli, streptococci, yeasts)<br>- anaerobic flora (clostridium, bactericides)<br>Mechanical obstruction:<br>- Resident faecal flora | Ø Fragile bactericides<br>Ø e. coli<br>Ø streptococci<br>Ø clostridium perfringens |
| Gallbladder | Cystitis: aerobe coliforms<br>Obstruction:<br>ü bactericides<br>ü clostridium | Ø Aerobe coliforms |
| Pancreas | Hemorrhagic pancreastitis: aerobe coliforms<br>Abscesses: aero-anaerobe flora | Ø Aerobe coliforms |

*After I. Vasile and D. Mogoş, in Nosocomial Infections in General Surgery, Craiova, 2001(3)*

*The occlusive pathology and faecal stasis* determine the logarithmic increases of the amount of germs, which greatly increase the postoperative infectious risk. Whatever the type of occlusion is, there are two main pathogenesis mechanisms: the obstacle fluid-gas upstream distension and the parietal necrosis. Both lead to septic shock and decrease of the circulating blood volume, an association which is extremely severe in the human pathology. The germs and their toxins are the decisive element; the most feared germs which produce toxins are: clostridia, beta haemolytic streptococcus, gram negative bacilli, anaerobic fragile cal Bactericides.

In Table no. 2, the intestinal flora appeared in pathological circumstance is centralized.

**Skin flora**: damaged skin (by burning, bedsores, wounds, etc.) causes the implantation and development of the hospital germs, especially the *gram negative bacilli*. An occlusive bandage applied on dry skin (e.g. the forearm), turns it into a muggy environment, conducive to rapid microbial multiplication of resident flora. On the other hand, in the case of the wounds, the occlusive dressings accelerate the epithelium,

reduce the inflammation and pain and stimulate the healing for chronic wounds, so it is recommended their use in the treatment. After seven days of hospitalization, the colonization appears with *gram negative bacilli* and ***diphtheroids*** (4). In the hospital are other risk factors as: sowing manual with pathogen or opportunistic germs (*Pseudomonas, Acinetobacter* and *SAH*); the prolonged use of antiseptics and local antibiotics (colonization with opportunistic germs); the antibiotics therapy determines the appearance of the resistant strains.

**Vaginal flora**: the risk factors for its modification in the hospital are antibiotics therapy and the presence of the surgical operations. The encountered germs are species of Enterococci, Enterobacter and Pseudomonas.

## CONCLUSIONS

After a few hours from the admission, the normal flora of the patients begins to change through the acquisition of the characteristic strains for the bacterial pool of the hospital, where these people came.

The most patients who develop nosocomial bacterial infections are prone to infection due to the invasive interventions or because of their defence mechanisms compromised due to underlying disease. The pathogenic process of the development of an infection involves several steps, because the immune-competent host has a number of local barriers to prevent the infection and the parasite is forced to use specific strategies for each phase. These are strategic details are unique for each category of organisms, even if there are remarkable conceptual similarities to bacteria, viruses and parasites (5).

The knowledge of both the normal bacterial flora and the specific flora to a particular hospital service have great importance for a prompt treatment of infections, the empirical antibiotics therapy should be equally necessary as the treatment of the shock, of the hypo-ventilation or of other complications which may occur (6). The antibiotics therapy must be according to the local epidemiological patterns of microbial susceptibility.

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