The Pathogenic Microflora and Microbial Interactions in Oral Cavity in Dogs

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Abstract
The existing microorganisms in the oral cavity are not harmless and they can express various pathogenic factors. These pathogenic factors are represented by the morphostructural and functional characteristics that ensure germ aggression against the host. They are represented by a particular chemical structure of the germs, different morphological characteristics, or different functional properties caused by certain enzymes or toxic discharge. The expression of pathogenicity of microorganisms is achieved when there is an imbalance that can affect the dynamics of the relations between macro and micro-organisms, the interaction of host antimicrobial capacities and pathogenic proprieties of microorganisms in favor of the latter. Depending on their pathogenic proprieties these bacteria can be: pathogenic, moderately pathogenic, highly pathogenic.

The objective of this study is to reveal the pathogenic proprieties and the interactions of the oral cavity bacteria.

Keywords: antimicrobial capacities, bacteria, host, pathogenic factors

Introduction

After their characteristics and their role in pathogenesis, the pathogenicity factors can be divided into two categories: factors and conditions that favour the multiplication of germs in the fluids of the body, tissues and cells, defined as virulent factors and the others are the toxicity factors that confer toxic properties to the germs [1].

The imbalance between the commensal and pathogenic microflora in the oral cavity occurs when there is a dysfunction in the local system of antimicrobial defense. This is achieved by genetic variations that confer temporary pathogenic capacity or can be caused by antimicrobial therapy that changes the oral cavity microbiota.

Depending on the predominance of one or other of the pathogenic functions the microbes can be classified into:

- virulent species which express their pathogenicity by the ability to multiply in the body's cells and humors;
- exclusive toxigenic species which do not have virulent functions and therefore cannot multiply in the fluids of the body and tissues - pathogenicity is only due to toxic secretions that they develop;
- toxigenic and virulent species - this category includes most bacteria and microscopic fungi [1].

The invasive ability of bacteria (virulence) is the basis for the colonization process (adhesion and initial multiplying), which facilitates the production of extracellular substances, and the invasive ability to fight against the body's own defense mechanisms.

In conclusion, the virulence capacity means the ability of microbes to multiply in the body's cells and humors. The capacity of virulence varies from
one species to another and is considered as virulence factor [2]. Virulence factors are represented either by certain structural components that favour the adhesion of germs to the cell surfaces of organs and tissues, where they begin the process of multiplication, colonization, or by agresinic secretions through germs disseminate into the body, so called the invasion step [1, 2]. The toxicity is given by the toxins that are important factors of pathogenicity of the bacteria and fungi. From the structural point of view there are two groups of toxins:
- endotoxins with a complex structure, such as lipopolysaccharides;
- exotoxins of protein nature.
Endotoxins are endocellular components of Gram-negative bacteria. These are macromolecular components that are not diffusible in the environment, and result from the disintegration of the bacteria by physical or chemical means. Exotoxins are produced by bacterial cell secretion capable of producing alone specific morphological and functional alterations [1]. In the oral cavity the virulence of microorganisms is expressed after their adherence on the dental soft and hard tissues after colonization and multiplication.

**Adherence of bacteria.**
Adherence of bacteria on the surface of dental tissues is achieved with the participation of adhesion factors [1, 3]. The factors of adhesion are: adhesins, receptors, lectins, ligands, mucus, fimbriae (type I fimbriae) pili (pili type 4), the S surface, glycopolyvalent bacterial capsule, lipopolysaccharides, teichoic and lipoteichoic acids [3]. Adhesins are proteic, lipoproteic or glycoproteic structural components, by means of which the germs adhere on the basis of their electrostatic complementarity, but differ from one group of microorganisms to another.

Cell receptors are structural surface components, that correspond to the different types of adhesins, which largely determine the species and tissue specificity of bacterial adhesion [1].

**Feno et al.** [4] showed that the virulence of the bacterium *Streptococcus parasanguis* is given by the Fim A adhesin, and for the *Streptococcus sanguis* by the Ssa adhesin [5]. It is considered that these adhesins have a very important role in spreading infection in the oral cavity and can produce endocarditis. *Fusobacterium nucleatum* is considered an adherent bacteria with the ability to enter different cells of the host which favour the attachment of other pathogens of the mouth which plays an important role in the formation of dental plaque. It has been shown that Fad A adhesin has the ability to bind to the cellular proteins of the mucosa of the oral cavity [6].

**Colonization and multiplication.** Microbial community development (i.e. phase of colonization and multiplication) in the oral cavity assumes a permanent communication and competition between about 500 bacterial species belonging to this bacterial community.

**Kolenbrander** and **London** highlighted that these interactions are responsible for the formation of bacterial plaque [10]. They highlighted the bacterial communication between: *Actinobacillus actinomycetemcomitans*, *Actinomyces israelii*, *Actinomyces naeslundii*, *Capnocytophaga ochracea*, *Capnocytophaga sputigena*, *Eikenella corrodens*, *Eubacterium spp*, *Fusobacterium nucleatum*, *Haemophilus parainfluenzae*, *Porphyromonas gingivalis*, *Prevotella denticola*, *Prevotella intermedia*, *Prevotella loeschei*, *Propionibacterium acnes*, *Fluegge Selenomonas*, *Streptococcus gordonii*, *Streptococcus mitis*, *Streptococcus oralis*, *Streptococcus sanguis*, *Treponema spp*, and *Veillonella atypica* [10, 11]. In the first minutes after cleaning, at the surface of the enamel is formed a film that is the source of the receptors recognized by the primary colonizers of the dental plaque. These receptors are represented by mucins, agglutinins, proteins, enzymes and alpha-amylase.
Each receptor is specific for a particular species of bacteria in the oral cavity. Kolenbrander et al. [10, 11] have been represented a space-time model of bacterial colonization in the oral cavity and they have shown this possible communication between primary, secondary and tertiary colonizing bacteria from the surface of the teeth. It has been seen that the primary colonizing bacteria adhere to the salivary pellicle receptors from the surface of the tooth with the help of adhesins. Secondary colonizers bind initially attached bacteria. Multiplication is achieved with the help of the enzymatic complex located at the surface of the bacterial cell with agresinic role known as agresins [1, 3, 11]. The recognition of salivary pellicle receptors is made by the early colonizing bacteria and there is a coaggregations between early colonizers, fusobacteria, and late colonizers of the tooth surface.

**Quorum sensing**

Quorum sensing (QS) is the communication between cells by means of chemical mediators. It is a system of stimulus and response to population density and plays an important role in the control of bacterial colonization and virulence expression in the biofilm [13]. Bacteria are using QS signals to determine gene expression within their own kind. These sensing signals are used to inhibit or to activate transcriptional programs among competing bacterial strains and other species that exist within the same microenvironment [14]. Until recently it was considered that this type of interaction occurs only on a few bacterial species (*Vibrio fisheri*, *Vibrio harveti*, *Enterococcus faecalis*, *Myxococcus xanthus*, *Streptomyces spp.* [13]).

As a result of research that has been conducted on the ability of the bacteria to communicate within the microbial biofilm by means of chemical signals, it was found that there are some molecules acting as QS called acylated homoserine lactones (AHLs) molecules for signalling, seen in Gram negative bacteria. These molecules are secreted by the bacterial cells and have the ability to cross the cell membrane and to interact with receptors on the surface of the bacterial cell and to emit signals involved in the expression of certain genes [13, 15]. It is now accepted that planktonic cells have the ability to secrete (AHLs) molecules. The low concentration of these molecules does not cause large genetic modifications. Within the microbial biofilm, the bacterial cells are attached and grouped in the form of microbial communities, and in this context increases the concentration of (AHLs) molecules secreted within the community. Thus, it is encouraged the establishment of the complex structure of the multicellular communities because (AHLs) molecules have the ability to cross cell membranes and cause changes in genetic activity [16].

The properties of these signals and the response that they induce are important in the bacterial survival and propagation in different environments (e.g., oral cavity) where many bacterial species coexist.

**Conclusions**

The positive bacterial interactions (mutualism, commensalism, synergism) and negative interactions (competition and antagonism) have an important role in the expression of bacterial virulence and the appearance of mixed infections with periodontopathogen species. The presence of an universal communication signal system synthesized by different bacterial species (interspecies communication), has an important role in the formation of multispecies biofilm communities that are responsible in oral infection and disease. However, there are situations in which the bacterial interactions have beneficial effects on preventing the expression of virulence and colonization of pathogenic bacteria in the supragingival and subgingival area.

**References**


