Role of Human Lactoferrin in Oral Diseases

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Oral bacteria have evolved over millions of years to colonize, evade the host, and establish their own ecological niche. The oral cavity is composed of over 600 different bacterial species exhibiting an incredible diversity of microorganisms in a given space [1]. The bacteria colonizing the oral cavity have the potential to invade connective tissues enter the vasculature, and cause bacteremia as determined by innumerable studies [2]. In many ways, the oral cavity can act as a focus of infection for the initiation/exaggeration of systemic diseases. Oral bacteria have been shown to be significantly associated with increased risk of systemic diseases including infective endocarditis, myocardial infarction, and preterm low birth weight infants [2].

Human saliva is composed of 98% water and possesses an enormous array of antimicrobial factors and plays a vital role in preventing the initiation and progression of diseases [3,4]. It is therefore possible that saliva possesses a wealth of unexplored information, which may be clinically relevant and extremely significant. Diseases of the salivary gland resulting in decreased salivary flow cause serious disturbances in the oral cavity including increased predisposition to caries, periodontal disease, halitosis, impaired digestion and malnutrition. Research in the past decades has focused on identifying factors in saliva that help in antimicrobial function. It is possible that imbalances in the composition of these antibacterial factors in saliva play a role in determining the susceptibility of individuals to disease.

Lactoferrin (LF), also known as lactotransferrin, a part of the transferrin family is one of the factors present in the saliva that play an important role in oral diseases [5]. LF is an 80-kDa iron-binding glycoprotein that has been found to possess antibacterial, antimycotic, antifungal, antiviral, antitumor, antineoplastic, anti-inflammatory, as well as immunoregulatory activities. The protein is present in exocrine secretions that are commonly exposed to normal flora including: saliva, bronchial mucus, milk, tears, nasal exudates, gastrointestinal fluids, cervicovaginal mucus and seminal fluid [6,7]. LF also exhibits bacteriostatic and bactericidal activity through direct interactions governed by its strongly basic N-terminal region against a wide range of Gram-negative and Gram-positive bacteria [8-10]. Salivary LF plays an important role in protecting the oral cavity against several oral pathogens including *Aggregatibacter actinomycetemcomitans* and *Streptococcus mutans* [5,11]. Our group was the first to identify and characterize a single nucleotide polymorphism (SNP) that occurs at position 29 (Arginine/Lysine) in the N-terminal region of LF, which is associated with localized aggressive periodontitis [11]. Following this discovery, greater attention was focused on this SNP, and several studies showed that this polymorphism is associated with other diseases including periodontal disease [12], dental caries [5,13], coronary artery stenosis [14], circulating LF concentration [15], fasting triglyceride concentration and chronic rhinosinusitis [16].

By investigating the effect of LF in *Aggregatibacter actinomycetemcomitans* induced periodontal disease in lactoferrin knockout mouse (LFKO/−/−) model, we showed that higher alveolar bone loss occurred with increased expression of pro-inflammatory cytokines and chemokine expression compared to wild-type mice. In another study iv. administration of hLF rapidly cleared *A. actinomycetemcomitans* in the blood stream and other organs in LFKO/−/− mice compared to control group [17,18]. These results confirmed that LF plays an important role in periodontal infections and the lack of it therefore, makes mice more susceptible to the disease. LF, apart from its effects in the oral cavity, also offers protection against several systemic infections either due to its direct antimicrobial effect or to its iron sequestration abilities. Future studies need to be carried out to elucidate the mechanism of the antibacterial action of LF and to explore the therapeutic potential of LF in oral diseases.

References


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