healthy gingival crevices (Table 2). In addition, amounts in inflamed anchor plate crevices were higher than in healthy anchor plate crevices, although the differences were not significant (Table 2). Anaerobic bacteria were predominant in anchor plate crevices, particularly in inflamed crevices, when compared with healthy gingival crevices. Crevices around dental implants with clinically healthy status have been reported to be similar to gingival crevices in the terms of bacterial density, proportion of anaerobes in bacterial flora, amount of fluid and profile of crevicular fluid constituents (Mombelli et al. 1987; Apse et al. 1989; Adonogianaki et al. 1995). In addition, upon challenge with bacteria on tooth and implant surfaces, inflammatory and immune responses of peri-implant mucosa have been reported to be similar to those of gingiva (Seymour et al. 1989; Tonetti et al. 1995; Liljenberg et al. 1997; Karoussis et al. 2004). In our study, however, bacterial density was lower in healthy plate crevices and the proportion of anaerobes among bacterial flora was higher than in healthy gingival crevices (Table 2) and crevices around dental implants (Mombelli et al. 1987). These results suggest that crevices around anchor plates differed from healthy gingival crevices and crevices around dental implant in the terms of amount and constituents of crevicular fluid, as well as inflammatory and immune responses.

The anatomical structure of crevices around anchor plates is different from that of gingival tissue and peri-implant tissue, and the anchor plates receive a continuous orthodontic force. Thus, anchor plate crevices may have sparse tissue structure and high secretion of tissue exudates. This situation may increase immune responses around the anchor plate and efflux of crevicular fluid, thus resulting in decreased bacterial density.

Our results showed that the loading periods were longer in the inflammatory subjects than in the healthy subject (Table 1), however, further studies on the relationship between loading periods and the microflora/inflammation are required.

In healthy gingival crevices, saccharolytic and facultative anaerobic bacteria, such as *Actinomyces* and *Streptococcus*, were predominant (Table 2), thus suggesting that the environment of healthy gingival crevices is fluctuant to pH and oxygen concentration. On the other hand, in healthy anchor plate crevices, anaerobic bacteria such as *Campylobacter*, *Fusobacterium* and *Selenomonas* were dominant in addition to *Actinomyces* and *Streptococcus* (Table 2). In inflamed anchor plate crevices, *Prevotella* and *Fusobacterium* were predominant (Table 2). These results suggest that anchor plate crevices are more anaerobic than healthy gingival crevices. Titanium anchor plates have deep crevices (4–7 mm) with clinically healthy status (Table 1) and crevices seemed to be largely shielded from atmospheric oxygen. Thus, anchor plate crevices may form an anaerobic environment and subsequently increase the proportion of anaerobic bacteria.

The presence of saccharolytic bacteria, i.e., *Actinomyces* and *Streptococcus* species, in healthy anchor plate crevices implies that the environment turns acidic upon sugar fermentation. However, *Fusobacterium* species, *Prevotella nigrescens* and *Prevotella intermedia*, detected in anchor plate crevices are known to be able to grow under acidic conditions and neutralize acidic environments by metabolizing amino acids and proteins (Takahashi & Schachtele 1990; Takahashi et al. 1997; Takahashi 2003). It has also been reported that the pathogenicity of *P. nigrescens* and *P. intermedia* increased in the absence of glucose via elevated proteolytic activity and production of cytotoxic end products (Saito et al. 2001). These results suggest that *P. nigrescens* and *P. intermedia* harbored in anchor plate crevices may increase their pathogenicity, particularly when the crevices are deep and the main nutrition source is nitrogenous compounds, such as proteins and amino acids.

In summary, this study suggests that the environment in crevices around titanium orthodontic anchor plates is anaerobic and supports anaerobic growth of bacteria, which may trigger inflammation in the tissue around the plates. Therefore, orthodontic treatment with titanium anchor plates requires strict self-care and regular professional plaque control in order to prevent infection.

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要旨

本研究では、細菌同定のための嫌気培養と分子生物学的手法を用いて、チタン製矯正用アンカープレート周囲溝内の細菌叢の構成を明らかにし、アンカープレート周囲溝と歯肉溝の細菌構成の比較を行った。被験者（20－29歳）のチタン・アンカープレート周囲溝（10名）と臨床的に健康な歯肉溝（7名）から採取した試料を嫌気条件で培養し、分離した細菌を16S rRNAシークエンス法により同定した。健康なアンカープレート周囲溝、炎症を伴うアンカープレート周囲溝および健康な歯肉溝内の、平均CFUs（log値）/mLは、それぞれ6.84、7.51、8.88であり、アンカープレート周囲溝の細菌密度は健康な歯肉溝より低いことが示された。7名の健康なアンカープレート周囲溝から分離された184菌株のうち、108（59%）は嫌気性菌であり、73（40%）は通性菌であった。このうち、*Campylobacter*（12%）、*Fusobacterium*（10%）、*Selenomonas*（10%）などのグラム陰性桿菌、*Actinomyces*（17%）、*Streptococcus*（8.2%）などのグラム陽性通性菌が優勢であった。3名の炎症を伴うアンカープレート周囲溝から分離した133菌株のうち、110（83%）は嫌気性菌であり、*Prevotella*（47%）、*Fusobacterium*（33%）、*Campylobacter*（16%）などのグラム陰性桿菌が優勢であった。一方、7名の健康な歯肉溝から分離した146菌株のうち、98（67%）は通性菌であり、45（31%）は嫌気性菌であった。これらの結果から、チタン製矯正用アンカープレート周囲溝内の環境は嫌気的であり、嫌気性菌の増殖が促進されることによって、プレート周囲の歯周組織の炎症が惹起される可能性が示唆された。

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