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THE POTENTIAL CAPABILITY OF MELATONIN TO ANTICIPATE POST-ORTHODONTIC TREATMENT RELAPSE: A LITERATURE REVIEW

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ABSTRACT : The long-term stability outcome is the main goal of orthodontic treatment. Retainers are the most commonly used appliances to prevent orthodontic relapse. However, they are not always sufficient to manage post-orthodontic treatment relapse. An effective approach is required to anticipate the occurrence of orthodontic relapse. This review was aimed to discuss the potential use of melatonin to modulate post-orthodontic treatment relapse. Melatonin may become a promising agent to regulate orthodontic relapse, through modulating alveolar bone remodeling by stimulating osteoblast and inhibiting osteoclast. Administration of melatonin in rodents model promotes bone mass, bone formation, impairs bone healing and inhibits bone loss. In addition, nightly melatonin supplementation in perimenopausal women is well tolerated and may improve the imbalance in bone remodeling. Melatonin also accelerates osteogenic differentiation in various cell cultures through melatonin 2 receptor (MT2R) by activating multiple signal cascade including MEK1/2 and 5, Wnt 5 α/β, BMP-2 and -4, PDGF/AKT signaling pathway. Furthermore, melatonin has also been reported to suppress osteoclastogenesis directly through melatonin 1 receptor (MT1R)/MT2R by inhibiting NF-κB signaling pathway and indirectly by decreasing RANKL/OPG ratio from osteoblast. Thus, melatonin may provide a new direction in controlling post-orthodontic relapse, by stimulating bone formation and inhibiting bone resorption.

Key words : Melatonin, orthodontic relapse, osteoblast, osteoclast, medicine.

INTRODUCTION

The primary goal of orthodontic treatment is to achieve a long-term stability outcome (Nugraha *et al*, 2019). In orthodontic tooth movement, the usage of both fixed and removable retainer is essential to inhibit the tooth return to their original positions after treatment, enabling periodontal tissues remodeling and maintain the space balance. On the other hand, this condition is difficult to obtain, and insufficient periodontal tissue remodeling often leads to orthodontic relapse (Han *et al*, 2010; Vieira *et al*, 2015; Liu *et al*, 2017). Generally, the prevalence of relapseis relatively high, ranges between 10% to 50%, depending on their follow-up time. In prolonged follow-up time, its prevalence became 30% of cases (Sadowsky and Sakols, 1982). Meanwhile, other studies stated that relapse occurred between 30%-50% cases after 10 years and declined to 10% cases after 20 years (De Bernabé *et al*, 2017; Yu *et al*, 2013).

Relapse can be caused by multifactorial factors, such as muscle disoders,changes in dental arch form, supracrestal fibers reorganization and reorientation insufficiency, lasting unwanted habits, improper skeletal growth, immature and slightly mineralized bone tissue surrounding the moved tooth (Vieira *et al*, 2015). The most general approach to obtain the long-term stability outcome is the use of a retainer. In the first 6 months, retainer must be used full-time and nightwear is necessary for at least 12 months. Long duration of active orthodontic treatment and the addition of a 2-year retention time frequently cause disappointment in patient. Thus, leading to rejection or poor patient compliance to wear retainer and cause the occurrence of relapse. Therefore, an effective method is required to modulate treatment stability and to shorten retention time (Zhang *et al*, 2014).

Enhancement of osteoblast activity and suppression of osteoclast activity in alveolar bone during the retention phase might effectively inhibit relapse and improve stability post orthodontic tooth movement, as the relapse pressure remains until alveolar bone remodeling is complete (Sitasari *et al*, 2020; Inayati *et al*, 2020; Hermawan *et al*,