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Review

Influence of nicotine on orthodontic tooth movement: A systematic review of experimental studies in rats

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ABSTRACT

Objective: The objective of this systematic review was to assess the impact of nicotine administration on orthodontic tooth movement (OTM).
Methods: A systematic search was conducted in PubMed, Scopus, EMBASE, MEDLINE (OVID) and Web of Knowledge databases and the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines were followed. Studies evaluating the influence of nicotine on OTM, and with the presence of a control group (OTM without nicotine administration), were included. Quality assessment of the selected studies was performed following the Animal Research Reporting in Vivo Experiment (ARRIVE) guidelines.
Results: Six of the initially identified 108 articles fulfilled the inclusion criteria and were selected. All included studies were performed in male rats, which underwent OTM with or without nicotine administration. Since there was a variation among the included studies regarding nicotine dosage and the duration and magnitude of force application during OTM only a qualitative analysis could be performed. The studies reported that nicotine administration accelerated OTM by inducing alveolar bone resorption around the moving teeth. It was also found that nicotine increased root resorption during experimental OTM. More standardized animal research or clinical studies are warranted to further evaluate the impact of nicotine on OTM.
Conclusions: On an experimental level, nicotine exposure in rats jeopardizes OTM by increasing alveolar bone loss and root resorption. From a clinical perspective, further studies are needed to assess the impact of habitual use of tobacco products on OTM.

1. Introduction

Tobacco smoke consists of more than 4000 potentially toxic compounds, of which nicotine is supposed to be the most detrimental (Hoffmann & Hoffmann, 1997). At a cellular level, nicotine impairs angiogenesis and the proliferation of erythrocytes; fibroblasts proliferation and adhesion, collagen synthesis, and osteogenesis are also affected (Davies & Ismail, 2016; Ghanem et al., 2017; Pinto, Bosco, Okamoto, Guerra, & Piza, 2002; Sherwin & Gastwirth, 1990). Nicotine increases platelet adhesiveness and decreases the number of macrophages (Sherwin & Gastwirth, 1990). Moreover, nicotine induces osteoblastic apoptosis and increases osteoclastic activity (Costa-Rodrigues, Rocha, & Fernandes, 2018; Marinucci et al., 2018). Furthermore, nicotine induces epinephrine and norepinephrine release from postganglionic sympathetic nerves, which limits tissue perfusion and induces vasoconstriction (Jones & Triplett, 1992). Macroscopically, this affects healing and tissue perfusion due to micro clot formation in the blood vessels (Ghanem et al., 2017; Mosely & Finseth, 1977; Sherwin & Gastwirth, 1990). Considering these effects, it is likely that nicotine impairs biological processes requiring higher metabolic activity (Pinto et al., 2002). In this context, studies have shown that nicotine exposure is associated with periodontal inflammation (Ge et al., 2016; Kubota et al., 2016; Wu et al., 2013) and impaired implant osseointegration (Cesar-Neto et al., 2003; Ghanem et al., 2017; Yamano et al., 2010).

Orthodontic tooth movement (OTM) involves an active process of

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bone remodeling which includes bone resorption and bone formation. On the pressure side, alveolar bone is resorbed by osteoclasts in Howship's lacunae; whereas, in the tension side bone is deposited (Meikle, 2006). The underlying mechanism during OTM includes an early phase of acute inflammatory response, vasodilatation, leucocytes migration and release of arachidonic acid, growth factors, metabolites, cytokines and various enzymes (Isola, Matarese, Cordasco, Perillo, & Ramaglia, 2016; Tsuge, Noda, & Nakamura, 2016). Studies (Bakathir, Linjawi, Omar, Aboqura, & Hassan, 2016; Kirschneck, Proff, Maurer, Reicheneder, & Romer, 2015; Kirschneck, Maurer, Wolf, Reicheneder, & Proff, 2017; Li et al., 2016; Sodagar, Donyavi, Arab, & Kharrazifard, 2011) have suggested that nicotine might impair the mechanobiology of OTM by accelerating bone resorption. For example, Bakathir et al. (2016) observed accelerated OTM with unbalanced bone resorption and apposition patterns around the moving teeth in rats receiving nicotine for 28 days compared with controls. Similarly, Sodagar et al. (2011) showed that nicotine accelerates OTM, and this effect is dose-dependent. However, controversial results have also been reported in animal models. For instance, Nagaie, Nishiura, Honda, Fujiwara, and Matsumoto (2014) observed that a comprehensive mixture of tobacco smoke components (TSCs) decreased OTM by osteoclastogenesis inhibition and delayed bone resorption in a rat model. Likewise, Shintcovsk, Knop, Tanaka, and Maruo (2014) reported that nicotine decreased the number of osteoclastic cells during OTM in a rat model after 21 days of nicotine administration.

There seems to be a debate over the pathophysiologic influence of nicotine on OTM; therefore, the aim of the present systematic review was to assess the influence of nicotine administration on OTM.

2. Materials & methods

2.1. Focused question

This systematic review was conducted by following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (Moher, Liberati, Tetzlaff, & Altman, 2009). The addressed focused question was "Does nicotine affect OTM?"

2.2. Eligibility criteria

The eligibility criteria were as follows: (a) original clinical and animal/experimental studies; (b) presence of a control group (orthodontic tooth movement without nicotine delivery); (c) intervention: effect of nicotine delivery on OTM. Letters to the Editor, historic reviews, commentaries, case-series and case-reports were excluded. Articles available online in electronic form ahead of print were considered eligible for inclusion.

2.3. Information sources, literature search strategy, and study selection

An electronic search without time or language restrictions was conducted up to and including March 2018 in PubMed (National Library of Medicine), Scopus, EMBASE, MEDLINE (OVID) and Web of Knowledge databases, in order to identify studies relevant to the focused question. Search term included Medical subject headings (MeSH) and text words (other relevant non-MeSH terms) to identify articles discussing influence of nicotine in OTM. These included the following MeSH terms: (1) nicotine; (2) tooth movement techniques; (3) orthodontic brackets and (4) orthodontic appliances; and text words: (5) orthodontic; (6) orthodontically; and (7) orthodontic forces. These keywords were used with Boolean operators (OR, AND) to combine the key words mentioned above.

Titles and abstracts of studies identified using the above-described protocol were screened by two authors (DM and FJ) and checked for agreement. Full-texts of studies judged by their titles and abstracts to be relevant were read and independently evaluated for the stated eligibility criteria. Reference lists of potentially relevant original and review articles were hand-searched to identify studies that have remained unidentified in the previous step. Once again, the articles were checked for disagreement via discussion among the authors. Kappa scores (Cohen kappa coefficient) were used to determine the level of agreement between the 2 reviewers (Kappa score = 0.94) (Roberts, 2008).

2.4. Quality assessment of included studies

The risk of bias of included studies was assessed by two authors (DM and ZA) using the Systematic Review Centre for Laboratory animal Experimentation (SYRCLE) risk of bias tool (Hooijmans et al., 2014). This tool is based on the Cochrane Collaboration that aims to assess methodological quality and adapted to appraise aspects of bias that play a role in animal experiments (Higgins et al., 2011). Briefly, subsequent sections are considered: selection bias (randomization and allocation concealment), performance bias (blinding of study personnel/caregivers), detection bias (blinding of outcome assessors), completeness of follow-up period (attrition bias) and other biases. Studies were classified as having "high risk of bias" (high), "low risk of bias" (low) or "unclear" (?) for each of these sections. Overall, studies were considered as: (i) low risk of bias if all criteria were met (adequate randomization and allocation concealment; "yes" answer to all questions about the completeness of outcome data and blinding, and "no" answer to selective reporting and other sources of bias); (ii) unclear risk of bias if one or more criteria were partly met; or (iii) high risk of bias if one or more criteria were not met.

In an attempt to increase the strength of the present systematic review, the selected studies underwent a quality assessment following the Animal Research Reporting in Vivo Experiment (ARRIVE) guidelines (Kilkenny & Altman, 2010; C. Kilkenny, W. Browne, Cuthill, Emerson, & Altman, 2010; C. Kilkenny, W.J. Browne, Cuthill, Emerson, & Altman, 2010) and to a pre-defined grading (Delgado-Ruiz, Calvo-Guirado, & Romanos, 2015; Kellesarian, Subhi, Saleh Binshabaib, & Javed, 2017; Schwarz, Iglhaut, & Becker, 2012) applied to the following 20 specific criteria: (1) Title (concise and accurate); (2) Abstract (summary of background, objectives, methods, main findings and conclusions); (3) Introduction (background objectives, relevance to human biology); (4) Introduction (primary and secondary objectives); (5) Methods (Ethical statement, national and institutional guidelines for the care and use of animals); (6) Methods (study design, steps taken to minimize bias such as allocation concealment, blinding and randomization); (7) Methods (experimental procedure with precise details); (8) Methods (experimental animals details including species, gender, age, weight and source); (9) Methods (housing and husbandry conditions such as, type of cage, light/dark cycle, temperature, access to food and water); (10) Methods (sample size); (11) Methods (allocation of animals to experimental groups, randomization); (12) Methods (experiment outcomes); (13) Methods (statistical analysis); (14) Results (baseline data, health status of animals); (15) Results (number of animals analyzed, reasons for exclusion); (16) Results (outcomes and estimation, results for each analysis); (17) Results (adverse events); (18) Discussion (interpretation, scientific implications, study limitations including animal model); (19) Discussion (generalizability and translation, relevance to human biology); and (20) Discussion (funding sources, role of the funders, conflicts of interest).

Each criterion was graded as "0" (not reported) or "1" (reported). The combined frequency of reporting for each criterion in all the included studies was also recorded.

3. Results

3.1. Study selection

One hundred and eight potential articles were initially identified. In