CLINICAL CROWN LENGTH CHANGES AND GINGIVAL RECESSION DEVELOPMENT IN ANTERIOR TEETH ASSOCIATED WITH ORTHODONTIC TREATMENT: A SYSTEMATIC REVIEW AND META-ANALYSIS

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ABSTRACT

AIM: To systematically investigate and appraise the quality of the available evidence regarding clinical crown length changes and gingival recession development following orthodontic treatment.

MATERIALS AND METHOD: A search without restrictions for published and unpublished literature and hand searching was performed. Data on incisor clinical crown length changes and gingival recession development following orthodontic treatment were reviewed. Measurements before and after orthodontic treatment, as well as case-control studies, were considered. Relevant information was extracted, methodological quality was evaluated using the ROBINS-I tool (Risk Of Bias In Non-randomized Studies of Interventions) and the random effects model was used to combine the retrieved data.

RESULTS: Ten studies following patients for up to 6 years after orthodontic treatment were finally identified. Clinical crown length increased after orthodontic treatment and during retention. However, in general, no differences were noted

between the groups that underwent proclination compared to the non-proclination group. Moreover, orthodontic treatment seemed to increase the odds for a patient to exhibit gingival recession in at least one tooth, compared to before treatment. In addition, orthodontic treatment seemed to increase the odds for a patient to exhibit gingival recession in at least one mandibular incisor, compared to before treatment or the untreated controls.

CONCLUSIONS: The present systematic review and meta-analysis showed that some increases in gingival recession prevalence may be encountered after orthodontic treatment. Although clinical crown length increases during orthodontic treatment and retention, overall no statistically significant differences were noted between incisors having undergone proclination compared to the non-proclination group. More high quality studies are needed in order to further elucidate possible associations.

DEDICATION

I would like to dedicate this thesis to my family who has supported me throughout my academic journey.

DECLARATION

I declare that all the content of the thesis is my own work. There is no conflict of interest with any other entity or organization.

Name: Budoor Salem Khalfan Juma Khalfan Bin Bahar

Signature:

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1. INTRODUCTION

Orthodontic treatment aims to create a long-term functionally satisfactory, healthy, esthetically pleasing, and stable occlusion that is in harmony with patient's facial appearance. According to Proffit et al. (2007) patients usually seek orthodontic treatment in order to:

1. Remove, or at least alleviate, the social handicap created by an unacceptable dental and/or facial appearance.
2. Enhance dental and facial appearance in cases where individuals are already socially acceptable but wish to improve their quality of life.
3. Maintain as normal development a developmental process as possible.
4. Improve jaw function and correct problems related to functional impairment.
5. Reduce the impact on the dentition of trauma or disease.
6. Facilitate other dental treatment, as an adjunct to restorative, prosthodontic or periodontal therapy.

Thus, the benefits of orthodontic treatment in enhancing esthetics, function, and self-esteem are numerous and, in most cases, outweigh the possible disadvantages. However, orthodontic intervention can involve some risks in terms of hard and soft tissue health, such as enamel damage associated with bonding and debonding procedures, enamel decalcification, root resorption, bone dehiscences and fenestrations with/without soft tissue periodontal damage, temporomandibular disorders, tooth devitalization, and allergic reactions (van Beek, 2009; Justus, 2015).

One of the undesirable side effects of orthodontic treatment possibly jeopardizing orthodontic intervention success involves the apical relocation of the gingivae leading to an increase in clinical crown length and possible recession. Gingival recession is

defined as “the migration of the marginal soft tissue to a point apical to the cemento-enamel junction of a tooth or the platform of a dental implant” (American Academy of Periodontology, 2017). Orthodontic tooth movement has been implicated as a possible etiological factor for this sequela (Joss-Vassalli et al., 2010). As teeth are moved to new positions, the spatial relationships of the soft and underlying osseous tissues are altered to accommodate changes in esthetics and function. Thus, it might be possible that, under certain circumstances, teeth acquire unfavorable positions relative to their supporting structures; potentially leading to apical soft tissue relocation, increases in clinical crown length and, eventually, the appearance of recession (Wennstrom et al., 1987).

Whether orthodontic tooth movement might cause recession, or if the teeth actually adapt unproblematically to their new positions remains a controversial issue. Contradictory opinions can be found in the current literature. The aim of the present study was to systematically investigate and appraise the quality of the available evidence regarding clinical crown length changes and gingival recession development following orthodontic treatment.

2. REVIEW OF LITERATURE

In addition to its benefits, orthodontic treatment presents risks and possible complications, similarly to any other medical intervention. Acknowledging these risks and complications constitutes an important step in delivering safe medical care (Preoteasa et al., 2012). Although in orthodontics, the risk of "doing harm" is considerably low compared to other medical interventions, the possibility for undesired side effects to arise during treatment due to various procedures, use of certain devices and materials still exists (Preoteasa et al., 2012).

2.1. Classification of risks and complications of orthodontic treatment

The risks and complications of orthodontic treatment can be classified, according to their localization, as systemic, local extra-oral, and local intra-oral (Travess et al., 2004).

2.1.1. Systemic risks and complications

Three possible systemic risks have been reviewed in the literature (Travess et al., 2004).

a) Spread of infection

Patient safety is an important part of medical discipline, which aims at improving the quality of patient care, minimizing treatment errors, and generally improving safety (Yamalik and Van Djik, 2013). To ensure patient safety, adequate infection control procedures should be implemented throughout the delivery of various procedures to

prevent the spread of infection between patients, between operator and patient, and involving third parties (Travess et al., 2004). The use of gloves, masks, sterilized instruments, and 'clean' working areas are of paramount importance. If resources allow, the choice of a higher level of infection control regimen is desirable, in addition, a detailed medical history must be taken from every patient to determine possible risk factors (Travess et al., 2004).

b) Infective endocarditis

Patients at risk of endocarditis should be treated in accordance to the appropriate guidelines and in consultation with a cardiologist (Khurana and Martin, 1999; Hobson and Clark, 1995). Meticulous oral hygiene is essential, and must be coupled with antibiotic cover for invasive procedures such as extractions, surgical canine exposures, placement of tooth separation elastics, band placement, and band removal. In order to minimize bacterial loading, chlorhexidine mouthwash has been advocated prior to any treatment and, in some cases, even daily use is mandatory/advised (Sonis, 2004).

c) Allergy

Orthodontists are sometimes required to treat patients with nickel allergy. This constitutes a concern since most of the orthodontic materials contain nickel. During the initial phase of orthodontic treatment, super-elastic-nickel-titanium (Ni-Ti) archwires are used for the leveling and aligning of the dental arches. These Ni-Ti archwires contain about 47-50% nickel (Eliades and Athanasiou, 2002). Additionally, stainless steel archwires also contain about 8% nickel, a metal also present in bands, and metallic brackets. However, nickel is bonded into a crystal lattice in the stainless

steel alloys used in the manufacturing of orthodontic components. Therefore, it is not free to react and thus highly unlikely to cause hypersensitivity reactions in the majority of orthodontic cases (Rahilly and Price, 2003). It is estimated that 11% of all women, and 20% of women between the ages of 16 and 35 years have nickel hypersensitivity (Nielson and Menne, 1992; 1993; Menne 1996). On the other hand, only 2% of males are sensitive to nickel and this is possibly due to less contact with nickel containing jewellery. Nickel often elicits contact dermatitis; a Type IV delayed hypersensitivity immune response (Van Loon et al., 1988). Barret and co-workers (1993) showed that nickel leaching from orthodontic bands, brackets, and stainless steel or Ni-Ti archwires in vitro occurs maximally within the first week and subsequently declines. Gjerdet et al. (1991) found a significant release of nickel and iron into the saliva of patients just after the placement of fixed appliances, which could be responsible for Type IV hypersensitivity reactions. However, there was no significant difference in nickel or iron concentrations between controls and subjects to whom the appliances had been in place for a number of weeks. The clinical significance of nickel release is, as yet, unclear, but constitutes a factor requiring consideration in sensitive patients. Factors including intra-oral temperature, pH, salivary composition, duration of exposure, wear of the wire due to friction from sliding mechanics, abrasion, presence of solder, strain in the wire and, most importantly, the degree of leaching are factors determining the concentration of nickel present due to a particular appliance (Jia et al., 1999). Fortunately, most individuals with nickel sensitivity do not report adverse clinical reactions to orthodontic appliances containing nickel (Noble et al., 2008). It is estimated that only 0.1-0.2% of patients show a negative reaction to nickel containing appliances (Menne, 1994). Research suggests that a much greater concentration of nickel is necessary to elicit an

allergic reaction in the oral mucosa than the skin (Dunlap et al., 1989). Alternative archwires such as twist-flex stainless steel, fibre-reinforced composite, plastic/resin coated Ni-Ti, pure titanium or even gold plated wires can be used without risk (Agarwal et al., 2011).

In addition to intra-oral reaction, Lau and Wong (2006) stated that allergy to nickel is more common in extra-oral situations, usually as a result of contacts with a face-bow or headgear strap. The use of sticking plaster to cover the areas in contact with the skin is sufficient to relieve symptoms (Lau and Wong, 2006).

Other allergens associated with orthodontic treatment procedures include components or chemical catalysts in bonding materials, cold curing acrylics, or latex components (Jacobsen and Hensten-Pettersen, 1989). A few patients have exhibited severe latex allergies caused by elastics or operators' gloves. Allergy to latex and bonding materials has been reported, however these cases are rare (Nattrass et al., 1999).

2.1.2. Localized extra-oral risks and complications

The localized extra oral risks associated with orthodontic treatment include the following (Travess et al., 2004).

a) Trauma

Extra-oral appliances can cause both extra- and intra-oral adverse reactions (Postlethwaite 1989; Samuels and Jones 1994). Following a well-documented case reported by Booth-Mason and Birnie (1988) involving an eye trauma in a patient wearing headgear, explicit guidelines became available, and a number of safety headgear products have been introduced. These measures include safety bows, rigid neck straps and snap release products to prevent the bow from disengaging from the

molar tubes or acting as a projectile.

A survey among British orthodontists found a 4% incidence of facial injury with headgear (Booth-Masonand Birnie, 1988). Of these injuries, 40% were extra-oral and 50% of these occurred to the mid face. Two patients became blind as a result of headgear trauma. Eye injury, although uncommon, constitutes a serious risk and all available methods of reducing the risk of penetrating eye injury must be employed. Every headgear must incorporate a relevant safety feature. Failure to observe safety guidelines on the use of headgear is medico-legally indefensible (Booth-Masonand Birnie, 1988).

b) Burns

According to Travess et al.(2004), burns, either thermal or chemical, are possible both intra- and extra-orally with inadvertent use of chemicals or instruments. Acid etch, electrothermal debonding instruments and sterilized instruments which have not been allowed to cool down all have the potential to cause burn injuries , and require care in use.

c) Tempromandibular dysfunction

Considerable attention has been drawn to the relationship between temporomandibular dysfunction (TMD) and orthodontic treatment. Irrespective of orthodontic treatment, TMD is common in the general population. In addition, there is no evidence to support any argument that relates orthodontic treatment to either the cause or cure of TMD (Luther 1998). Furthermore, orthodontic patients are not at greater risk of developing TMD than the general population, leading to the conclusion that there is no direct relationship between the two. However, pre-existing TMD

should be recorded, and the patient warned that treatment would not necessarily improve their condition. Prior to commencing treatment, patients should also be informed that there is the possibility of suffering increased symptoms during orthodontic treatment. The standard approach to assessing TMD should be taken whenever patients experience symptoms during treatment. Conservative treatment should be directed at alleviating discomfort, occlusal disharmony and joint noises and reassuring the patient. Other forms of standard treatment (e.g. soft diet, jaw exercises) may also be indicated (Travess et al., 2004).

2.1.3. Localized intra-oral risks and complications

The localized intra-oral risks associated with orthodontic treatment may involve the dental or the tooth supporting tissues (Travess et al., 2004).

a) Side effects related to dental tissues

Enamel damage

Bonding of orthodontic appliances may induce irreversible changes in the tooth surface. The enamel changes depend on numerous factors such as the type and concentration of the acid etch material, its time of application, and enamel surface characteristics (Tiro et al., 2017). The most severe modifications appear when conventional resins are used as bonding materials. The self-etching adhesive resin bonding technique is safer and better than conventional resins, which require separate phases of etching. This technique produces less enamel damage, albeit with the disadvantage of lower bond strength. Moreover, resin-modified glass ionomer cement is the material of choice for cementing orthodontic bands as it reduces enamel damage

due to its fluoride releasing properties (Tiro et al., 2017) and achieves bond strength similar to resins (Fjeld and Ogaard, 2006; Pessan et al., 2008; Lill et al., 2008).

The debonding phase of orthodontic treatment may cause tooth damage in the form of enamel cracks or even loss. The extent of enamel loss depends on the type of brackets used, the bonding materials and the debonding technique (Hosein et al., 2004). Enamel damage is usually less with metallic brackets or when glass ionomer cement is used as a bonding agent (Naini and Gill, 2008); while using ceramic brackets and conventional adhesive resins causes more severe enamel damage. The proper debonding technique is to break the bond between bracket and adhesive. The presence of horizontal enamel cracks after debonding is a direct result of the orthodontic debonding technique (Ogaard and Fjeld, 2010).

Enamel demineralization/caries

Unfortunately, a common irreversible complication at risk of occurring during fixed appliance therapy is enamel demineralization in the absence of adequate oral hygiene measures; this usually occurs on smooth surfaces (Travess et al., 2004; van Beek, 2009). The prevalence of this problem ranges from 2% up to 96% of orthodontic patients, depending on the sample and methodology used (Chang et al., 1997) with maxillary lateral incisors, maxillary canines and mandibular premolars being the most commonly affected teeth (Geiger et al., 1988). However, any tooth in the mouth can be affected, and often a number of anterior teeth show decalcification. There is a possibility of remineralization and reversal of the lesion whilst the demineralized surface remains intact. Frank cavitation can be seen in severe cases requiring restorative interventions (Travess et al., 2004). In patients with fixed orthodontic appliances, the plaque index is higher compared to patients without orthodontic

treatment (Klukowska et al., 2011). The evidence shows a decrease in salivary pH during orthodontic treatment together with an increased level of two main caries favoring factors such as Streptococcus mutans and Lactobacillus (Vizitiu and Ionescu, 2010). The evaluation of the patient's oral hygiene habits is mandatory before starting orthodontic therapy. In some situations, a high frequency of dental caries and poor oral hygiene may be reasons for postponing, or even terminating orthodontic treatment. It is recommended that primary prevention methods be employed to avoid this complication (i.e., educate patients about proper oral hygiene and diet), together with the use of fluoride-releasing materials. In some cases, a secondary prevention method, such as fluoridation, is recommended (Travess et al., 2004). Studies have shown that a daily fluoride mouth rinse or a fluoride-containing cement reduces tooth decay during treatment with fixed orthodontic appliances (Ionescu et al., 2008; Delbem et al., 2004). When white spot lesions are present at the end of treatment, spontaneous remineralization sometimes occurs, and the chewing of sugar-free gums is recommended to improve salivation (Burt, 2006). Other procedures implemented for the management of white spot lesions include laser, tooth whitening, resin infiltration and micro-abrasion (Morrier, 2014).

Enamel wear

In addition to any enamel fractures resulting from debonding, enamel damage can occur when ceramic brackets cause occlusal interferences with the opposing teeth (Viazis et al., 1990). This rapidly occurring type of damage can be more severe than the abrasion caused by metallic brackets (Gibbs 1992; Viazis et al., 1990). Single-crystalline ceramic brackets have demonstrated the highest abrasion scores. It has been suggested that when occlusal interferences occur between teeth and ceramic

brackets, a correlation exists between the design and form of the ceramic brackets and the degree of enamel damage (Viazis et al., 1990). Therefore, contact of the opposing teeth with the ceramic brackets must be avoided. To achieve this, special elastomeric rings covering the occlusal surface of the ceramic brackets, or techniques eliminating occlusal interferences and which control parafunctional habits should be used (Birnie, 1990; Douglass, 1989).

Root resorption

Although unpredictable in extent, some degree of external root resorption is inevitably associated with orthodontic treatment (Brezniak and Wasserstein, 1993). This comprises the most frequent iatrogenic consequence of orthodontic treatment (Rafiuddin et al., 2015). The etiology, severity, and degree of root resorptions are multifactorial, involving both host and environmental factors (Rafiuddin et al., 2015). Resorption might occur on the apical and lateral surface of the roots, but radiographs only reveal a degree of apical resorption (Travess et al., 2004). In many instances, no clinically significant resorption is evident or visualized by routine radiography, but microscopic surface changes are nevertheless likely to have occurred (Travess et al., 2004). It has been shown that root resorption is highly correlated with longer treatment duration, fixed appliance treatment, individual susceptibility, orthodontic forces and the type of orthodontic tooth movement (Roscoe et al., 2015). Segal et al. (2004), indicated that factors associated with the duration of active treatment might result in an increase in apical root resorption. They also showed in their meta-analysis that the mean apical root resorption is highly correlated with intrusive forces and total treatment duration. They suggested pauses in active treatment with the use of passive archwires for 2-3 months to minimize root resorption (Segal et al., 2004; Mahida et

al., 2015). According to Jones and Darendeliler (2006), heavy forces are more likely to produce root resorption than light forces. In addition, intrusive force causes about four times more root resorption than extrusive force (Han et al., 2005). From the whole dentition, Ketcham (1929) suggested that maxillary incisors are more frequently involved than any other tooth. Higher susceptibility of the mandibular incisors has been also reported (Goldsonand Henrikson, 1975) while the buccal roots of the first maxillary molars and premolars frequently exhibit root resorption (Reitan, 1972). The degree of resorption is usually less than 2 mm, but can be more extensive (Hollender, 1980). However, root shortening rarely compromises the longevity of affected teeth (Hendrix, 1994). Linge and Linge (1983) suggested that pre-treatment root form or tooth length (blunt and short roots), previous history of tooth trauma, and treatment mechanics are all risk factors for increased incidence and severity of root resorption.

b) Side effects related to tooth supporting tissues

The complications related to tooth supporting hard and soft tissues are the most evident side effects linked to orthodontics, and frequently constitute the reason for malpractice complaints (Preoteasa et al., 2012). These sequelae can be encountered in various forms, from gingivitis to periodontitis, dehiscences, fenestrations, interdental folds, apical soft tissue relocation and gingival recession or overgrowth and the development/establishment/presence of black triangles. In certain situations, even tooth prognosis might be compromised. Orthodontic tooth movement has been implicated as a possible etiological factor for apical soft tissue relocation and gingival recession (Joss-Vassalli et al., 2010). As teeth are moved to new positions, the spatial relationships of the soft and underlying osseous tissues are altered to accommodate

changes in esthetics and function. Thus, it is possible under certain circumstances teeth to become unfavorably positioned relative to their supporting structures, which consequently could lead to apical soft tissue relocation, increase in clinical crown length and eventually gingival recession formation (Wennstrom et al., 1987).

2.2. Gingival recession

The muco-gingival complex comprises the free and attached gingiva, the muco-gingival junction and the alveolar mucosa. An adequate muco-gingival complex, in which the muco-gingival tissues can sustain their bio-morphological integrity and maintain an enduring attachment to the teeth and the underlying soft tissue, is always essential for long-term periodontal health (Chrysanthakopoulos, 2011). When a muco-gingival problem occurs, there are basically two ways in which it presents itself: either, as a closed disruption of the muco-gingival complex resulting in pocket formation, or as an open disruption of the muco-gingival complex resulting in gingival clefts and gingival recession (Löe et al., 1992).

Gingival recession is defined by the American Academy of Periodontology as "the migration of the marginal soft tissue to a point apical to the cemento-enamel junction of a tooth or the platform of a dental implant" (American Academy of Periodontology, 2017). It may be localized or generalized and can be associated with one or more tooth surfaces (Kassab and Cohen, 2003). It is a common condition seen in both dentally aware populations and those with limited access to dental care. Gingival recession development can constitute a significant problem from the clinical and patient reported outcome perspective. For any individual, gingival recession usually creates an esthetic problem, especially when such a problem affects the

anterior teeth, and creates a fear of losing the affected tooth or teeth due to further destruction. In addition, it can also be associated with dentine hypersensitivity, and/or root caries, abrasion and/or cervical wear, erosion because of exposure of the root surface to the oral environment and an increase in the accumulation of dental plaque (Tugnait and Clerehugh, 2001).

Abrasion or erosion of the cementum exposed by recession leaves an underlying dentinal surface that can be sensitive. Al-Wahadni and Linden (2002) demonstrated that gingival recession of 3 mm or more was the best independent predictor of dentin hypersensitivity. In another study, Costa et al.(2014) showed that gingival recession was associated with increased dentin hypersensitivity in 1023 Brazilian adults aged 35 or older. Moreover, difficulty in the maintenance of oral hygiene (Newman et al., 2012) and compromised esthetics have been observed (Dorfman, 1987). For instance, Rocha et al. (2011) showed that when 160 dental students evaluated the esthetic perception of smiles, there was a statistically significant difference between smiles with and without gingival recession. This presents a problem as esthetics is a major motivational factor for patients who seek orthodontic treatment (Riedmann et al., 1999). It has been shown that for 75% of patients seeking orthodontic treatment, dental esthetics was their primary motive/objective (Reidman et al., 1999).

Two main types of marginal gingival recession have been identified; the generalized, which may involve interproximal areas and is mainly found in periodontally untreated populations with poor oral hygiene, while other type is usually related to traumatic factors and often involves only a few, or a group of teeth. The latter frequently occurs in buccal areas where the lesions commonly are associated with plaque-free, smooth, and well-polished hard tissue defects (Maynard, 2004; Miller, 1987).

Miller (1985) proposed a useful recession defect classification based on the height of

the interproximal papillae and interdental bone adjacent to the defect area, and the relation of the gingival margin to the muco-gingival junction (Table 1). This classification is useful when deciding on treatment options (Maynard, 2004).

Table 1. Classification of gingival recession defects by Miller (1985).

Class I	Recession within attached gingiva. No loss of interdental bone and soft tissue papillae covering interdental bone at full height
Class II	Recession extending to or beyond the mucogingival junction. No loss of interdental bone and soft tissue papillae covering interdental bone at full height
Class III	Recession extending to or beyond the mucogingival junction. Loss of interdental bone but interdental bone height coronal to apical extent of recession defect. Reduction in height of the soft tissue papillae covering interdental bone
Class IV	Recession extending to or beyond the mucogingival junction. Loss of interdental bone apically to recession defect. Gross flattening of interdental soft tissue papillae

The management of gingival recession and its sequelae is based on a thorough assessment of the etiological factors and the degree of tissue involvement. The initial phase of the management of a patient with gingival recession should be focused on correcting the etiological factors. The degree of gingival recession has to be monitored for signs of further progression. Surgical root coverage is indicated when esthetics is the prime concern and periodontal health is good. The techniques used for root coverage are based on tissue displacement, whether by translation (pedicle flap procedures) or by grafting (free gingival or connective tissue graft procedures), and the adjunct utilization of either resorbable or non-resorbable membranes as per the principles of guided tissue regeneration (GTR) (Wennstrom, 1996).

2.2.1. Epidemiology of gingival recession

Epidemiological studies have shown that more than 50% of subjects present one or

more sites with recession of at least 1 mm, with the buccal sites being the most commonly affected (Susin et al., 2004). Gingival recession has been found to occur in populations of industrialized (O'Leary et al., 1968; Gorman, 1967) and non-industrialized countries (Kumar, 1986; Akpata and Jackson,1979).

The occurrence of gingival recession is age dependent (Loe et al., 1992) but can be seen at all ages, starting early in life in some populations (Gorman, 1967; Björn et al., 1981). For example, gingival recessions were noticed in more than 60% of Norwegian 20-year-olds and in more than 90% of the older population (above 50 years) (Loe et al.,1992). Similar trends were found in Brazil (Susin et al., 2004) and France (Sarfati et al., 2010). In addition, Beck (1996) and Albandar (2000) stated that gingival recession increases in both prevalence and severity with age. It should be noted, however, that secondary tooth eruption may play a role in the establishment of gingival position (Theytaz et al., 2011). A study investigating 8 years of changes in the gingival contour of maxillary central incisors and first molars in adolescents and adults utilized digital tracing of the gingival tooth contours by means of calibrated photographs of two dental casts (taken 2 and 10 years post-orthodontic treatment) (Theytaz et al., 2011). The conclusion was that adolescents and adults presented apical displacement of the gingival contour of the maxillary first molar, as was the case for maxillary incisors in adolescents.

Moreover, higher levels of recession have been found in males than females (Susin et al., 2004; Slutzkey and Levin, 2008; Albandar and Kingman, 1999). According to data collected from the Third National Health and Nutrition Examination Survey, men present significantly more gingival recession, gingival bleeding, subgingival calculus, and teeth with total calculus than women (Albandar and Kingman, 1999). This can be the result of the larger tooth dimensions in men, among other factors (Othman and

Harradine, 2006; Chu, 2007).

2.2.2. Etiology of gingival recession

The etiology of gingival recession is multifactorial. Chan et al. (2015) categorized the etiological factors of gingival recession into predisposing and precipitating factors. Although predisposing factors are mainly linked to anatomical factors, such as the presence of underlying bone dehiscence and the thickness of gingival biotype, inflammation can also be implicated. There are two types of gingival biotype according to Ochsenbien and Ross (1969); the gingiva could be scalloped and thin, or flat and thick. They proposed that the contour of the gingiva closely followed the contour of the underlying bone. A gingival thickness of $\geqslant$2 mm was considered as a thick tissue biotype and a gingival thickness of <1.5 mm was referred as a thin tissue biotype (Claffey and Shanley, 1986). Data from a study suggest that in 85% of the population, the thick periodontal biotype was more prevalent than the thin scalloped form (15%) (Olsson and Lindhe, 1991). Thin gingival biotypes are delicate, highly scalloped and translucent in appearance. The soft tissue appears delicate and friable with a minimal amount of the attached gingiva. The underlying bone is thin or minimal bone is present over the labial roots with possible presence of fenestrations and dehiscences (Richard et al., 2008).

Additional anatomical predisposing factors include frenal pull and lack of adequate keratinized gingiva. Lang and Löe (1972) suggested that at least 2 mm of keratinized gingiva, corresponding to approximately 1 mm of attached gingiva, is recommended in order to maintain gingival health. Contradicting this, other studies have questioned this suggestion. Dorfman (1978) and Wennstrom (1990) stated that less than 1 mm of keratinized/attached gingiva might also be compatible with gingival health.

Furthermore, Coatoam et al. (1981) suggested that teeth with less than 2 mm of keratinized gingiva could withstand orthodontic forces.

Preventive measures have been advocated by some authors in order to avoid the development or progression of gingival recession in patients with the thin biotype (Holmes et al., 2005; McComb, 1994). Nonetheless, others have stated that muco-gingival surgical therapy could not be justified by the absence of keratinized gingiva alone (Farnaoush and Schonfeld, 1983; Kennedy et al., 1985). Two cross-sectional studies in children, adolescents and adults demonstrated that the width of keratinized gingiva increased with age (Bimstein and Eidelman, 1988; Vincent et al., 1976). Moreover, Bimstein and Eidelman (1988) observed that the attached gingiva tends to be narrower in the permanent when compared to the primary dentition. Others, on the other hand, did not find any increase in the width of attached gingiva from the deciduous to the permanent dentitions (Bosnjak et al., 2002; Tenenbaum and Tenenbaum, 1986). In a 10-year longitudinal study of untreated mucogingival defects, it was concluded that in the absence of gingival inflammation, areas with small amounts of keratinized gingiva may remain stable over long periods of time (Freedman et al., 1992).

Although lack of oral hygiene and subsequent inflammation are precipitating factors in gingival recession, overzealous oral hygiene can also have negative effects on the gingiva. Many studies found that recession occurred more in patients with good rather than poor oral hygiene (Loe et al., 1992; O'Leary et al., 1971; Serino et al., 1994). Recession at the buccal surfaces of teeth is common in populations with good oral hygiene (Serino et al., 1994; Neely et al., 2005; Sangnes and Gjermo, 1976), whereas in cases of poor oral hygiene, it might affect other tooth surfaces also (Baelum et al., 1986). Gingival recession at the lingual surfaces of lower anterior teeth has shown a

strong association with the presence of supragingival and subgingival calculus (van Palenstein Helderman et al., 1998). Gingival abrasion, also called toothbrush trauma, is a localized acute injury caused by vigorous brushing, excessive brushing pressure, or hard toothbrush bristles (Eden, 2008). Gingival abrasion and gingival recession are associated with improper vigorous tooth brushing (Vehkalahti, 1989). Additionally, Slutzkey and Levin (2008) showed that gingival inflammation had no correlation with gingival recession. Moreover, they found a negative correlation between plaque and gingival recession.

Other precipitating factors include smoking and oral piercing. There are inconsistencies in the literature regarding the relationship between cigarette smoking and gingival recession. In a cross-sectional study Albandar et al. (2000), examined a group of 705 individuals (21 to 92 years old) and it was found that cigarette and cigar/pipe smokers had a higher prevalence of moderate and severe periodontitis and a higher prevalence and extent of attachment loss and gingival recession than non-smokers; suggesting poorer periodontal health in smokers. In addition, smokers had less gingival bleeding and higher numbers of missing teeth than non-smokers. In another case-control type study (Calsina et al., 2002), it was found that probing depth, gingival recession and clinical attachment level were greater in smokers than in former smokers or non-smokers. Therefore, it was concluded that smoking is a risk factor strongly associated with periodontitis and the effects of smoking on periodontal tissues depend on the number of cigarettes smoked daily and the duration of the habit. The effect of tobacco on periodontal tissues seems to be more pronounced in men than in women (Calsina et al., 2002). On the other hand, there are several studies that found no association between gingival recession and smoking. In a 6-month follow-up study, a group of young subjects failed to show that smokers had an increased risk for

recession (Muller et al., 2002). Also, Slutzkey and Levin (2008), found no relationship between smoking and recession.

Another factor to be considered in the etiology of gingival recession is malocclusion. Anterior crossbite has been associated with this sequela due to causing occlusal trauma. Ustun et al. (2008) stated that a form of traumatic occlusion might develop when mandibular incisors erupt into an ectopic position, particularly in the labial direction. In these cases, a very narrow band of keratinized mucosa may usually be present. In addition, Lindhe et al. (2005) stated that trauma from occlusion promotes adaptive alterations or changes in the periodontal tissue as a result of excessive masticatory forces. It was also suggested that this phenomenon might occur in patients with anterior crossbite, which, due to its occurrence in an unfavorable anatomic region, can be a predisposing factor for bone loss and lead to gingival lesions. The combination of deficient oral hygiene and inflammation, compounded by occlusal trauma, may predispose to more rapid development of bone absorption than the one that would have occurred in the presence of gingival inflammation alone. Therefore, reduction of inflammation and plaque control are essential before, during, and after orthodontic treatment (Kessler, 1976). Tooth repositioning by orthodontic treatment may lead to spontaneous improvement of periodontal health, as placing the tooth in its proper alveolar envelope allows better distribution of forces, as well as bone remodeling (Andrade et al., 2014). Eismann and Prusas (1990) evaluated patients with anterior crossbite and gingival recession and found an increase in the gingival margin level of the mandibular incisors after one year of treatment.

Yared et al. (2006) investigated the pathogenesis of mucogingival defects in proclined mandibular incisors. They found that several factors such as brushing trauma, thin gingival biotype and the underlying alveolar bone contribute to the development of

mucogingival defects. Therefore, careful examination of the bony structure and the width of keratinized gingiva are necessary prior to orthodontic treatment, particularly when labial movement of the incisors will be required (Andrade et al., 2014). Although some authors claim that recession increases with labial movement of the incisors, even in the presence of adequate width of keratinized tissue (Closs et al., 2007), others have not found any correlation (Ruf et al., 1988). Geiger and Wasserman (1976) reported that the retroclined mandibular incisors of skeletal Class III patients are more susceptible to labial gingival recession and alveolar bone loss; thus, related the dentoalveolar adaptation of skeletal Class III expressed as mandibular incisor linguoversion to increased labial gingival recession. Therefore, orthodontic tooth movement in skeletal Class III patients may aggravate the periodontal support of the mandibular incisors. In accordance to the above findings, a correlation has been found between gingival recession and retroclined mandibular incisors in untreated adults with mandibular prognathism (Andlin-Sobocki and Persson, 1994). Vasconcelos et al. (2012) evaluated the gingival recessions in 57 patients using intra-oral slides. Their results showed that 10.4% of their patients presented gingival recessions on at least one of the mandibular incisors after orthodontic treatment. In addition, they showed that the risk of getting more severe recession is increased in cases with retroclined mandibular incisors such as in Class III cases.

2.2.3. Orthodontic treatment and gingival recession

As there is a general belief that during orthodontic treatment soft tissue attachment moves with the tooth (Zachrisson and Zachrisson, 1972), it has been proposed that a certain amount of attached gingiva is necessary for maintaining the integrity of the dento-gingival junction. However, the exact extent of the zone of keratinized gingiva

required before tooth movement for minimizing the occurrence of gingival recessions has never been established (Farnaoush and Schonfeld, 1983). Contrary to observations in untreated populations, it has been proposed that any existing mucogingival defect might be aggravated during orthodontic treatment (Coatoam et al., 1981), making it important to identify and treat any potential or actual areas of stress to the gingival tissues before starting orthodontic treatment. However, it has been shown that placing a free gingival graft prior to orthodontic treatment had no effect on the extent of the improvement of gingival architecture occurring during treatment (Ngan et al., 1991) and the subject still remains controversial in the orthodontic and periodontic literature (Dorfman, 1978; Kennedy et al., 1985; Wennstrom, 1990). Nevertheless, if recession increases during orthodontic treatment, then a gingival graft procedure can be justified.

Moreover, as already mentioned, one of the long-term complications of fixed appliance orthodontic treatment is apical soft tissue relocation and gingival recession. The precise biological mechanism for these processes remains unclear and orthodontic therapy has long been debated as a precipitating factor. However, a systematic review suggested that the incidence of gingival recession in patients undergoing orthodontic treatment is less than 10% (Joss-Vassalli et al., 2010).

Proclination of mandibular incisors in particular has been suggested to be a risk factor for progressive bone loss and development of gingival recession (Vasconcelos et al., 2012). Some authors have reported that excessive proclination of mandibular incisors in adults will result in labial bone dehiscence and the consequent retraction of the gingival margin (Artun and Krogstad, 1987, Yared et al., 2006). Dorfman (1978) showed that labial movement of the mandibular incisors resulted in a decrease in the width of keratinized gingiva, whereas significant lingual positioning of the

mandibular incisors caused an increase in keratinized gingiva. Other studies, however, found no correlation between the amount of proclination of mandibular incisors and gingival recession (Ruf et al., 1998, Djeu et al., 2002). Artun et al. (2001) concluded that adolescent orthodontic patients with dentoalveolar retrusion may be treated with pronounced advancement of mandibular incisors without increasing the risk of recession. Furthermore, a correlation has been found between gingival recession and retroclined mandibular incisors in untreated adults with mandibular prognathism (Andlin-Sobocki and Persson, 1994).

In several clinical situations, pronounced orthodontic expansion is implemented as an alternative to extractions in order to achieve optimal esthetic and occlusal results. Garib et al. (2006) concluded that rapid maxillary expansion appliances reduced the buccal bone plate thickness of the supporting teeth and increased the lingual plate thickness. The reduction in the buccal plate thickness induced bone dehiscences on the buccal aspects of the anchor teeth, especially in subjects with thinner buccal bone plates. Therefore, the risk of developing bony dehiscences and gingival recession may lead clinicians to prefer extraction to expansion in borderline cases (Bassarelli et al., 2005).

Active orthodontic treatment is typically followed by a retention phase. This involves the use of different retainers, fixed or removable, to maintain the results achieved during orthodontic treatment. The use of a fixed retainer made of wire and composite resin bonded to the lingual and/or palatal surfaces of the anterior teeth is a common practice at completion of orthodontic treatment. Gingival health can be affected when the retainer is placed close to the gingival tissues due to the obstruction of proper oral hygiene measures and the consequent plaque accumulation (Pender, 1986; Lundstrom and Krasse, 1987; Olympio et al., 2006). The role of fixed retainers and the

development of gingival recession is controversial. It had been suggested that orthodontic treatment followed by retention by fixed lingual retainers aggravates the development of recession (Pandis et al., 2007). A study by Levin et al. (2008) investigated the association between orthodontic treatment and post-orthodontic retention with gingival health. They measured plaque and gingival indices, gingival recession, probing depth, and bleeding on probing of the anterior sextants of 92 subjects who presented at the military dental clinic for routine dental examinations. These authors found that labial gingival recession was significantly higher in treated (0.13 ±0.22mm) compared to untreated patients (0.05 ±0.2mm; p=0.03). Therefore, they concluded that orthodontic treatment and fixed retainers were associated with an increased incidence of recession. Contrary to this, other authors found that fixed retainers do not exert any negative side effects on periodontal health (Artun et al., 1997; Booth et al., 2008).

Overall, the level of available evidence regarding the association of orthodontic treatment with gingival recession is low, the amount of recession found in studies with statistically significant differences is of questionable clinical consequence, and should be interpreted with caution (Joss-Vassalli et al., 2010). Whether orthodontic tooth movement can cause gingival recession, or whether the alveolar bone and gingiva adapt to the new position of the tooth in the absence of other traumatic factors, like vigorous tooth brushing, still remains the subject of controversy.

3. AIM

3.1. Aim of the systematic review

To systematically investigate and appraise the quality of the available evidence regarding clinical crown length changes and gingival recession development of anterior teeth following orthodontic treatment.

3.2. Objectives of the systematic review

To retrieve data on clinical crown length changes and gingival recession development of anterior teeth following orthodontic treatment.

3.3. Null hypotheses

a) There is no change in clinical crown length of anterior teeth following orthodontic treatment.

b) There is no change in gingival recession prevalence of anterior teeth following orthodontic treatment.

c) There is no difference in the prevalence of gingival recession in anterior teeth following orthodontic treatment in comparison to non-treated subjects.

d) There is no change in the gingival recession measurement of anterior teeth following orthodontic treatment.

e) There is no difference in the gingival recession measurement of anterior teeth following orthodontic treatment in comparison to non-treated subjects.

4. MATERIALS AND METHODS

4.1 Protocol development and registration

The present review was based on a specific protocol developed and piloted following the guidelines outlined in the PRISMA-P statement (Shamseer et al., 2015) and registered in PROSPERO (CRD42018080945) (Appendix I). In addition, conduct and reporting followed the Cochrane Handbook for Systematic Reviews of Interventions (Higgins and Green, 2011) and the PRISMA statement (Moher et al., 2009), respectively.

4.2. Eligibility criteria

The selection criteria for the domains of study design, participants' characteristics, intervention characteristics and principal outcome measures applied for the present review were as follows:

4.2.1. Types of study design

Studies included in the present thesis had to be experimental studies evaluating changes in clinical crown length and the development of gingival recession in anterior teeth before and at specific time points after the completion of comprehensive orthodontic treatment. Case-control studies were also considered. Animal studies, case reports and reviews (traditional reviews, systematic reviews and meta-analyses) were not included in the present investigation.

The type of study design was assessed using the algorithm available from SIGN (Scottish Intercollegiate Guidelines Network) available from http://www.sign.ac.uk (Appendix II).

4.2.2. Types of participants

The included studies could involve patients of any age and gender. Studies focusing on specific groups instead of the general orthodontic population, like patients with pre-existing recessions prior to the initiation of orthodontic treatment, or developed after orthodontic treatment, were excluded. Moreover, patients with clefts, syndromes or congenital anomalies of the craniofacial region, congenitally missing teeth and developmental dental anomalies were not considered.

4.2.3. Types of interventions

The included studies should involve patients who completed a full course of comprehensive orthodontic treatment with full fixed appliances in both arches. Interventions including orthognathic surgery; limited orthodontic treatment or treatment adjuncts that have been shown to induce tooth proclination (like the Herbst appliance) were excluded.

4.2.4. Types of outcome measures

The studies included in the present review had to primarily provide measurements on clinical crown length changes (in mm), in the labial/buccal aspect of individual anterior teeth, following orthodontic treatment.

Moreover, included studies must provide measurements on recession development in the labial/buccal aspect of anterior teeth, following orthodontic treatment, either in the form of prevalence (presence or absence) in individual anterior teeth or groups of teeth, or recession measurements (in mm) in individual anterior teeth.

Studies reporting pooled clinical crown length changes (in mm) or recession measurements (in mm) for groups of teeth (i.e. all incisors, all anterior teeth, etc.)

were excluded because such measurements cannot be considered to be statistically independent of each other.

4.3. Information sources and search strategy

The principal investigator (BS) developed detailed search strategies for each database searched. These were based on the strategy developed for MEDLINE, but revised appropriately for each database to take account of the differences in controlled vocabulary and syntax rules. The following electronic databases were searched (Appendix III): MEDLINE via PubMed, CENTRAL, Cochrane Systematic Reviews, Scopus, Web of Science™ Core Collection, Arab World Research Source, Clinical Trials registry and ProQuest Dissertations and Theses Global database.

No restriction was placed on the language, date or status of publication. In addition, efforts were made to obtain conference proceedings and abstracts where possible and the reference lists of all eligible studies for additional records were searched.

4.4. Study selection

The principal investigator (BS) and a co-investigator (Shatha Al-Khalidy) assessed the retrieved records for inclusion independently. They were not blinded to the identity of the authors, their institution, or the results of the research. They obtained and assessed, again independently, the full report of records considered by either reviewer to meet the inclusion criteria. Disagreements were resolved by discussion or consultation with the thesis co-supervisor (EGK). A record of all decisions on study identification was kept.

4.5. Data collection and data items

The same two persons (BS and EGK) performed data extraction independently and any disagreements were again resolved by discussion or consultation with the thesis principal supervisor (AEA). Data collection forms were used to record the desired information.

a. Bibliographic details of the study.

b. Details on study design and verification of study eligibility.

c. Participant characteristics (inclusion and exclusion criteria and where available number, age, gender).

d. Intervention characteristics (e.g. type of appliances, extraction/non-extraction treatment).

e. Details on outcomes assessed and assessment procedures.

f. Additional information: other variables considered, a prior sample size calculation, methodology reliability assessment.

If clarifications were needed regarding the published data, or additional material was required, then attempts to contact the corresponding authors would be made.

The retrieved data on the assessed outcomes was categorized as follows:

a. Changes in clinical crown length following orthodontic treatment.

b. Changes in recession prevalence following orthodontic treatment.

c. Differences in recession prevalence following orthodontic treatment in comparison to non-treated subjects.

d. Changes in recession measurement following orthodontic treatment.

e. Differences in gingival recession measurements following orthodontic treatment in comparison to non-treated subjects.

4.6. Risk of bias in individual studies

The principal investigator (BS) and the thesis co-supervisor (EGK) assessed the risk of bias in the included studies, independently and in duplicate, during the data extraction process, using the ROBINS-I tool (Risk Of Bias In Non-randomised Studies of Interventions) (Sterne et al., 2016). Any disagreements were resolved by discussion or consultation with the thesis principal supervisor (AEA).

The ROBINS-I tool assessment tool includes the following domains:

a. Bias due to confounding.

b. Bias in selection of participants into the study.

c. Bias in classification of interventions.

d. Bias due to deviations from intended interventions.

e. Bias due to missing data.

f. Bias in measurements of outcomes.

g. Bias in selection of the reported results.

After entering in the data extraction form the information reported in each study, every domain would receive a judgment of either low, moderate, serious or critical risk of bias or the label “no information” (indicating no information on which to base a judgment about risk of bias for this domain) (Sterne et al., 2016).

Subsequently, studies were to be judged as being of low, moderate, serious or critical risk of bias (Sterne et al., 2016):

a. Low risk of bias: The study is comparable to a well-performed randomized control.

b. Moderate risk of bias: The study provides sound evidence for a non-randomized study but cannot be considered to be a well performed randomized trial.

c. Serious risk of bias: The study has some important problems.

d. Critical risk of bias: The study is too problematic to provide any useful evidence and should not be included in any synthesis.

e. No information: There is no information on which to base a judgment about risk of bias.

4.7. Summary measures and synthesis of results

In situations where the retrieved data used different continuous variables measuring the same concept on different scales with a high degree of correlation, the effects of the interventions were planned to be expressed as standardized values (i.e. the Standardized Mean Difference (SMD) together with the relevant 95% Confidence Interval (CI)), in order to enable quantitative synthesis (Deeks et al., 2001). In cases where a particular comparison of the same variable was recorded, the intervention effect was planned to be expressed as the Weighted Mean Difference (WMD) together with the 95% CI. Data from dichotomous outcomes were expressed as Odds Ratios (OR) together with the 95% CI.

The random effects method for meta-analysis was to be used to combine data from studies that reported similar measurements in appropriate statistical forms (Der Simonian and Laird, 1986, Borenstein et al., 2009), since they were expected to differ across studies due to clinical diversity in terms of participant and intervention characteristics.

To identify the presence and extent of between-study heterogeneity, the overlap of 95% CI for the results of individual studies was to be inspected graphically, and Cochrane's test for homogeneity and the I^2 statistic were to be calculated (Higgins and Green, 2011). The results of the I^2 statistic were to be interpreted as follows (Higgins and Greene, 2011):

- I^2 from 0% to 40%: heterogeneity might not be important;
- I^2 from 30% to 60%: may represent moderate heterogeneity;
- I^2 from 50% to 90%: may represent substantial heterogeneity;
- I^2 from 75% to 100%: considerable heterogeneity.

All analyses were to be carried out with Comprehensive Meta-analysis software 2.2.046 (©2007 Biostat Inc., New Jersey, USA). Significance (a) was set at 0.05, except for 0.10 used for the heterogeneity tests (Ioannidis, 2008).

4.8. Risk of bias across studies and additional analyses

If a sufficient number of trials were identified, analyses were planned for "small-study effects" and publication bias (Higgins and Green, 2011). If deemed possible, exploratory subgroup analyses were planned according to participant and intervention characteristics. Finally, the quality of evidence for the comparison of incisor and anterior teeth recession prevalence following orthodontic treatment at the longest follow-up available was assessed based on the Grades of Recommendation, Assessment, Development and Evaluation (GRADE) approach (Guyatt et al., 2011).

5. RESULTS

5.1. Study selection

The flowchart of records through the reviewing process is shown in Figure 1. Initially 5045 records were identified, 203 were excluded as duplicates and 4702 more were excluded on the basis of their title and abstract as they were irrelevant to the topic of the present review (cadaver studies, animal studies, reviews, case reports, etc.). The remaining 140 articles were examined in full-text and 130 were excluded for various reasons including the following: case reports (n=45), discussed treatment of recession (n=30), factors related to the methodology of the article (n=20), treatment in mixed dentition cases (n=10), issues regarding follow-up period (n=10), systematic reviews (n=6), discussed pre-surgical orthodontics (n=4), treatment with Herbst appliance (n=3), meta-analysis (n=1), or an irrelevant paper in Chinese after translation (n=1). Finally, ten full-text reports were included in the systematic review (Djeu et al., 2002; Allais and Melsen, 2003; Melsen and Allais, 2005; Konikoff et al., 2007; Renkema et al., 2013a; Renkema et al., 2013b; Renkema et al., 2013c; Kamak et al., 2015; Renkema et al., 2015; Juloski et al., 2017).

5.2. Study characteristics

The general characteristics of the studies included in the present systematic review, together with sample characteristics, are presented in Tables 1 and 2. They were published between 2002 and 2017, and followed patients up to 6 years into retention. They investigated either changes in clinical crown length (Djeu et al., 2002; Konikoff et al., 2007; Renkema et al., 2013a; Kamak et al., 2015; Renkema et al., 2015) or recession development (Djeu et al., 2002; Renkema et al., 2013b, Renkema et al.,

2013c; Juloski et al., 2017; Allais and Melsen, 2003; Melsen and Allais, 2005). Most studies compared measurements before and then at specific time points following orthodontic treatment (Djeu et al., 2002; Melsen and Allais, 2005; Konikoff et al., 2007; Renkema et al., 2013a; Renkema et al., 2013c; Renkema et al., 2015; Kamak et al., 2015; Juloski et al., 2017). The studies that followed patients during retention all used fixed lingual retainers, either Type 1 (bonded to the mandibular canines) or Type 2 (bonded to all 6 mandibular anterior teeth) retainers (Renkema et al., 2013a, Renkema et al., 2013b; Renkema et al., 2013c; Renkema et al., 2015; Juloski et al., 2017). In addition, three case-control studies were identified with orthodontically treated subjects, with fixed retention (Renkema et al., 2013b; Juloski et al., 2017) or without (Allais and Melsen, 2003), which were compared to untreated control groups.

With regards to age, most of the studies selected patients less than 18 years old at the start of treatment, except for 4 studies (Djeu et al., 2002; Allais and Melsen, 2003, Melsen and Allais, 2005; Konikoff et al., 2007). The majority of the studies involved a mixture of treatments including extraction and non-extractions, with two exceptions (Allais and Melsen, 2003; Melsen and Allais, 2005), which included non-extraction treatment plans with labial movement of the lower incisors. Although most of the studies did not mention the type of fixed appliances used, Djeu et al. (2002) specifically mentioned that the patients they investigated were treated with 3 different bracket systems (standard edgewise, straight wire, and Tip-Edge) while Kamak et al. (2015) stated that all their patients were treated using straight-wire appliances.

The majority of the studies examined the mandibular incisors (Djeu et al., 2002; Allais and Melsen, 2003; Melsen and Allais, 2005; Renkema et al., 2013a; Renkema et al., 2015; Kamak et al., 2015), except for Konikoff et al. (2007), which studied the

upper incisors, Renkema et al. (2013b; 2013c) studied the development of recession in all teeth and Juloski et al. (2017) investigated the mandibular anterior teeth. Other variables considered in the retrieved studies included: a) *dental and occlusal characteristics*: overjet, overbite, Little's Irregularity Index, mandibular arch length, Angle's classification, lack of space, incisor rotation, width of incisors; b) *lateral cephalometric measurements representative of mandibular incisor inclination and position*; and c) *periodontal parameters*: visible plaque, gingival inflammation, gingival biotype, width of keratinized tissue and calculus accumulation.

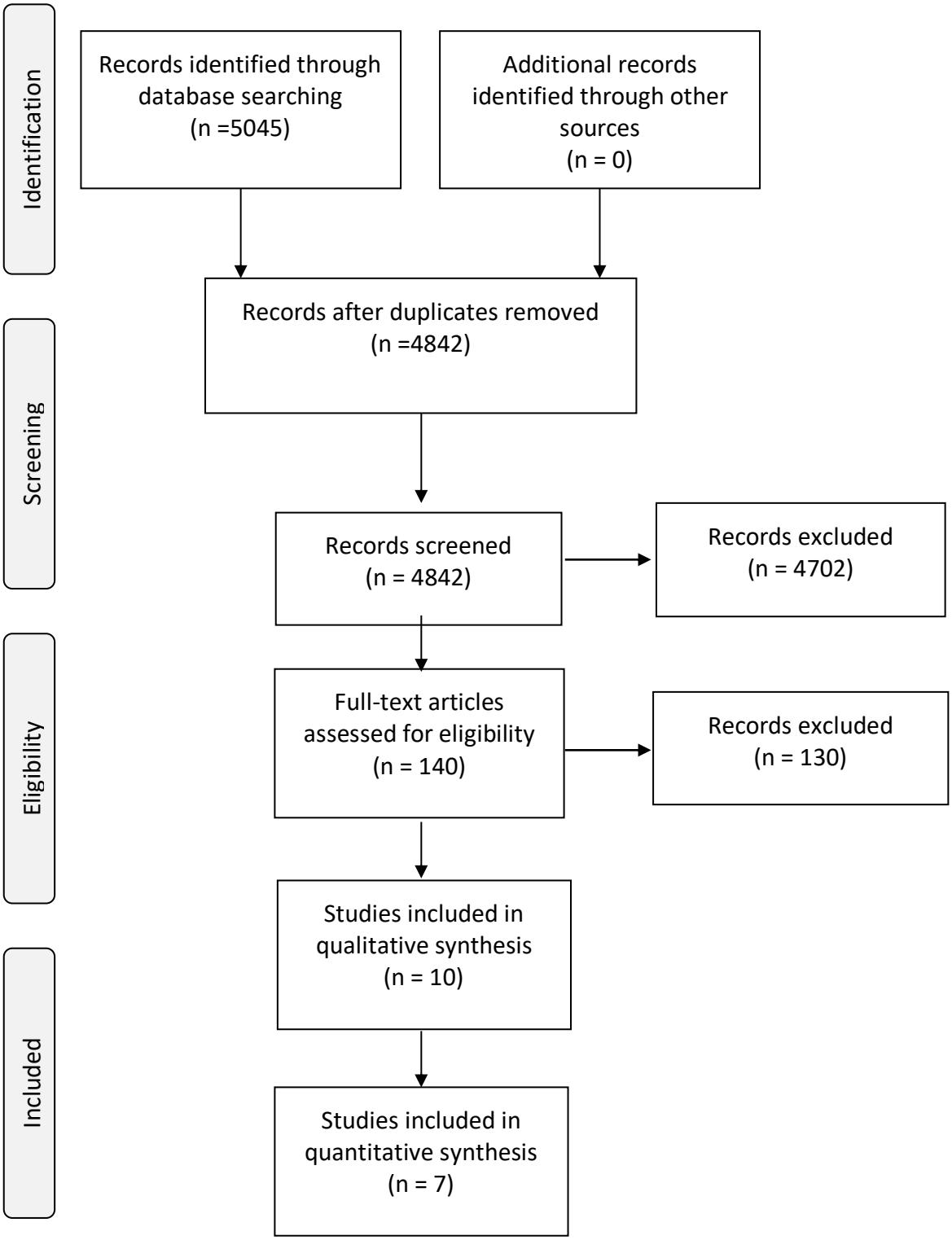

Figure 1. Flowchart of records through the reviewing process.

Table 2. General characteristics of the studies included in the systematic review.

Study	Intervention characteristics	Study outcomes and other variables considered [including methods of assessment]	Additional information
Djeu et al. [2002] Before and after study	Fixed appliances on both arches [27 standard edgewise, 34 straight wire, 6 tip edge] [52 non-ex, 12 all 4s ex, 3 all 5s ex]	**Clinical crown length [mm]: Md central Is; Pre-Tx, Post-Tx** On study models, from the deepest point of the curvature of the vestibule-gingival margin to the incisal edge. These measurements were made to the nearest 0.5 mm using a Boley gauge accurate to 0.05 mm. **Recession measurement [mm]: Md central Is; Pre-Tx, Post-Tx** Intraoral photographic slides were projected. All measurements were made from the deepest point of the curvature of the gingival margin to the cementoenamel junction to the nearest 0.5 mm. All data were obtained at the same magnification and extrapolation of the actual recession height was performed. **Other variables considered:** Inc-MP [°]; Inc-A-Pog [mm]; Inc-NB [°]; Inc-NB [mm]; Pre-Tx, Post-Tx Sagittal changes in mandibular central incisor inclination and position that occurred during fixed appliance therapy from lateral cephalometric radiographs. Proclination was defined as a positive difference between the final angle/distance and the beginning angle/distance. A negative difference or no difference was considered not proclined.	**Power calculation:** None **Method error:** paired t-test; p>0.05
Allais and Melsen [2003] Case-control study	Fixed orthodontic appliances with labial movement of Md Is	**Gingival recession [mm]; Md Is; Pre-Tx, Post-Tx** IO slides and study models, measured at the mid-labial site of each of the four lower incisors as the distance between the gingival margin and the cemento–enamel junction (CEJ). When the CEJ was not visible a 0 mm measurement was recorded. **Other variables considered:** IO slides: Gingival biotype, width of keratinized gingiva, gingival inflammation, and visible plaque. Lateral ceph: Inc-A-pog line (mm), Inc-ML [°], ML-NL [°]; Pre-Tx – Inc-A-pog line (mm) measured as the linear distance between the Md incisal point (incision inferior) and the A-Pog line – Inc-ML [°] measured as the angle between the Md incisor axis and the Md line – ML-NL [°] measured as the angle in degrees between the Md line and the Mx line Study models: - Mandibular arch length [mm] Pre-Tx, Post-Tx;On study models, measured as the difference between the Post-Tx distance from the mesial contact point of the first mandibular molars to the inter-incisal point of the lower central incisors using an electronic caliper with a scale, which included hundredths of millimetres (0.01 mm). - Overjet, overbite, canine relationship, lack of space, incisor rotation, width of incisors Pre-Tx.	**Power calculation:** Yes **Method error:** paired t-test; p>0.05
Melsen and Allais [2005] Before and after study	Fixed appliances without extraction and with proclination of Md Is.	**Gingival recession [mm]; Md Is; Pre-Tx, Post-Tx** IO slides and study models, measured at the mid-labial site of each of the four lower incisors as the distance between the gingival margin and the cemento–enamel junction (CEJ). When the CEJ was not visible a 0 mm **Other variables considered:** IO slides: Gingival biotype, width of keratinized gingiva, gingival inflammation, and visible plaque. Lateral ceph: Inc-A-pog line (mm), Inc-ML [°], ML-NL [°]; Pre-Tx – Inc-A-pog line (mm) measured as the linear distance between the Md incisal point (incision inferior) and the A-Pog line – Inc-ML [°] measured as the angle between the Md incisor axis and the Md line – ML-NL [°] measured as the angle in degrees between the Md line and the Mx line Study models: - Mandibular arch length [mm] Pre-Tx, Post-Tx;On study models, measured as the difference between the Post-Tx distance from the mesial contact point of the first mandibular molars to the inter-incisal point of the lower central incisors using an electronic caliper with a scale, which included hundredths of millimetres (0.01 mm). - Overjet, overbite, canine relationship, lack of space, incisor rotation, width of incisors Pre-Tx.	**Power calculation:** yes **Method error:** paired t-test; p>0.05

4s: first premolars; 5s: second premolars; Ex: extraction; I: Incisor; m: months; Md: Mandibular; Mx: Maxillary; Pre-Tx: Pre-treatment, Post-Tx: Post-treatment; Tx: treatment; y: years; +/-; presence or absence; Inc: Inclination;

Table 2. General characteristics of the studies included in the systematic review. [Continued]

Study	Intervention characteristics	Study outcomes and other variables considered [including methods of assessment]	Additional information
Konikoff et al. [2007] Before and after study	No information regarding type of orthodontic treatment	**Clinical crown length [mm]; Mx Is** **Part 1 of study: Pre-Tx, Post-Tx; Study models** Measurements were made from the gingival margin to the incisal edge, and both apical and coronal to a line drawn between the tips of the papillae on either side of these teeth. They were also measured for the distance between the interproximal contacts as seen from the frontal view. This was accomplished by marking the mesial and distal dimension of each tooth as seen from directly in front of the model on a sheet of graphing paper. The distance as seen from the front was then measured on the paper with the digital caliper. **Part 2 of the study: Clinical examination** All clinical measurements were made by the same digital caliper from part 1, which included the distance from the zenith od the scallop to the incisal edge for Mx Is **Other variables:** Part 1 of the study: – Clinical crown width [mm]; Mx Is; Pre-Tx, Post-Tx – Calculated the ratio of gingival vs. incisal measurements – Calculated the ratio of width-to-length – Comparisons of all measurements to accepted values. Part 2 of the study: – Clinical crown width [mm]; Mx Is: used same measurements of part 1 for the clinical crown width for the 2nd part of the study. – Calculated the ratio of width-to-length from Pre-Tx and Post-Tx models and clinical measurements. – Comparisons of all measurements to accepted normal values.	**Power calculation:** none **Method error**: none
Renkema et al. [2013a] Before and after study	Full fixed appliances	**Clinical crown length [mm]: Md central Is; Pre-Tx, Post-Tx, 2y post-Tx, 5y post-Tx** On study models, the distances between the incisal edges and the deepest points of the curvature of the gingival margin of all four mandibular incisors were measured with an electronic caliper with an accuracy of 0.01 mm. **Recession prevalence [+/-]: Md central Is; Pre-Tx, Post-Tx, 2y post-Tx, 5y post-Tx** On study models, a recession was noted if the labial cementoenamel junction was exposed [pre-Tx recession was scored for all teeth]. **Other variables considered:** Inclination of the incisors [Inc_Incl]: Pre-Tx, Post-Tx, 2y post-Tx, 5y post-Tx On lateral cephalometric radiographs, angle between the long axis of the Md I and the line connecting menton (the lowest point of the mandibular symphysis) and gonion (the most inferior posterior point of the mandibular angle). Retroclined group: Δinc_Incl≤–1º; Stable group: Δinc_Incl>–1º and ≤1º; Proclined group: Δinc_Incl>1º. Gender; age; ex vs. non-ex treatment	**Power calculation:** none **Method error:** Spearman's correlation, duplicate measurement error (DME), paired t-test; various differences noted
Renkema et al. [2013b] Case-Control study	Cases: full fixed appliances with bonded lingual retainer after orthodontic treatment Controls: untreated subjects	**Recession prevalence [+/-]; all teeth** On study models, a recession was noted yes if the labial cement-enamel junction was exposed at T0, T1, T3, and T6 on all teeth	**Power calculation:** none **Method error:** Kappa Statistics
Renkema et al. [2013c] Before and after study	Full fixed appliances with bonded lingual retainer after orthodontic treatment. Exo/Non- exo	**Recession prevalence [+/-]; all teeth** On study models, a recession was noted yes if the labial cement-enamel junction was exposed at T0, T1, T2, and T5 on all teeth	**Power calculation:** none **Method error:** Kappa Statistics (Mean Kappa value was 0.98)

4s: first premolars; 5s: second premolars; Ex: extraction; I: Incisor; m: months; Md: Mandibular; Mx: Maxillary; Pre-Tx: Pre-treatment, Post-Tx: Post-treatment; Tx: treatment; y: years; +/-; presence or absence; Inc: Inclination

Table 2. General characteristics of the studies included in the systematic review. [Continued]

Study	Intervention characteristics	Study outcomes and other variables considered [including methods of assessment]	Additional information
Renkema et al. [2015] Before and after study	Fixed appliances in both dental arches [the type of fixed appliance was not determined]	**Clinical crown length [mm]: Md Is; Pre-Tx, Post-Tx, 5y post-Tx** On study models, the distances between the incisal edges and the deepest points of the curvature of the vestibulo-gingival margin of all four mandibular incisors were measured with an electronic calliper with an accuracy of 0.01 mm. **Recession prevalence [+/-]: Md Is; Pre-Tx, Post-Tx, 2y post-Tx, 5y post-Tx** On study models, a recession was noted if the labial cementoenamel junction was exposed [pre-Tx recession was scored for all teeth]. The presence of gingival recession 5 years after treatment was scored only for the Md Is **Other variables considered:** Inclination of the incisors [Inc_Incl]: Pre-Tx, Post-Tx, 5y post-Tx On lateral cephalometric radiographs, angle between the long axis of the Md I and the line connecting menton (the lowest point of the mandibular symphysis) and gonion (the most inferior posterior point of the mandibular angle Gender; age; ex vs. non-ex treatment	**Power calculation:** none **Method error:** Spearman's Correlation, duplicate measurement error (DME), paired t-test
Kamak et al. [2015] Before and after study	Upper and lower straight-wire appliances for at least 12 months	**Clinical crown heights [mm]; Md Is; Pre-Tx & Post-Tx** On study models, measured as the distances between the incisal edges and the deepest points of the curvature of the vestibule-gingival margin of all four mandibular incisors, with an electronic caliper with an accuracy of 0.01 mm. **Amount of labial recession [mm]; Md Is; (CCHafter-CCHbefore)** The amounts of labial gingival recessions were measured by extracting T0 clinical crown height from T1 crown height (clinical crown heights at T1 − clinical crown heights at T0) **Other variables considered:** Lateral ceph: Inc-MP Pre-Tx & Post-Tx The angle between mandibular plane (MP) and the long axis of most forward lower incisor (lower incisor inclination)	**Power calculation:** none **Method error:** intra-class coefficients
Juloski et al. [2017] Before and after study Case-control study	Full fixed appliances Exo, Non-exo	**Recession prevalence [+/-]: Md anteriors; Pre-Tx, Post-Tx, 5y post-Tx** On study models, a recession was noted if the labial cementoenamel junction was exposed. IO photographs were used to confirm the presence of gingival recession in the experimental groups but no intraoral color photographs were available for the control group. **Other variables considered:** Overjet, Overbite, Little's Irregularity Index at T0, T1, and T5, Yes/No calculus accumulation	**Power calculation:** yes **Method error:** Interclass coefficient used for intrarater reliability and Cohen's kappa statistics

4s: first premolars; 5s: second premolars; Ex: extraction; I: Incisor; m: months; Md: Mandibular; Mx: Maxillary; Pre-Tx: Pre-treatment, Post-Tx: Post-treatment; Tx: treatment; y: years; +/-; presence or absence; Inc: Inclination

Table 3. Sample characteristics in the studies included in the systematic review.

Study	Inclusion and exclusion criteria	Analyzed sample
Djeu et al. [2002] Before and after study	**Inclusion criteria:** Complete sets of pre-treatment and post-treatment study casts, lateral cephalograms, and clinical intra-oral slides **Exclusion criteria:** No exclusion criteria (due to the paucity of complete records available), but none of the patient charts noted pre-existing systemic diseases or medications associated with gingival changes.	**67 patients (39F, 28M)** **Pre-Tx age:** mean 16.4 y (range: 10-45 y); 15 patients were 18 y or older **Tx duration:** mean 33.2 m (range: 8-71 m) **Race:** African Americans (n=7), Asian Americans (n=4), White (n=56) **Type of Maloccluion:** Angle Class I (n=50), Angle Class II (n=11), Angle Class III (n=6)
Allais and Melsen [2003] Case-control study	**Inclusion Criteria:** Class II and I patients only **Exclusion Criteria:** Surgical cases and Class III	**Treated Group:**150 adult patients (114F, 36M) **Pre-Tx Age:** Females: 33.7 ± 9.5 y (Range: 22-65 y) Males: 33.9 ± 7.4 y (Range: 23-50 y) **Race:**White Caucasians **Type of malocclusion:** Class I and Class II **Control Group:** 150; matched with respect to sex and age, deviating by no more than 1 y when comparing Post-Tx record-age of the samples with the Pre-Tx record-age of the controls • All patients had received hygiene instructions Pre-Tx and had been routinely checked throughout tx. • Post-Tx IO photos and casts were taken one month after removal of appliances
Melsen and Allais [2005] Before and after study	**Inclusion Criteria:** Active treatment finished during period of 1990-1996, White, More than 20 y of age at referral, Class I and II patients, Non-extraction, Tx complete, Pre-Tx and Post-Tx records **Exclusion Criteria:** Dental or Skeletal Class III malocclusion, Mandibular orthognathic surgery, Pathology influencing bone turnover, Medicine intake, Fillings or endodontic-periodontic lesion at mandibular incisors.	**150patients (114F, 36M)** **Pre-Tx Age:** Females: 33.7 ± 9.5 y (Range: 22-65 y) Males: 33.9 ± 7.4 y (Range: 23-50 y) **Race:** Whites **Type of malocclusion:** Class I and Class II
Konikoff et al. [2007] Before and after study	**Inclusion Criteria:** Subjects with completed orthodontic movement of maxillary centrals, laterals, and canines.	**Part 1:** 200 plaster models (Age: 8-31+ y) **Part 2:** 31 subjects were recalled after 5 y Post- Tx for re-examination. Post-Tx records taken at the day of appliance removal
Renkema et al. [2013a] Before and after study	**Inclusion Criteria:** 11-14 y of age at the start of orthodontic treatment; Presence of four fully erupted lower incisors before and after treatment; A bonded canine to canine retainer placed directly after active treatment with full fixed appliances; No visible wear of lower incisor edges; No retreatment; Dental casts and lateral cephalometric radiographs available Pre-Tx, Post-Tx, 2 y Post-Tx, and 5 y Post-Tx	**179 subjects (77M, 102F)** **Retroclined group (N=34):** **Pre-Tx age**: mean 12.52 y (SD: 0.88 y) **Tx duration**: mean 2.79 y (SD: 0.75 y) **Stable group (N=22):** **Pre-Tx age:** mean 12.38 y (SD: 0.86 y) **Tx duration:** mean 2.45 y (SD: 1.03 y) **Proclined group (N=123):** **Pre-Tx age**: mean 12.32 y (SD: 0.74 y) **Tx duration:** mean 2.67 y (SD: 0.73 y)

Pre-Tx: Pre-treatment, Post-Tx: Post-treatment; Tx: treatment; y: years; +/-; presence or absence; Inc: Inclination

Table 3. Sample characteristics in the studies included in the systematic review. [Continued]

Study	Inclusion and exclusion criteria	Analyzed sample
Renkema et al. [2013b] Case-Control study	**Inclusion Criteria for Cases:** Treated with full fixed appliances, A bonded lingual retainer placed directly after active orthodontic treatment (either bonded only at mandibular canines or bonded to all 6 anterior teeth), No orthodontic re-treatment, Initial, end of treatment, and long term after treatment casts made at ages of 12 (Pre-Tx), 15 (Post-Tx), 18 (3 y Post-Tx), and 21 (6 y Post-Tx). E**xclusion Criteria for Cases:** Combined orthodontic/surgical treatment, Restorative treatment (except for single crowns) after orthodontic therapy, Dental casts of poor quality, especially in the area of gingival margin. **Inclusion Criteria of Control group:** Healthy patients, Called for examination at 6 years and then every 3 years until the age of 21, Norwegian Caucasians with normal occlusion in sagittal, vertical and transverse dimensions, Only minor deviations in form of rotations and/or spacing, No facial disharmony, dental casts made at age of 12, 15, 18, and 21 available and presence of full dentition at 21years. **Exclusion Criteria of Control group:** Missing teeth/tooth, Restorative treatment (except for single crowns), and Dental casts of poor quality in the gingival area.	**220 subjects** **Cases:** 100 orthodontically treated patients (mean length of treatment was 2.8 y and ranged from 1.4 to 4.4 y) **Pre-Tx age**: 12.13 y (0.54) **Post-Tx age:** 14.86 y (0.68) **3 y Post-Tx:** 17.24 y (0.84) **6 y Post-Tx:** 20.37 y (0.86) **Controls:** 120 healthy subjects **T at age of 12**: 11.82 y (0.38) **T at age of 15**: 14.80 y (0.38) **T at age of 18:** 18.13 y (0.37) **T at age of 21:** 21.37 y (0.55) *Age not matched between cases and controls *No association between Angle Class and No. of recessions in cases was found (p=0.938).
Renkema et al. [2013c] Before and after study	**Inclusion Criteria:** Treated with full fixed appliances, A bonded lingual retainer placed directly after active orthodontic treatment (either bonded only at mandibular canines or bonded to all 6 anterior teeth), No orthodontic re-treatment, Initial, end of treatment, and long term after treatment casts made at ages of Pre-Tx, Post-Tx, 2y Post-Tx, and 5y Post-Tx	**302 orthodontic patients (117 male; 185 female)** **Mean Tx-duration:** 2.8 y **Pre-Tx age:** 13.6 y (3.6) **Post-Tx age:** 16.2 y (3.5) **2 y Post-Tx age**: 18.6 y (3.6) **5 y Post-Tx age:** 21.6 y (3.5)

Pre-Tx: Pre-treatment, Post-Tx: Post-treatment; Tx: treatment; y: years; +/-; presence or absence; Inc: Inclination

Table 3. Sample characteristics in the studies included in the systematic review. [Continued]

Study	Inclusion and exclusion criteria	Analyzed sample
Renkema et al., [2015] Before and after study	**Inclusion Criteria:** 11-14 y of age at the start of orthodontic treatment, Presence of four fully erupted lower incisors before and after treatment, A bonded canine to canine retainer placed directly after active treatment with full fixed appliances, No visible wear of lower incisor edges, No retreatment, Dental casts and lateral cephalometric radiographs available Pre-Tx, Post-Tx, 2 years Post-Tx, and 5y Post-Tx **Exclusion Criteria:** Combined orthodontic/surgical treatment, Restorative treatment of the mandibular incisors after orthodontic therapy, Dental casts of poor quality, particularly in the area of the gingival margin.	**179 subjects (77M, 102F)** Ended up with 117 subjects as they excluded the group of stable inclination (N=62) **Proclined group: N=60** **Pre-Tx age:** 12.2 y (0.83) **Post-Tx age:** 14.79 y (1.1) **Tx- duration:** 2.8 y (0.81) **Non-proclined group: N= 57** **Pre-Tx age:** 12.42 y (1.26) **Post-Tx age:** 15.32 y (1.46) **Tx-duration:** 2.91 y (0.85)
Kamak et al. [2015] Before and after study	**Inclusion Criteria:** Radiographic and plaster dental casts of good quality **Exclusion Criteria:** Subjects with missing teeth, Subjects with gingival recession, Subjects with any congenital deformities such as cleft palate.	**109 subjects (79F, 39M)** **Retroclination (N=32)** **Pre-Tx age:** 14.20 y (3.67) **Post-Tx age:** 15.96 y (3.71) **Tx-duration**: 1.76 y (0.53) **Stable position (N=13)** **Pre-Tx age:** 12.59 y (1.49) **Post-Tx age:** 14.35 y (1.42) **Tx-duration:** 1.77 y (0.91) **Proclination (N=64)** **Pre-Tx age:** 14.58 y (4.30) **Post-Tx age:** 16.39 y (4.23) **Tx-duration:** 1.81 y (0.64) Pre-Tx: records: taken one month prior to active orthodontic treatment Post-Tx records: taken on the day of removal of active orthodontic appliances

Pre-Tx: Pre-treatment, Post-Tx: Post-treatment; Tx: treatment; y: years; +/-; presence or absence; Inc: Inclination

Table 3. Sample characteristics in the studies included in the systematic review. [Continued]

Study	Inclusion and exclusion criteria	Analyzed sample
Juloski et al. [2017] Before and after study Case-control study	**Inclusion Criteria for experimental group:** Treated with full fixed appliances, Orthodontic treatment started before the age of 18, Good quality dental casts and intra oral photographs before treatment (Pre-Tx), 4-6 weeks after debonding (Post-Tx), 5y Post-Tx. **Exclusion Criteria for the experimental group:** Missing or extracted anterior teeth in the mandibular jaw, Restorative treatment due to caries or trauma, Orthognathic surgery treatment **Inclusion Criteria for untreated group:** Healthy subjects, Records available (models, panoramic radiographs, and cephalometric radiographs), Recalled for dental examination at ages of 6, 9, 12, 15, 18 and 21, No orthodontic treatment	**144 patients (65 males, 79 females)** **Group 1: Treatment without retention experimental group (n=48)** **Pre- Tx age:** 12.39 ± 1.52 y **Post-Tx age:** 14.61 ± 1.67 y **5y Post-Tx age**: 19.93 ± 1.65 y **Tx-duration**: 2.18 ± 0.77 y **Group 2: Treatment with retention experimental group (n=48)** **Pre- Tx age:** 12.78 ± 1.36 y **Post-Tx age:**14.98 ± 1.41 y **5y Post-Tx age:**20.27 ± 1.47 y **Tx-duration:** 2.17 ± 0.65 y **Group 3: untreated controls (n=48)** **Pre- Tx age:**11.73 ± 0.36 y **Post-Tx age:**14.71 ± 0.34 y **5y Post-Tx age**: 21.18 ± 0.46 y **Tx-duration:** 2.98 ± 0.11 y

Pre-Tx: Pre-treatment, Post-Tx: Post-treatment; Tx: treatment; y: years; +/-; presence or absence; Inc: Inclination

5.3. Risk of bias within studies

Table 3 presents the summary of findings regarding risk of bias assessment for the ten included studies (Djeu et al., 2002; Allais and Melsen, 2003; Melsen and Allais, 2005; Konikoff et al., 2007; Renkema et al., 2013a; Renkema et al., 2013b; Renkema et al., 2013c; Renkema et al., 2015; Kamak et al., 2015; Juloski et al., 2017). All of these were considered overall as being of serious risk of bias.

In general, all studies included in the present review were considered to present serious risk of bias regarding confounding, as important parameters (like the natural progression of the related phenomena with age, gender, occlusal relationships, oral hygiene measures and habits, gingival biotype, plaque and calculus accumulation, existing periodontal disease and previous periodontal treatment, smoking, force systems employed during orthodontic treatment, orthodontic treatment duration, wear of the incisal or occlusal aspects of teeth, etc.) were not always appropriately controlled. With regards to bias in classification of interventions, Konikoff et al. (2007) was assessed to be at serious risk as no specific information on whether orthodontic treatment was completed was provided; instead, a statement that subjects had completed movement of maxillary central, lateral and canine was provided. The risk of bias in the measurement of outcomes was considered low for seven studies (Djeu et al., 2002; Allais and Melsen, 2003; Melsen and Allais, 2005; Renkema et al., 2013c; Renkema et al., 2015; Kamak et al., 2015; Juloski et al., 2017) but of serious risk for the study of Konikoff and co-workers (2007), as there was no method error and inter- and intra-examiner reliability of assessing clinical crown lengths. In addition, Renkema et al. (2013b) showed serious risk of bias in this section since the observers were not blinded to the origin of the dental casts (i.e. whether they scored

the presence or absence of recession in cases or controls). Also, Renkema et al. (2013a) showed serious risk of bias in the measurement of outcomes due to issues observed during method error assessment. Finally, the risk of bias in selection of the reported result was deemed to be moderate all ten studies.

Table 4. Summary of the risk of bias assessment. [Domains examined: 1: Bias due to confounding 2: Bias in selection of participants, 3: Bias in classification of intervention, 4: Bias in measurement of outcomes, 5: Bias in selection of the reported result]

Domain	Djeu et al. [2002]	Allais and Melsen [2002]	Melsen and Allais [2005]	Konikoff et al. [2007]	Renkema et al. [2013a]	Renkema et al. [2013b]	Renkema et al. [2013c]	Renkema et al. [2015]	Kamak et al. [2015]	Juloski et al. [2017]
1	Serious	Serious	Serious	Serious	Serious	Serious	Serious	Serious	Serious	Serious
2	Low	Low	Low	Low	Low	Low	Low	Low	Low	Low
3	Low	Low	Low	Serious	Low	Low	Low	Low	Low	Low
4	Low	Low	Low	Serious	Serious	Serious	Low	Low	Low	Low
5	Moderate	Moderate	Moderate	Moderate	Moderate	Moderate	Moderate	Moderate	Moderate	Moderate
Overall	**Serious**	**Serious**	**Serious**	**Serious**	**Serious**	**Serious**	**Serious**	**Serious**	**Serious**	**Serious**

5.4. Results of individual studies and synthesis of results

The results of the studies included in the present review are presented below.

5.4.1. Changes in clinical crown length following orthodontic treatment.

Four studies investigated changes in lower incisor clinical crown lengths following orthodontic treatment (Djeu et al., 2002; Renkema et al., 2013a; Kamak et al., 2015; Renkema et al., 2015). One study reported measurements on the upper incisors (Konikoff et al., 2007).

Overall, compared to the baseline, incisor clinical crown lengths increased immediately after orthodontic treatment and 5 years into retention (Table 5).

Table 5. Changes in clinical crown length compared to pre-treatment [mm].

	WMD [95% CI]; *p*-value; N; I^2	
Mx teeth	**Immediately post-treatment**	**5 years post-treatment**
11	0.100 [0.053 to 0.147]; 0.000*; 1; NA *Konikoff et al., 2007*	0.600 [0.312 to 0.888]; 0.000*; 1; NA *Konikoff et al., 2007*
12	0.400 [0.226 to 0.574]; 0.000*; 1; NA *Konikoff et al., 2007*	1.000 [0.563 to 1.437]; 0.000*; 1; NA *Konikoff et al., 2007*
21	0.100 [0.057 to 0.143]; 0.000*; 1; NA *Konikoff et al., 2007*	0.800 [0.451 to 1.149]; 0.000*; 1; NA *Konikoff et al., 2007*
22	0.500 [0.280 to 0.720]; 0.000*; 1; NA *Konikoff et al., 2007*	1.300 [0.732 to 1.868]; 0.000*; 1; NA *Konikoff et al., 2007*
Md teeth		
31	0.092 [0.018 to 0.167]; 0.015*; 5;0% *Kamak et al., 2015*	0.673 [0.517 to 0.829]; 0.000*; 3; 41% *Renkema et al., 2013a*
32	0.148 [0.019 to 0.277]; 0.025*; 3; 49% *Djeu et al., 2002; Kamak et al., 2015*	0.955 [0.820 to 1.090]; 0.000*; 3; 27% *Renkema et al., 2013a*
41	0.119 [0.043 to 0.195]; 0.002*; 5; 75% *Djeu et al., 2002; Kamak et al., 2015*	0.642 [0.379 to 0.904]; 0.000*; 3; 74% *Renkema et al., 2013a*
42	0.166 [0.080 to 0.251]; 0.000*; 3;1% *Kamak et al., 2015*	0.929 [0.825 to 1.034]; 0.000*; 3;0% *Renkema et al., 2013a*

*Statistically significant difference
I^2: Inconsistency; Md; mandibular; Mx: maxillary; N: number of groups in the synthesis; NA: not applicable; WMD: Weighted Mean Difference; 95% CI: 95% Confidence Interval

5.4.2. Changes in recession prevalence following orthodontic treatment.

Four studies investigated changes in recession prevalence following orthodontic treatment (Melsen and Allais 2005; Renkema et al., 2013b; Renkema et al., 2013c; Juloski et al., 2017). Overall, orthodontic treatment did not seem to increase the odds for a patient to exhibit gingival recession immediately post-treatment in individual teeth compared to before treatment (Table 6).

Table 6. Changes in recession prevalence following orthodontic treatment in individual teeth [Melsen and Allais, 2005].

	OR [95% CI]; *p*-value; N; I^2
Teeth	**Immediately post-treatment**
31	1.261 [0.993 to 1.602]; 0.057; 1; NA
31 [Recession > 2mm]	1.227 [0.843 to 1.786]; 0.285; 1; NA
32	1.216 [0.970 to 1.523]; 0.090; 1; NA
32 [Recession > 2mm]	1.276 [0.910 to 1.788]; 0.157; 1; NA
41	1.286 [1.018 to 1.624]; 0.035*; 1; NA
41 [Recession > 2mm]	1.276 [0.910 to 1.788]; 0.157; 1; NA
42	1.217 [0.958 to 1.545]; 0.108; 1; NA
42 [Recession > 2mm]	1.276 [0.910 to 1.788]; 0.157; 1; NA

*Statistically significant difference
I^2: Inconsistency; Md; mandibular; Mx: maxillary; N: number of groups in the synthesis; NA: not applicable; WMD: Weighted Mean Difference; 95% CI: 95% Confidence Interval

In follow-ups up to 6 years, orthodontic treatment seemed to increase the odds for a patient to exhibit gingival recession, in at least one mandibular incisor or anterior tooth compared to before treatment (Table 7).

Table 7. Changes in recession prevalence following orthodontic treatment in groups of teeth.

	OR [95% CI]; *p*-value; N; I^2	
Follow-up	**Mandibular incisors**	**Mandibular anterior teeth**
Post-Tx	1.347 [1.113 to 1.630]; 0.002*; 3; 0% *Melsen and Allais, 2005; Renkema et al., 2013b; 2013c*	1.242 [0.891 to 1.733]; 0.201; 2; 0% *Juloski et al., 2017*
2 years	1.934 [1.427 to 2.621]; 0.000*; 1; 0% *Renkema et al., 2013c*	
3 years	2.018 [0.751 to 5.426]; 0.164; 1; 0% *Renkema et al., 2013b*	
5 years	2.078 [1.661 to 2.600]; 0.000*; 1; 0% *Renkema et al., 2013c*	1.775 [1.350 to 2.333]; 0.000*; 2; 0% *Juloski et al., 2017*
6 years	2.126 [1.414 to 3.196]; 0.000*; 1; 0% *Renkema et al., 2013b*	

*Statistically significant difference
I^2: Inconsistency; Md; mandibular; Mx: maxillary; N: number of groups in the synthesis; NA: not applicable; OR: Odds Ratio; 95% CI: 95% Confidence Interval

5.4.3. Difference in recession prevalence following orthodontic treatment in comparison to non-treated subjects.

Three studies investigated the prevalence of recession after orthodontic treatment in comparison to untreated controls immediately after treatment; 3 years post-treatment (Allais and Melsen, 2003; Renkema et al., 2013b), 5 years post-treatment (Juloski et al., 2017), and 6 years post-treatment (Renkema et al., 2013b) (Table 8). Overall, orthodontic treatment seemed to increase the odds for a patient to exhibit gingival recession, in at least one mandibular incisor, compared to the untreated controls.

Table 8. Changes in recession prevalence following orthodontic treatment in treated cases versus untreated controls.

	OR [95% CI]; p-value; N; I2			
Tooth	**Immediately post-treatment**	**3 Years Post-Treatment**	**5 Years Post-Treatment**	**6 Years Post-Treatment**
31	1.475 [0.797 to 2.728]; 0.216; 1; NA *Allais and Melsen, 2003*			
32	2.718 [1.399 to 5.278]; 0.003*; 1; NA *Allais and Melsen, 2003*			
41	1.871 [1.007 to 3.479]; 0.048*; 1; NA *Allais and Melsen, 2003*			
42	1.904 [0.963 to 3.762]; 0.064; 1; NA *Allais and Melsen, 2003*			
Md Ant	1.462 [0.609 to 3.506]; 0.395; 1; NA *Juloski et al. (2017)*		2.083 [1.091 to 3.977]; 0.026*; 1; NA *Juloski et al. (2017)*	
Md I	3.367 [1.990 to 5.698]; 0.000*; 1; NA *Allais and, Melsen, 2003; Renkema et al., 2013b*	6.117 [0.290 to 128.901]; 0.244; 1; NA *Renkema et al. (2013b)*		8.816 [1.939 to 40.078]; 0.005*; 1; NA *Renkema et al. (2013b)*

*Statistically significant difference
I^2: Inconsistency; Md; mandibular; N: number of groups in the synthesis; NA: not applicable; OR: Odds Ratio; 95% CI: 95% Confidence Interval

5.4.4. Changes in recession measurement following orthodontic treatment.

Djeu et al. (2002) investigated the change in recession following orthodontic treatment. It was shown that recession length increased after orthodontic treatment [31: mean 0.030; 95%CI: -0.004 to 0.064; p=0.079][41: mean 0.080; 95%CI: 0.011 to 0.149; p= 0.024].

5.4.5. Difference in gingival recession measurement following orthodontic treatment in comparison to non-treated subjects.

Allais and Melsen (2003) investigated the difference in gingival recession measurement following orthodontic treatment in comparison to non-treated subjects. Overall, no statistically significant differences were noted [31; Treated: mean ±SD: 0.3 ± 0.81 mm; Untreated: mean ±SD: 0.3 ± 0.83 mm; p=1.000; t-test] [32; Treated: mean ±SD: 0.4 ± 0.86 mm; Untreated: mean ±SD: 0.2 ± 0.71 mm; p= 0.0357; t-test] [41; Treated: mean ±SD: 0.4 ± 0.86 mm; Untreated: mean ±SD: 0.3 ±0.80 mm; p=

0.2995; t-test] [42; Treated: mean ±SD: 0.3 ± 0.83 mm; Untreated: mean ±SD: 0.2 ± 0.62 mm; p= 0.2402; =t-test].

5.5. Risk of bias across studies and additional analyses

As it was not possible to retrieve a sufficient number of trials, we were not able to conduct analyses for "small-study effects" and publication bias (Higgins and Green, 2011).

Comparison of changes in clinical crown length following orthodontic treatment between groups of patients where the lower incisors had been proclined during treatment to those where the lower incisors had been kept stable or retroclined, showed that, in general, no differences were noted between the two groups immediately after and 5 years following treatment (Djeu et al., 2002; Renkema et al., 2013a; Kamak et al., 2015) (Table 9).

Table 9. Comparison of changes in clinical crown length [mm] between groups of patients with proclined and non-proclined incisors.

	WMD [95% CI]; *p*-value; N; I^2	
Md teeth	**Immediately post-treatment**	**5 years post-treatment**
31	0.008 [-0.133 to 0.150]; 0.906; 3;0% *Kamak et al., 2015*	-0.214 [-0.454 to 0.026]; 0.080; 2;0% *Renkema et al., 2013a*
32	-0.086 [-0.0295 to 0.124]; 0.424; 2; 34% *Djeu et al., 2002; Kamak et al., 2015*	-0.193 [-0.440 to 0.053]; 0.124; 2; 0% *Renkema et al., 2013a*
41	0.002 [-0.181 to 0.184]; 0.986; 3; 59% *Djeu et al., 2002; Kamak et al., 2015*	-0.320 [-0.575 to -0.064]; 0.014*; 2; 0% *Renkema et al., 2013a*
42	-0.025 [-0.194 to 0.145]; 0.777; 2;6% *Kamak et al., 2015*	-0.117 [-0.325 to 0.091]; 0.268; 2;0% *Renkema et al., 2013a*

*Statistically significant difference; I^2: Inconsistency; Md; mandibular; Mx: maxillary; N: number of groups in the synthesis; WMD: Weighted Mean Difference; 95% CI: 95% Confidence Interval

Comparison of changes in clinical crown length following orthodontic treatment between groups of patients where the post-treatment inclination of lower incisors was

<95º or >100.5º, showed that, in general, no differences were noted between the two groups 5 years following treatment (Renkema et al., 2015) (Table 10).

Table 10. Comparison of changes in clinical crown length [mm] between groups of patients where the post-treatment inclination of lower incisors was <95º or >100.5º.

	WMD [95% CI]; *p*-value; N; I^2
Md teeth	**5 years post-treatment**
31	-0.080 [-0.330 to 0.190]; 0.583; 1; NA *Renkema et al., 2015*
32	-0.270 [-0.550 to -0.220]; 0.048*; 1; NA *Renkema et al., 2015*
41	0.090 [-0.180 to 0.360]; 0.521; 1; NA *Renkema et al., 2015*
42	-0.030 [-0.260 to 0.200]; 0.783; 1; NA *Renkema et al., 2015*

*Statistically significant difference
I^2: Inconsistency; Md; mandibular; Mx: maxillary; N: number of groups in the synthesis; NA: Not applicable; WMD: Weighted Mean Difference; 95% CI: 95% Confidence Interval

Comparison of recession prevalence 5 years following orthodontic treatment between groups of patients where the lower incisors had been proclined during treatment to those where the lower incisors had been kept stable or retroclined, showed that no significant difference in the odds ratio [OR: 2.524; 95% CI: 0.820 to 7.768; *p*=0.106] (Renkema et al., 2013a). The same was observed when recession prevalence 5 years following orthodontic treatment was compared between groups of patients where the final inclination of the lower incisors were <95º and >100.5 º [OR: 1.06; 95% CI: 0.347 to 3.238; *p*=0.919] (Renkema et al., 2015).

Comparison of changes in recession measurement following orthodontic treatment between groups of patients where the lower incisors had been proclined during treatment to those where the lower incisors had been kept stable or retroclined, showed that no differences were noted between the two groups immediately after

treatment (Djeu et al., 2002) [31; Non-proclined: mean ±SD: 0.022 ±0.116 mm; Proclined: mean ±SD: 0.048 ±0.175mm; p= 0.500; t-test] [41; Non-proclined: mean ±SD: 0.041 ±0.212 mm; Proclined: mean ±SD: 0.105 ±0.326 mm; p= 0.3721; t-test]. Overall, the quality of evidence for the prevalence of recession in mandibular incisor and anterior teeth at the longest follow-up was considered as low (Table 11).

Table 11. Quality of available evidence.

Quality assessment						Patients	Effect	Quality
Studies	**Risk of bias**	**Inconsistency**	**Indirectness**	**Imprecision**	**Other**		**Absolute Odds Ratio (95% CI)**	
Recession prevalence following orthodontic treatment | Mandibular incisors [6 years]								
1	Serious[1]	Not serious	Not serious	Serious[2]	No	302	**2.216 higher** (1.414 to 3.196) ***p*=0.000**	⨁⨁◯◯ **LOW**
Recession prevalence following orthodontic treatment | Mandibular anterior teeth [5 years]								
1	Serious[1]	Not serious	Not serious	Serious[2]	No	96	**1.775 higher** (1.350 to 2.333) ***p*=0.000**	⨁⨁◯◯ **LOW**

CI: Confidence interval
[1] Studies were considered as being of serious risk of bias. [2] The results are based only on one study.

6. DISCUSSION

6.1. Summary of evidence

Although the movement of teeth outside the envelope of alveolar bone has been reported as a risk factor for gingival recession, until now, the subject still remains controversial. Based on the information provided in the present review, although clinical crown length increases during orthodontic treatment and retention, overall no statistically significant differences were noted between incisors having undergone proclination compared to the non-proclination group. Moreover, some increases in gingival recession prevalence may be encountered after orthodontic treatment. However, the concerns raised during quality assessment of the available evidence provide food for thought regarding the confidence in the observed estimates.

From the initially identified records, only ten full-text studies provided data regarding changes in clinical crown length and the development of gingival recession in anterior teeth before, and at specific time points after, the completion of comprehensive orthodontic treatment or relevant information from the comparison with untreated populations. The consequent lack of extensive data is somewhat surprising, since it is likely that under certain circumstances, teeth following orthodontic treatment are positioned in unfavorable positions relative to their supporting structures, something which could possibly lead to apical soft tissue relocation, increase in clinical crown length and eventually gingival recession (Wennstrom et al., 1987). In addition, labial movement of the incisors is the only alternative to extraction and combined surgical-orthodontic therapy (Allais and Melsen, 2003). The current trend in orthodontics is for increasing numbers of patients to be treated without extraction of teeth. For example,

in 1986 almost 35% orthodontic patients in the USA had teeth extracted, whereas in 2008 only 18% patients were treated with extractions (Keim et al., 2008). Animal experiments have shown that movement of the tooth outside the bony envelope, as may occur in the excessive proclination of incisors, might result in the development of gingival recession (Batenhorst et al., 1974). As a result, it has been speculated that labial movement of incisors in humans is also a risk factor for the development of recession (Artun and Krogstad, 1987).

The present systematic review and meta-analysis demonstrated that clinical crown length increased after orthodontic treatment and during retention (Djeu et al., 2002; Renkema et al., 2013a; Kamak et al., 2015). When patients with orthodontically proclined incisors were compared to patients without proclination, no overall differences were shown (Djeu et al., 2002; Renkema et al., 2013a; Kamak et al., 2015). The same lack of differences was noted after comparing changes in clinical crown length between groups of patients where the post-treatment inclination of lower incisors was <95° or >100.5°, 5 years following treatment (Renkema et al., 2015). These findings seem to be in agreement with the results of various investigations with no explicitly defined time points of follow-up. Ruf et al. (1998) analyzed the changes in mandibular incisor inclination in teenagers treated with the Herbst appliance and found that the mean proclination of lower incisors by 8.9° did not increase the clinical crown length. Artun and Grobéty (2001) also followed groups with pronounced and no advancement of mandibular incisors during treatment and reported no difference in the increase in clinical crown height from after treatment to follow-up. Similarly, Antonarakis et al. (2017) concluded that proclination of lower incisors beyond a 10° limit by means of orthodontics alone, or in combination with anterior mandibular alveolar process distraction osteogenesis did not significantly increase the labial

clinical crown height. In addition, Choi et al. (2015) showed that clinical crown length increased (0.30-0.37mm) during the pre-surgical orthodontic treatment in both groups, with proclined and minimally proclined teeth. They stated that apical movement of the free gingival margin occurs regardless of the amount of labio-version of the mandibular incisors. According to Artun and Krogstad (1987), clinical crown height increased 0.76 mm and 0.31mm from before treatment to removal of appliances in the proclination and minimal-change groups, respectively. Coatoam et al.(1981) investigated the clinical crown length changes in the 12 anterior teeth. It was shown that the anterior teeth presented a statistically significant increase in the length of the clinical crown. The greatest increase in the length of the clinical crown was seen in the mandibular cuspids (average 0.95 mm). The smallest increase during orthodontic treatment was seen in the maxillary central incisors (average 0.18 mm). It was suggested that the increase in the clinical crown lengths of the cuspids was due to the continued active eruption of these teeth, which would have occurred in the absence of orthodontic treatment. Villard and Patcas (2015) concluded that there are noticeable increases in clinical crown length of the incisors and canines during the retention period, although this lengthening is not statistically significant. Canines appear to be more susceptible to gingival recession than incisors. Few studies showed that the clinical crown lengths did not increase following orthodontic treatment. De Olivera et al. (2017) showed that, in spite of the difference in the amount of vestibular inclination of the mandibular incisors during treatment, the clinical crown length remained unaltered in both groups (proclined and minimally proclined).

In general, immediately post-treatment, a patient was not at greater risk of exhibiting gingival recession in individual teeth compared to before treatment (Melsen and Allais, 2005; Renkema et al., 2013b; Renkema et al., 2013c; Juloski et al., 2017).

However, in follow-up up to 6 years, orthodontic treatment seemed to have increased the likelihood for a patient to exhibit gingival recession in at least one mandibular incisor or anterior tooth, compared to before treatment and to untreated controls (Melsen and Allais, 2005; Renkema et al., 2013b; Renkema et al., 2013c; Juloski et al., 2017). Comparison of recession prevalence 5 years following orthodontic treatment between groups of patients where the lower incisors had been proclined during treatment to those where the lower incisors had been kept stable or retroclined, showed that no significant difference in the odds ratio. Artun and Krogstad (1987) showed more gingival recessions following lower incisor proclination. The correct amount of proclination of the lower incisors in which no negative periodontal defects such as alveolar bone dehiscences and fenestrations of the buccal and lingual cortical plates and gingival recessions, is a topic of continuing debate. Slutzkey and Levin (2008) stated that the prevalence of recession was correlated with previous orthodontic treatment and the use of dental piercing. They examined 303 young adults (18-22 years) and found a strong correlation between severity and extension of recession and orthodontic treatment. Investigating Class III patients decompensated before orthognathic surgery, Choi et al. (2015) evaluated whether the periodontal alterations in the mandibular incisors that underwent minimal vestibular inclination were similar to those that were significantly tipped bucally. They observed that the mandibular incisors that had been substantially inclined towards the vestibular region during dental decompensation presented greater retraction of the vestibular cortical bone as well as a reduction in the strip of keratinized gingiva. Nevertheless, the amount of gingival recession appears to be clinically insignificant. Ruf et al., (1998) analyzed the changes in mandibular incisor inclination in teenagers treated with the Herbst appliance and development of gingival recessions 6 months after treatment.

They found that the mean proclination of lower incisors by 8.9° did not increase the risk of recessions. Antonarakis et al. (2017) showed that proclination of lower incisors of 10° or more, regardless of whether this was achieved by orthodontic tooth movement or by surgical displacement of the whole alveolar process, can increase the risk of developing lingual gingival recessions by 17 times in any particular patient; whereas this was not the case for labial gingival recessions. Contrary to the above findings, there are various studies showing that there is no correlation between the amount of proclination and gingival recession (de Olivera et al., 2016; Yared et al., 2006; Artun and Grobety 2001). De Olivera et al. (2016) showed that, even in the proclined group, no appearance of gingival recessions occurred in the mandibular incisor region 3 years after orthodontic treatment. Similarly, Yared et al. (2006) evaluated the periodontal condition of mandibular incisors after orthodontic treatment and concluded that there was no correlation between gingival recession and the degree of vestibular inclination of these teeth. They also stated that recession is not a direct consequence of incisor proclination, but excessive final inclination of this tooth, in addition to an individual characteristic of thin gingival margin, can render it susceptible to the development of recession defects. From Artun and Grobety (2001) it may be concluded that the mandibular incisors may be advanced considerably during active treatment of Class II malocclusions in young adolescent patients without any increase in the risk of gingival recession

Another study investigated the long-term prevalence of gingival recession after orthodontic treatment (Morris et al., 2017). It was found that there was little recession immediately after orthodontic treatment, with most (86.5%-97.8%) teeth exhibiting no recession. However, significant recession occurred in the time period after removal of the appliances until the follow-up period. The mandibular central incisors showed the

second highest prevalence of recession during the follow up period after maxillary first premolars, with almost 53% exhibiting recession, and 10.3% showing greater than 1 mm of recession. They concluded that only 0.6% of teeth exhibited 1mm or more recession, suggesting that the amount of recession observed was not caused by orthodontic treatment but might be age related. Substantially greater degrees of recession occurred during the 15.8 years that the patients were followed after orthodontic treatment. At approximately 32.3 years of age, 55.7% of the patients in this study demonstrated gingival recession on at least 1 tooth, and 41.7% of all teeth exhibited recessions. Post-treatment gingival recession was greater for the mandibular central incisors than the lateral incisors. Compared with the lateral incisors, the prevalence of recession on the mandibular central incisors was 15.8% greater and the extent of recession (≥ 1 mm) was 6.4% more.

No differences were noted in gingival recession measurements immediately following orthodontic intervention compared to before treatment and to untreated controls (Djeu et al., 2002; Allais and Melsen, 2003). Morris et al. (2017) measured labial gingival recessions of mandibular incisors. They found that the mandibular central incisors showed the most recession, with 12.8% exhibiting 0.1 to 1.0 mm of recession and 0.7% with greater than 1.0 mm.

Closs et al. (2007) showed that, in terms of severity, at the beginning of orthodontic treatment it was not possible to quantify any amount of recession in the majority of teeth (44%), 32% showed recession of less than 1 mm, and only 6 teeth (24%) presented with recession between 1 and 3 mm. At the end of treatment, teeth with recessions between 0 and 2 mm represented 47% of the sample; 5.1% had recession ≥ 3 mm. Their conclusion was that alterations in the gingival margin, especially gingival recession, occur in patients after orthodontic therapy, but the extent and

severity of this finding are low. This is in concordance with several studies (Levin and Samorodnitzky, 2008; Sluzkey and Levin, 2008), which showed that, in orthodontically treated patients, the prevalence, severity, and extent of recession have been reported to be higher than in untreated controls.

Overall, the quality of the retrieved evidence was considered as low, based on the Grades of Recommendation, Assessment, Development and Evaluation (GRADE) approach (Guyatt et al., 2011). Apart from concerns regarding the precision of the observed estimates, in general, all studies were considered to present serious risk of bias as confounding parameters were not always appropriately controlled for. With regards to bias in classification of interventions, Konikoff et al. (2007) was assessed to be at serious risk as no specific information on whether orthodontic treatment was completed was provided; instead a statement that subjects had completed movement of maxillary central incisors, lateral incisors and canines was provided. Additionally, the risk of bias in the measurement of outcomes was considered a serious risk in the study of Konikoff et al. (2007) as there was no method error and inter- and intra-examiner reliability performed for the assessment of clinical crown lengths. In addition, Renkema et al. (2013b) showed serious risk of bias in this respect due to the fact that the observers were not blinded to the origin of the dental casts (i.e. whether they scored the presence or absence of recession in cases or controls) and Renkema et al. (2013a) reported issues during the method error assessment process.

6.2. Strengths and limitations

The strengths of the present review include using a methodology following well-established guidelines. Moreover, the search strategy employed in the present review

was both exhaustive, covering electronic, manual, and gray literature material up to October 2017, and comprehensive, including every available study investigating the impact of orthodontic treatment on the development of gingival recession, irrespective of language, date and status of publication. Every effort to minimise bias in the methodology employed was made. Screening, verification of eligibility, abstraction of information, assessment of risk of bias and of the quality of evidence were all performed in duplicate, and any disagreement was resolved by discussion or consultation until a final consensus was achieved. Finally, the random effects model was employed during exploratory quantitative data synthesis to incorporate any observed heterogeneity (Lau et al., 1997).

However, despite these precautions, many limitations to the present review still exist, arising mainly from the nature and the characteristics of the data retrieved during the review process, which resulted in the assessment of the level of available evidence as low. The scarcity of relevant information led to meta-analytic procedures that aspired to be regarded as only exploratory until additional research becomes available. Nevertheless, current concepts support that even data from as few as two studies can be combined, provided that these can be meaningfully pooled (Ryan, 2013), as all other summarizing techniques are less transparent and/or are less likely to be valid (Valentine et al., 2010). In addition, analyses for "small-study effects" and publication bias (Higgins and Green, 2011) could not be carried out, despite being incorporated as possibilities according to the review protocol. Moreover, in some cases the small number of patients finally analyzed resulted in subsequent problems regarding the precision of the effect estimates.

Another limitation concerning the data retrieved in this study stems from the absence of control groups in most of the reviewed studies. It should be stressed that

orthodontic treatment per se may be conducive to development of gingival recessions irrespective of the direction of tooth movement. Elucidation of this issue would require a control group comprising subjects not treated orthodontically. Only 3 studies compared, in a cross-sectional manner, orthodontically treated patients to untreated healthy controls (Allais and Melsen, 2003; Renkema et al., 2013b; Juloski et al., 2017).

Most of the selected studies were also found to have methodological limitations. One of the limitations was the method of measuring gingival recession from the clinical crown height on a dental cast. Clinical crown height might vary during orthodontic treatment due to occlusal changes such as tooth wear from appliances, which can result in a reduction of clinical crown lengths. Additionally, crown fracture or restoration of the teeth might also alter the height of the clinical crowns. Occlusal changes can also occur due to treatment mechanics, such as extrusion, which could potentially have an effect on the clinical crown heights in the form of an increase in the clinical crown height post treatment.

Also, it is worth mentioning that the studies available in the literature used either clinical examination and/or photos-slides and/or study models. The lack of standardisation in retrieving intraoral photos-slides and the quality of laboratory manufacturing of study models pertains with certain problems of appropriateness of these methods to detect the clinical parameters of interest. The precision of the plaster models is influenced significantly by the processing aspects and impression technique (Rudolph et al., 2015). Therefore any impression and/or casting errors, such as volumetric changes of impression materials and expansion of dental stone (Richert et al., 2017), could possibly have an effect on the results of the study. In addition, clinical examination has to be performed by calibrated and experienced assessors.

To overcome these difficulties, new digital impression methods, using intra-oral scanners, are currently available in the market (Richert et al., 2017). The greatest benefit for dental lab technicians and dentists in adopting digital technology lies in eliminating conventional impression-taking procedures, clinicians no longer need to worry about the possibility of error due to air bubbles breaking the impression materials, displacement and movement of the tray, tray deflection, insufficient impression material, inadequate impression adhesive, or distortion resulting from disinfecting procedures (Birnbaum 2010). Although there is limited information about this subject, intraoral scanners are evolving to become valuable tools for the recording of intraoral soft and hard tissues and the monitoring of changes. Kattadiyil et al. (2014) succeeded in the fabrication of a removable partial denture framework using an intraoral scanner to capture soft tissue precisely in the case of a limited tooth-supported clinical situation.

Another weakness of some of the included studies is the short time period between the end of active treatment and the evaluation of outcomes. When records are taken on the day of debonding, periodontal measurements may be affected, often by gingival inflammation and swelling due to the difficulty in maintaining good oral hygiene during active orthodontic treatment. This was the case for four studies; no follow-up data was available to ascertain whether gingival recession developed in the long term after fixed appliances were removed. Steiner (1981) indicated that thinning of the gingiva during orthodontic therapy could create a long-term risk for recession. These studies could have been improved with records taken at several time points after fixed appliance treatment (Djeu et al., 2002; Allais and Melsen, 2003; Melsen and Allais, 2005; Kamak et al., 2015). Artun and Krogstad (1987) suggested an observation time of 3 years, since up to 3 years after treatment, the clinical crown height increased

significantly in patients with excessive proclination compared to patients with minimal change. However, after the first 3 years of follow up, the difference between the groups was not significant.

In addition, various confounding parameters were not always appropriately controlled for, such as the natural progression of the related phenomena with age, gender, occlusal relationships, oral hygiene measures and habits, gingival biotype, plaque and calculus accumulation, existing periodontal disease and previous periodontal treatment, smoking, force systems employed during orthodontic treatment, orthodontic treatment duration, wear of the incisal or occlusal aspects of teeth etc. In addition, the investigations did not cover the whole extent of the dentition although an association between orthodontic tooth movement and gingival recession has been mentioned in both the orthodontic and periodontic literatures, many of these studies are relevant to mandibular incisor teeth only, without taking into account the entire dentition. Only two studies (Renkema et al.(2013b); Renkema et al.(2013c) evaluated the recession development in all teeth. AlBandar and Kingman (1999) stated that the buccal surfaces of the central mandibular incisors and maxillary molars are the most frequently affected sites. Therefore, future studies should focus on the total dentition, and not be restricted to the anterior teeth.

Last, but not least, the included studies investigated labial gingival recession, without taking into consideration the lingual aspect of the incisors. Antonarakis et al. (2017) concluded that orthodontic or surgical proclination of lower incisors beyond a 10° limit increases the risk of inducing lingual gingival recessions. It was also stated that all previous studies have seemed to have focused on labial gingival recessions, overlooking the possibility of development of lingual gingival recessions.

6.3. Recommendations for future research

It has been suggested that well-designed and properly executed Randomized Control Trials provide the best evidence, with a decreased risk of bias, on the efficacy of health care interventions (Altman et al., 2001; Oxford Centre for Evidence-based Medicine, 2009). Since this might be regarded as unethical under certain situations, the random allocation of subjects, either to a group where they would receive orthodontic treatment or to a group of no intervention, it would be advisable to conduct at least well-controlled prospective non-randomized studies that were comparable to well-performed randomized studies in order to clarify the effect of orthodontic changes in the occurrence of gingival recession. The associated confounding factors include age, gender, occlusal relationships, oral hygiene measures and habits, gingival biotype, plaque and calculus accumulation, existing periodontal disease and previous periodontal treatment, smoking, force systems employed during orthodontic treatment, orthodontic treatment duration, wear of the incisal or occlusal aspects of teeth etc.

Unfortunately, no single study of those included in the present review had a prospective design with clinical examinations before, during, after and long term after treatment. Prospective designs would give more precise information regarding the interaction of orthodontic tooth inclination and the development of gingival recession. Particular importance should be placed on possible ways to control bias in measurement of outcomes. Finally, since special reference has been made to the fact that gingival recession development is a long-term process and does not necessarily appear immediately post orthodontic treatment (Juloski et al., 2017), future studies

should investigate the development of gingival recession in the long term by designing studies with long- term follow-up periods.

7. CONCLUSIONS

Some increases in gingival recession prevalence may be encountered after orthodontic treatment. Although clinical crown length increases during orthodontic treatment and retention, overall no statistically significant differences were noted between incisors having undergone proclination compared to the non-proclination group. More high quality studies are needed in order to further elucidate possible associations.

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APPENDICES

Appendix I. Systematic review protocol used for registration with the international prospective register of systematic reviews (PROSPERO).

Citation

Clinical crown height changes following treatment with fixed orthodontic appliances

Review question(s)

To investigate current data on clinical crown height changes following treatment with fixed orthodontic appliances and to critically evaluate the quality of available evidence.

Searches

Comprehensive electronic database searches will be undertaken without language restriction in the following databases:

MEDLINE via PubMed (http://www.ncbi.nlm.nih.gov/pubmed), Scopus (www.scopus.com), Web of Science™ Core Collection (http://apps.webofknowledge.com/), Arab World Research Source (http://0-web.a.ebscohost.com.amclb.iii.com) and ProQuest Dissertations and Theses Global database.

Efforts will be made to obtain conference proceedings and abstracts where possible. Authors will be contacted to identify unpublished or ongoing clinical trials and to clarify methodology and data as necessary. Reference lists of included studies will be screened for additional relevant research.

Types of study to be included

The trials to be included should be studies evaluating clinical crown height changes following treatment with fixed orthodontic appliances.

Condition or domain being studied

Periodontal condition in patients undergoing fixed orthodontic appliance treatment.

Participants/ population

Subjects of any age and gender undergoing fixed orthodontic appliance treatment.

Intervention(s), exposure(s)

Fixed orthodontic appliance treatment.

Comparator(s)/ control

Clinical crown height before treatment with fixed orthodontic appliances.

Outcome(s)

Primary outcomes

Clinical crown height measurements at specific teeth and at specific time points following treatment.

Secondary outcomes

Recession assessment at specific teeth and at specific time points following treatment.

Data extraction, (selection and coding)

All assessments including titles and/or abstract screening, full text evaluation, and extraction of data will be performed independently and in duplicate by two investigators (BS and SA). The investigators will not be blinded to the authors or the results of the research. Disagreements will be resolved by discussion and consultation with a third author where necessary (EGK).

Risk of bias (quality) assessment

Assessment of risk of bias will be performed independently and in duplicate by two investigators (BS and SA) using the using the ROBINS-I tool (Risk Of Bias In Non-randomised Studies of Interventions). Disagreements will be resolved by discussion and consultation with a third author where necessary (EGK).

Strategy for data synthesis

In situations where the retrieved data use different variables measuring the same concept on different scales with a high degree of correlation, the effects of the interventions are planned to be expressed as standardized values (i.e. the Standardized Mean Difference (SMD) together with the relevant 95% Confidence Interval (CI)), in order to enable quantitative synthesis. Where, in a particular comparison, the same variable is recorded, the intervention effect is planned to be expressed as the Weighted Mean Difference (WMD) together with the 95% CI. The random effects method for meta-analysis is to be used to combine data from studies that report similar measurements in appropriate statistical forms, since they are expected to differ across studies due to clinical diversity, in terms of participant and intervention characteristics. Heterogeneity will be assessed using both the Chi-squared test and the I-squared statistic. If an adequate number of trials are identified, we will carry out analyses for “small-study effects” and publication bias.

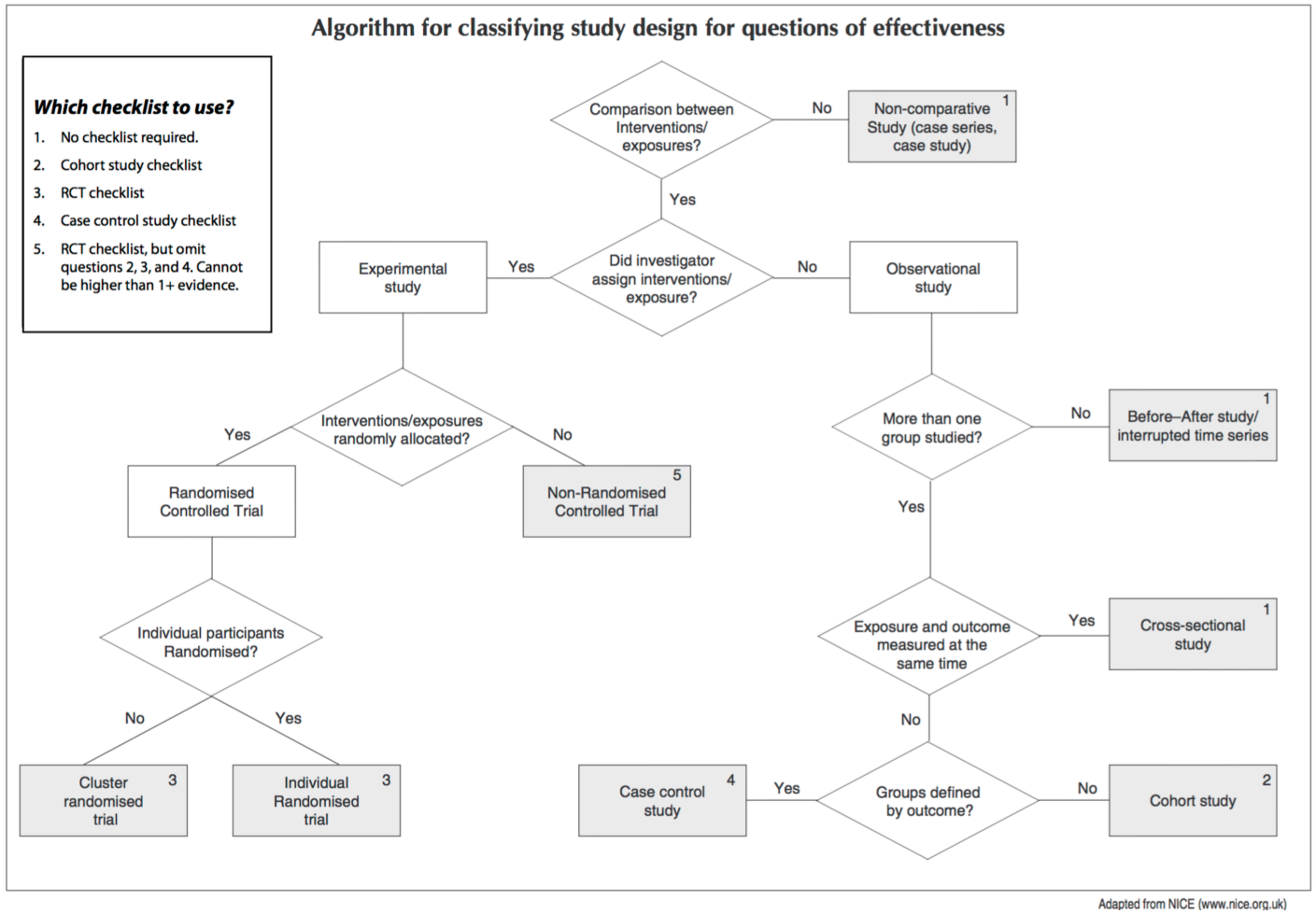
Analysis of subgroups or subsets

If the necessary data are available, subgroup analysis will be performed.

Dissemination plans

Peer-reviewed orthodontic journal

Appendix II. Scottish Intercollegiate Guidelines Network (SIGN) algorithm for classifying study design for questions of effectiveness.



Appendix III. Strategy for database search [until October 31st, 2017].

Database [2017 10 31]	Search strategy	Hits
General Sources		
PubMed	(recession*[tiab]OR gingiva*[tiab]OR periodont*[tiab]) AND ("fixed appliance"[tiab] OR orthodon*[tiab] OR "fixed orthodontic"[tiab] OR bracket*[tiab] OR multibracket[tiab])	**4419**
Cochrane Central Register of Controlled Trials	(recession*OR gingiva*OR periodont*) AND ("fixed appliance" OR orthodon* OR "fixed orthodontic" OR bracket* OR multibracket) in Title, Abstract, Keywords in Trials'	**382**
Cochrane Database of Systematic Reviews	orthodon* {Including Limited Related Terms}	**42**
Scopus	TITLE-ABS-KEY ((recession*ORgingiva*ORperiodont*)AND("fixed appliance"ORorthodon*OR"fixed orthodontic"ORbracket*ORmultibracket))AND(LIMIT-TO (SUBJAREA ,"DENT"))AND(LIMIT-TO (EXACTKEYWORD ,"Gingival Recession"))	**218**
Web of Science™	TITLE: ((recession* OR gingiva* OR periodont*) AND ("fixed appliance" OR orthodon* OR "fixed orthodontic" OR bracket* OR multibracket)) Refined by: RESEARCH AREAS: (DENTISTRY ORAL SURGERY MEDICINE) Timespan: All years. Search language=Auto	**705**
Regional sources		
Arab World Research Source	(recession*OR gingiva*OR periodont*) AND ("fixed appliance" OR orthodon* OR "fixed orthodontic" OR bracket* OR multibracket) Subject: orthodontics	**6**
Grey literature sources		
ClinicalTrials.gov	(orthodontic OR orthodontics) AND (recession OR recessions OR gingiva OR gingival)	**18**
ProQuest Dissertations and Theses Global	ti((recession*OR gingiva*OR periodont*) AND ("fixed appliance" OR orthodon* OR "fixed orthodontic" OR bracket* OR multibracket)) OR ab((recession*OR gingiva*OR periodont*) AND ("fixed appliance" OR orthodon* OR "fixed orthodontic" OR bracket* OR multibracket)) Full text included	

Appendix IV. Details of risk of bias assessment. [Domains examined: 1: Bias due to confounding 2: Bias in selection of participants, 3: Bias in classification of intervention, 4: Bias due to deviations from intended interventions, 5: Bias due to missing data, 6: Bias in measurement of outcomes, 7: Bias in selection of the reported result]

Study	Rating		Reasons for rating
Djeu et al. [2002] Before and after study	1	Serious	Important parameters (Age, Incisor relationship, gingival biotype, oral hygiene measures and habits, existing periodontal disease, etc.) were not controlled.
	2	Moderate	Did not have any exclusion criteria (they did not exclude any surgical cases, cleft lip and palate etc.) due to lack of complete records available.
	3	Low	It is mentioned that all the participants had received orthodontic treatment in both arches (type of brackets and type of extraction mentioned)
	4	Not applicable	Not applicable
	5	Not applicable	Not applicable
	6	Low	Examiner reliability was performed and none of the measurements showed any statistical difference between original and repeated values
	7	Moderate	The outcome measurements and analyses are consistent with an *a priori* plan.
Allais and Melsen [2003] Case-control study	1.	Serious	Important parameters (treatment duration, force systems, oral hygiene habits, existing periodontal disease, smoking, etc.) were not controlled.
	2.	Low	No reason to believe that the selection of the participants was biased.
	3.	Low	No statement about the details of the orthodontic treatment (type of appliance, treatment duration). However, it is clearly mentioned that all the participants had received orthodontic treatment.
	4.	Not applicable	Not applicable
	5	Not applicable	Not applicable
	6	Low	An external examiner, who was not an orthodontist and was not informed about the design of the study, was asked to measure the records (blinded about the aim and design of the study), after being calibrated by an orthodontist and periodontologist. The reproducibility of the measurements were assessed, the mean difference between first and second measurement 2qw 0.01mm for both slides and casts.
	7	Moderate	The outcome measurements and analyses are consistent with an *a priori* plan.

Appendix IV. Details of risk of bias assessment. [Domains examined: 1: Bias due to confounding 2: Bias in selection of participants, 3: Bias in classification of intervention, 4: Bias due to deviations from intended interventions, 5: Bias due to missing data, 6: Bias in measurement of outcomes, 7: Bias in selection of the reported result] [Continued]

Study	Rating		Reasons for rating
Melsen and Allais [2005] before and after study	1	Serious	Important parameters (Incisor relationship, treatment time, force systems, existing periodontal disease, smoking, etc.) were not controlled.
	2.	Low	No reason to believe that the selection of the participants was biased.
	3.	Low	No statement about the details of the orthodontic treatment (type of appliance, treatment duration). However, it is clearly mentioned that all the participants had received orthodontic treatment.
	4.	Not applicable	Not applicable
	5.	Not applicable	Not applicable
	6.	Low	An external examiner, who was not involved in orthodontic practice, was asked to measure the records (blinded about the aim and design of the study)
	7.	Moderate	The outcome measurements and analyses are consistent with an *a priori* plan.
Konikoff et al. [2007] before and after study	1.	Serious	Important parameters (Age, Gingival biotype, Incisor relationship, treatment time, force systems, oral hygiene measures, treatment, duration, existing periodontal disease, smoking, etc.) were not controlled.
	2.	Low	No reason to believe that the selection of the participants was biased.
	3.	Serious	No information regarding type of orthodontic treatment (subjects who had completed movement of maxillary central, lateral and canine). No information about appliance type.
	4.	Not applicable	Not applicable
	5.	Not applicable	Not applicable
	6.	Serious	Because of no method error and no intra and inter examiner reliability. Also no blinding.
	7.	Moderate	The outcome measurements and analyses are consistent with an *a priori* plan.

Appendix IV. Details of risk of bias assessment. [Domains examined: 1: Bias due to confounding 2: Bias in selection of participants, 3: Bias in classification of intervention, 4: Bias due to deviations from intended interventions, 5: Bias due to missing data, 6: Bias in measurement of outcomes, 7: Bias in selection of the reported result] [Continued]

Study	Rating		Reasons for rating
Renkema et al. [2013a] Before and after study	1.	Serious	Important parameters (gingival biotype, incisal relationships, force systems, oral hygiene habits, existing periodontal disease, smoking, etc.) were not controlled.
	2.	Low	No reason to believe that the selection of the participants was biased.
	3.	Low	No statement about the details of the orthodontic treatment (type of appliance). However, it is clearly mentioned that all the participants had received orthodontic treatment in both dental arches.
	4.	Not applicable	Not applicable
	5.	Not applicable	Not applicable
	6.	Serious	Although assessment of error of measurements of clinical crown heights showed a coefficient of reliability ranging between 0.973 and 0.995. They reported one statistically significant difference of the clinical crown height measurements between both observers at pre-tx tooth 42. The reliability of the measurements of incisal inclination performed by 2 observers ranged between 0.985-0.988.
	7.	Moderate	The outcome measurements and analyses are consistent with an *a priori* plan.
Renkema et al. [2013b] Case-Control study	1	Serious	Important parameters (gingival biotype, incisal relationships, force systems, plaque accumulations, oral hygiene habits, existing periodontal disease, bleeding on probing of gingival pockets, smoking, etc.) were not controlled.
	2	Low	No reason to believe that the selection of the participants was biased.
	3	Low	It is clearly mentioned that all the cases had received fixed orthodontic treatment in both dental arches with fixed lingual bonded retainers.
	4	Not applicable	Not applicable
	5	Not applicable	Not applicable
	6	Serious	Observers were not blinded to the origin of the dental cast (i.e. whether they scored the presence or absence of recession in cases or controls)
	7	Moderate	The outcome measurements and analyses are consistent with an *a priori* plan.

Appendix IV. Details of risk of bias assessment. [Domains examined: 1: Bias due to confounding 2: Bias in selection of participants, 3: Bias in classification of intervention, 4: Bias due to deviations from intended interventions, 5: Bias due to missing data, 6: Bias in measurement of outcomes, 7: Bias in selection of the reported result] [Continued]

Study	Rating		Reasons for rating
Renkema et al. [2013c] before and after study	1	Serious	Important parameters (gingival biotype, incisal relationships, force systems, plaque accumulations, oral hygiene habits, existing periodontal disease, smoking, etc.) were not controlled.
	2	Low	No reason to believe that the selection of the participants was biased.
	3	Low	It is clearly mentioned that all the cases had received fixed orthodontic treatment in both dental arches with fixed lingual bonded retainers.
	4	Not applicable	Not applicable
	5	Not applicable	Not applicable
	6	Low	Examiner reliability was performed and non of the measurements showed any statistical difference between original and repeated values
	7	Moderate	The outcome measurements and analyses are consistent with an *a priori* plan.
Renkema et al., [2015] before and after study	1.	Serious	Important parameters (gingival biotype, incisal relationships, force systems, oral hygiene habits, existing periodontal disease, smoking, etc.) were not controlled.
	2.	Low	No reason to believe that the selection of the participants was biased.
	3.	Low	No statement about the details of the orthodontic treatment (type of appliance). However, it is clearly mentioned that all the participants had received orthodontic treatment in both dental arches.
	4.	Not applicable	Not applicable
	5.	Not applicable	Not applicable
	6.	Low	Assessment of error of measurements of clinical crown heights showed coefficient of reliability greater than 0.970.the reliability of the measurements of incisal inclination performed by 2 observers was greater than 0.98.
	7.	Moderate	The outcome measurements and analyses are consistent with an *a priori* plan.

Appendix IV. Details of risk of bias assessment. [Domains examined: 1: Bias due to confounding 2: Bias in selection of participants, 3: Bias in classification of intervention, 4: Bias due to deviations from intended interventions, 5: Bias due to missing data, 6: Bias in measurement of outcomes, 7: Bias in selection of the reported result] [Continued]

Study	Rating		Reasons for rating
Kamak et al. [2015] before and after study	1.	Serious	Important parameters (Incisor relationship, gingival biotype, oral hygiene measures, force systems, parafunctional habits, existing periodontal disease, etc.) were not controlled.
	2.	Low	No reason to believe that the selection of the participants was biased.
	3.	Low	It is clearly mentioned that all the participants had received orthodontic treatment (upper and lower straight-wire appliances for at least 12 months)
	4.	Not applicable	Not applicable
	5.	Not applicable	Not applicable
	6.	Low	All measurements on cephalograms and models were made by the same investigator. 40 study records were randomly selected and re-measured. The coefficient foal l measurements was between 0.93 and 0.98 and was considered acceptable.
	7.	Moderate	The outcome measurements and analyses are consistent with an *a priori* plan.
Juloski et al. [2017] Before and after study Case-control study	1	Serious	Important parameters (gingival biotype, oral hygiene measures and habits, existing periodontal disease, etc.) were not controlled.
	2.	Low	No reason to believe that the selection of the participants was biased.
	3.	Low	No statement about the details of the orthodontic treatment (type of appliance). However, it is clearly mentioned that all the participants had received orthodontic treatment with/without fixed lingual retainer.
	4.	Not applicable	Not applicable
	5.	Not applicable	Not applicable
	6.	Low	Examiner reliability was performed and all measurements showed very good agreement between measurements.
	7.	Moderate	The outcome measurements and analyses are consistent with an *a priori* plan.