วิทยาสารออนไลน์สมาคมทันตแพทย์จัดฟันแห่งประเทศไทย
The Online Journal of Thai Association of Orthodontists
ว ออนไลน์ ทันต จัดฟัน ปีที่ 5 2558 OJ Thai Assoc Orthod Vol 5 2015

สารบัญ

รายงานผู้ป่วย

บทความปริทัศน์

บทสัมภาษณ์พิเศษ

CONTENTS

Case Report

Review Article

Special Interview

วิทยาสารออนไลน์สมาคมทันตแพทย์จัดฟันแห่งประเทศไทย
The Online Journal of Thai Association of Orthodontists

www.thaiortho.org/journal

จดหมายจากสาราณียากร

วิทยาสารสมาคมทันตแพทย์จัดฟันแห่งประเทศไทย (Journal of the Thai Association of Orthodontists) ได้เริ่มเผยแพร่ตั้งแต่ พ.ศ. 2545 เพื่อเผยแพร่ความรู้ความก้าวหน้าทางวิชาการจัดฟัน วิทยาศาสตร์พื้นฐานที่น่าสนใจ ตลอดจน ส่งเสริมการศึกษาและการวิจัยด้านทันตกรรมจัดฟัน โดยตีพิมพ์เป็นรูปเล่มปีละหนึ่งฉบับ จนถึงปี พ.ศ. 2553 รวมแล้ว 9 ฉบับ (Volume 1 - 9) จากนั้นในปี พ.ศ. 2554 ได้เปลี่ยนชื่อเป็น วิทยาสารออนไลน์สมาคมทันตแพทย์จัดฟันแห่งประเทศไทย (The Online Journal of the Thai Association of Orthodontists) และเผยแพร่ทางเว็บไซต์ของสมาคมทันตแพทย์จัดฟันแห่งประเทศไทย

วิทยาสารออนไลน์สมาคมทันตแพทย์จัดฟันแห่งประเทศไทย ได้เผยแพร่มาแล้ว 4 ฉบับ (Volume 1 - 4) ในฉบับปัจจุบันนี้เป็นฉบับที่ 5 ซึ่งเผยแพร่ทั้งทางออนไลน์ และเริ่มนำวิทยาสารออนไลน์มาตีพิมพ์รวมกับหนังสือ Braces News เป็นเล่มเดียวกันสองปก เพื่อเพิ่มช่องทางให้สมาชิกเข้าถึงวิทยาสารได้มากขึ้น และเพื่อเป็นการเริ่มต้นการเผยแพร่วิทยาสารเป็นปีละ 2 ฉบับ ให้ตรงกับงานประชุมวิชาการของสมาคมฯ

ข้าพเจ้าขอเชิญชวนให้สมาชิกส่งบทความวิชาการต่าง ๆ มาเผยแพร่ ทั้งงานวิจัย รายงานผู้ป่วย บทความปริทัศน์ (review article) หรือคลินิกพิจารณ์ (clinical forum) โดยส่งมาที่ mail2mui@gmail.com หรือโทร. 08 9114 1374 และขอเชิญเข้าเยี่ยมชมเว็บไซต์ของสมาคมฯ ในส่วนวิทยาสาร (www.thaiortho.org/journal) ที่ได้รับการปรับปรุงเพื่อรวบรวมวิทยาสารทั้งหมดตั้งแต่ฉบับแรกจนปัจจุบัน เพิ่มช่องทางการสืบค้นบทความด้วยคำสำคัญ (keyword) และสามารถ download บทความที่ท่านต้องการได้ หากท่านมีข้อแนะนำในการปรับปรุงวิทยาสาร หรือเว็บไซต์ กรุณาแจ้งให้ข้าพเจ้าหรือกองบรรณาธิการทราบ จะเป็นประโยชน์อย่างยิ่ง

เรืองรัตน์ โกมลภิส
สาราณียากร
9 พฤศจิกายน 2558

หมายเหตุ : กรุณาอ่านรายละเอียดเพิ่มเติม เช่น คำแนะนำการเขียนบทความได้ที่ www.thaiortho.org/journal/rv2/home.php

รายงานผู้ป่วย
Case Report

A Different Approach to Class II Skeletal Correction and Extraction Treatment Using the Invisalign System :A Case Report

Eugene Chan*

Abstract

Traditional extraction therapies in the treatment of dental Class II cases involve either the extraction of upper and lower premolars, or a camouflage type treatment with the extraction of the upper first premolars only. Skeletal Class II correction in growing patients with functional appliances has been well documented. The advancement of the mandible in the treatment and/or prevention of obstructive sleep apnoea has often been prescribed as the treatment of choice. The combination of such therapies in conjunction with the extraction of lower premolar teeth using the Invisalign system is however less prevailing. This paper reports an ideal case selected for such treatment modality and widens the clinician's perspective on using these appliances.

Keywords: *Class II correction, Invisalign extraction, functional appliance*

Introduction

The prevalence of Class II dental malocclusions in growing children varies between 33% to 38%,[1-3] whereas skeletal Class II cases may extend to 70% of the all children of that age group between 6 and 14 years old.[4] Documented treatment modalities of Class II dental malocclusions include extraction or non-extraction therapies with functional appliances (fixed or removable),[5-7] head gear,[8, 9] the placement of temporary anchorage devices (TADs),[10] dental elastics,[11] and other distalizing devices.[12, 13] Often a combination of any of the above may be required.[14, 15]

Obstructive sleep apnoea (OSA) occurs when a person's airway becomes partially or completely blocked several times during sleep. The result of this interrupted breathing pattern is severely fragmented sleep. This leads to constant awakening and patients do not get sufficient, good quality sleep, resulting in sleepiness and/or fatigue.[16]

Population-based epidemiologic studies have uncovered the high prevalence of undiagnosed obstructive sleep apnoea, and have consistently

* Private Practice in Zetland, Australia

found that even mild obstructive sleep apnoea is associated with significant morbidity. Studies have suggested that the obstructive sleep apnoea may be an important risk factor for stroke[17] and there has been evidence of genetic predisposition.[18] One of the common non-surgical approaches for the treatment of obstructive sleep apnoea is a mandibular advancement oral device.[19] The repositioning of the mandible forwards also allows the tongue to be in a more anterior position, hence preventing it from obstructing the airways.[20] It is noted that such oral devices are very useful in mild to moderate cases.

The use of a functional appliance to improve the dento-skeletal appearance of growing patients has been previously documented.[21] However the use of such appliance in a Class I dental malocclusion is less noted. The notion of bringing the mandible forward, into a reverse overjet, and subsequently the extraction of lower premolars to retract the anterior teeth back into an ideal overjet and overbite is daunting as treatment success relies heavily on favourable patient growth, appliance design as well as patient compliance.

Invisalign™ is a series of clear, transparent, custom made polymer-based aligners used to gradually align teeth in stages. Aligners are worn full time between 20 to 22 hours per day and a new set changed every 2 weeks. Since its global launch in 1998, it has gained much popularity.[22, 23] However many clinicians are still not well versed in its capabilities and have not embarked on using this appliance to treat complex cases. Invisalign Teen® is a newer treatment option with features that allows under-erupted permanent teeth to be incorporated into the system. There is also a blue compliance indicator incorporated into the aligners that reacts with the patient's salivary enzymes, which denatures the encapsulated protein in the indicator and fades to a lighter blue tinge after the prescribed hours of full-time wear; usually 300 hours within the 2 week period.

A 2-stage treatment was prescribed in this case to allow the improvement of the patient's dento-skeletal appearance using a stage I functional appliance, followed by full comprehensive orthodontic treatment using the Invisalign Teen® appliance with the extraction of teeth 34 and 44.

This paper reports of a case where the functional appliance was prescribed for reducing the risks of future development of OSA, and effective dento-skeletal changes, followed by full comprehensive orthodontics with extraction therapy using Invisalign Teen®.

Diagnosis

A male adolescent, aged 12 years 2 months attended our office with the chief concern of lower dental crowding (Figs. 1-3). Medical history revealed mild episodes nocturnal snoring and the patient's

Fig. 1 Pre-treatment photographs

Fig. 2 Pre-treatment Cephalometric radiograph

Fig. 3 Pre-treatment panoramic radiograph

mother has been diagnosed of obstructive sleep apnoea (OSA) and is currently on a sleep CAP machine. Although the patient's recent sleep test did not reveal any hypopnoea, nor reduced oxygen saturation, his parents were still concerned that his retrusive mandible may be a risk for OSA in the future.

He has a mesocephalic ovoid head form, with a convex overall profile with a retrusive mandible. His soft tissue characteristics included a slightly open naso labial angle, thin lips, and a deep labial mental fold. Skeletally, he had a normal maxilla with a retrusive mandible (Table I). Clinically he has a Skeletal Class II, dental Class I malocclusion with a horizontal direction of growth. There are moderate degrees of lower dental crowding and the lower right second molar appears slightly hypoplastic.

Measurements	Initial 17/02/2012	Final 22/04/2014
SNA angle (dg)	86.4	86.5
SNB angle (dg)	79.1	83
ANB angle (dg)	7.3	3.6
SN-MP angle (dg)	31.4	25.7
IMPA angle (dg)	108.3	100.3
FMA angle (dg)	19.5	21.3
FMIA angle (dg)	54.3	60.2
Mandibular 1 to APo (mm)	4.9	2
Maxillary 1 to NA (mm)	2.9	1
Interincisal angle (dg)	116.7	132.9
Wits (mm)	2	-1.7

Table I

Treatment alternatives

Several treatment alternatives were presented to the patient and his parents at the consultation appointment. The first option was to commence non-extraction full comprehensive orthodontic treatment with either braces or Invisalign Teen®, re-evaluate after preliminary alignment, followed by the likely extraction of four second premolar teeth. The second option was to commence Stage I functional appliance with twin blocks, followed by the likely extraction of either 2 lower first premolars or a lower incisor, and Stage II braces or Invisalign Teen®.

It was explained to the patient and his parents why a more aesthetic outcome could be expected with the functional appliance treatment therapy. However, they were warned that this treatment plan was not absolute in the prevention of any OSA in the future.

After all the pros and cons for both the treatment options were discussed, it was agreed upon to proceed with the second treatment option. As the patient plays the saxophone regularly, they would prefer a more aesthetic, removable appliance. Functional appliance with twin blocks, followed by Invisalign Teen® was the treatment of choice.

Treatment objectives

The treatment objectives included the advancement of the mandible through functional appliance therapy, improving on his dento-facial profile, correcting the retrusive chin, relieving the lower anterior crowding while maintaining an ideal overjet, and achieving a full unit Class III molar and Class I canine dental relationship.

Treatment plans

Full comprehensive treatment plan involved 2 stages. Stage I was a prescription of full-time wear of Clark Twin Blocks for a period of 6 to 9 months. A transverse expansion screw was incorporated into the upper component of the twin block, there was no lower incisor capping in this design. Once a permanent forward position of the mandible was achieved, selective posterior trimming of the appliance was performed to allow the closure of the posterior open bite. We anticipated the patient to go into a reverse overjet at the completion of the twin block therapy. The molars will be in a Class III dental relationship.

Stage II treatment involved the extraction of teeth 34 and 44 followed by Invisalign Teen® treatment. Class II and/or Class III elastics was required to control anchorage during alignment and space closure.

At the completion of treatment, fixed and removable retainers were designed and prescribed.

Treatment progress

The twin blocks were issued with instructions of full time wear except for eating, brushing and flossing, during swimming and contact sports. The patient was instructed to commence turning transverse expansion screw once every 5 days at the second month into active wear. At the fourth month, a stable prognathic sagittal position was obtained. The posterior bite block of the upper twin block was trimmed down approximately 2 mm to allow closure of the posterior open bite. After 7 months of good compliant wear and positive biological response, completion of stage I treatment was achieved (Fig. 4).

Fig. 4 Post functional appliance photographs

Intra-oral scans (iTero, Cadent) were performed to commence Invisalign Teen® treatment. Teeth 34 and 44 were extracted by his dentist and he suffered a short bout of dry socket; healing was inconsequential thereafter. Due to the 'bowing' effect of the aligners during space closure, compensatory dental movements were incorporated into the ClinCheck treatment setup. This included increased mesial root tip of the teeth distal to the extraction sites, and increased distal root tip of the teeth mesial to the extraction sites (Fig. 5).

Fig. 5a ClinCheck Plans showing the reverse overjet after the completion of functional appliance therapy and lower extractions.

Fig. 5b ClinCheck Plans showing the attachment designs and compensatory movements.

The sequential aligners were issued uneventfully. There were 34 aligners planned in the first stage of Invisalign Teen® treatment. Attachments prescribed were placed at the seventh week of aligner wear. Positive overjet and overbite was achieved after aligner 23 (Fig. 6), thereafter the patient commenced night time wear of Class II elastics (Chucks ¼" 3.5 oz elastics, 3 M) to maintain anchorage.

Fig. 6 Intra oral photographs showing the correction of overjet at aligner 23.

Fig. 7a Invisalign first refinement photographs

Fig. 7b Refinement OPG

As the patient was going away to boarding school for 6 months and would like to have the new refinement aligners arrive before he goes away, intra-oral scans were done for an early refinement after aligner 31 (Fig. 7).

The new aligners (total of 11) were issued with new attachment designs (Fig. 8). Triangular posterior

Fig. 8 First refinement ClinCheck Plans showing attachment designs and button cut outs for posterior elastic wear.

elastics were prescribed to improve the posterior occlusion (Chucks ¼" 3.5 oz elastics, 3 M). Treatment completed with the removal of the attachments and buttons. Upper and lower fixed lingual retainer wires were placed. Upper and lower night time removable retainers (Raintree Essix C+) were also prescribed.

Fig. 9 Post treatment photographs

Fig. 10 Post treatment cephalometric radiograph

Fig. 11 Post treatment panoramic radiograph

Post treatment results completed the occlusion is a full unit Class III molar and Class I canine dental relationship (Figs. 9 -11).

Treatment results

The post-treatment records demonstrated that facial aesthetics improved from stage I to stage II and also to completion (Fig. 12). The mandible appeared less retrusive and the patient and parents were pleased with his appearance. The upper and lower dental midlines were coincident with each other and also with the midsagittal plane. The molars were in full unit Class III, the canines in Class I dental relationships with normal overjet and overbite. The final occlusion had good interdigitation and canine guidance. Root parallelism is satisfactory.

Fig. 12 Profile changes

His oral hygiene was well maintained throughout his orthodontic treatment.

Cephalometrically, SNA remained the same while the SNB angle increased from 79.1° to 83.0°, with the ANB angle reduced from 7.3° to 3.6°. The SN-MP angle decreased from 31.4 to 25.7 and the IMPA reduced from 108.3° to 100.3°. The Interincisal angle increased from 116.7 ° to 132.9°. The absolute maxillary length increased from 91.6mm to 100.8 mm while the mandibular length increased from 108.7 mm to 125.7 mm (Table I).

The overall superimposition on SN demonstrated that the patient grew favourably downwards and forwards throughout treatment (Fig. 13). The Y axis and upper and lower facial heights remained relatively

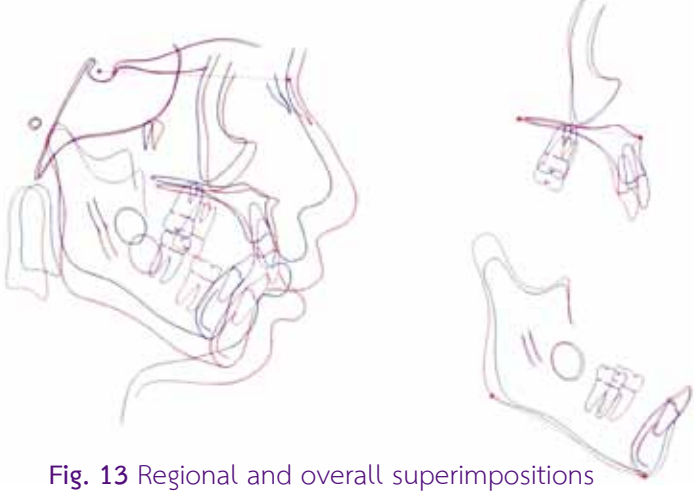


Fig. 13 Regional and overall superimpositions

unchanged, however the SN-MP angle reduced rather significantly from 31.4° to 25.7°. This anti-clockwise rotation of the mandible made the patient's mandible less retrusive, decreases the depth of the labial mental fold and the eversion of the lower lip.

The upper and lower lips came forwards with the lower lip increasing almost double the distance of the upper lip. This extent of increase was replicated in the increase of the maxilla and mandibular length as well.

Maxillary regional superimposition showed restriction of dental movements and remained stable. The upper molar erupted from the maxillary plane likely due to the anti-clockwise rotation of the mandible and posterior elastic wear during the Invisalign Teen® treatment. Mandibular regional superimposition showed a retraction of the lower incisors as crowding was relieved backwards; the IMPA reduced from 108.3° to 100.3°. The molars also protracted forwards and extruded slightly. The extrusion of the lower molars was likely due to the over-eruption at the end of the twin block treatment when the upper posterior bite blocks were trimmed down. This further encouraged the increase of the lower facial height.

The post-treatment radiographs showed no evidence of root resorption or any other pathology. The third molars are present and still unerupted. We discussed further with his dentist and parents about the prognosis of the hypoplastic lower right 2nd molar and suggestions included its removal to allow the lower right 3rd molar to erupt into its position.

Upper and lower fixed lingual retainers were placed with the patient also wearing night time removable vacuum formed retainers as well.

Active functional appliance treatment was 7 months. Active treatment with Invisalign Teen® was 17 months. Total active orthodontic treatment was 24 months.

Discussion

The patient commenced treatment at 12 years and 2 months old. Based on the cervical vertebrae maturation index, the C3 and C4 of the lateral cephalogram demonstrated skeletal maturity of CS3. This indicated that the patient was going to hit his growth spurt rather immediately.[24] The commencement of functional treatment with twin blocks requires the favourable growth of the child and timing of treatment is paramount.

Routine functional appliance therapy could range between 6-12 months. The success of treatment is rather multifactorial. It relies heavily on the timing of the treatment (in concurrent with the patient's growth spurt), appliance design, favourable growth direction, and patient compliance. Although there was no imminent posterior crossbites, the patient was instructed to commence turning transverse expansion screw once every 5 days after the second month into active wear. As the mandible comes forwards, the transverse discrepancy between the upper and the lower arch form will become eminent. The final planned occlusion is a full unit Class III molar and a Class I canine dental relationship. Therefore although there was no evident posterior crossbite initially, it was essential to have the upper and lower arch forms articulate into a correct transverse relationship bearing the final occlusion in mind.

At the fourth month, a stable prognathic sagittal position was obtained. This was confirmed by relaxing

the patient's mandible and guiding its condyles back into their most posterior, superior position. A postured prognathic mandibular position is due to muscular adaptation and is not a stable, reproducible position, often leading to a 'false positive' correction. It is essential to have the corrected position maintained for a few months, and allow the settling of the occlusion, obtain a good interdigitation to maintain the corrected mandibular position. The result was achieved with judicious trimming of the bite plane of the upper component of the twin blocks to allow the closure of the posterior open bite often seen at the completion of sagittal correction in functional appliance therapy. The twin block appliance was worn with excellent compliance and stage I treatment was completed in 7 months.

The treatment prescribed in this paper is not unlike the 'surgical first' technique. The 'surgery first' technique in combined orthodontic-orthognathic surgery cases was first described in 2009.[25] The premise of this technique was to perform the jaw movements via surgery prior to the orthodontic treatment phase. The alignment, space closure and decompensation movements were all performed after the surgical procedure.[26] It has been noted in these cases that the efficiency of treatment improved. The correction of the skeletal imbalance was first achieved, the complexity of the treatment decreases with the elimination of soft tissue imbalances that might hinder treatment.[27]

Using functional appliance therapy to correct the skeletal discrepancy, eliminating any soft tissue imbalances may pave the way to more efficient orthodontic treatment. The ability to achieve a stable mandibular position post twin block treatment gives us the assurance that extraction on the lower jaw only in a skeletal Class II case will complete the case in an ideal overjet and overbite. The increase in vertical dimensions through the patient's growth and functional therapy has also allowed the bite opening to occur, thus further enhancing the efficiency of the orthodontic treatment.

The ANB angle reduced quite remarkably during treatment underlying the effects of the functional appliance therapy. The optimal timing of treatment and favourable vertical growth of the patient also allowed the enhancement of his natural downward and forwards growth of both the maxilla and the mandible. The forward rotation of the mandible during treatment is observed with the decrease in the SN-MP angle. The IMPA reduced despite the relieving of the lower dental crowding as the lower 1st premolar teeth were extracted.

Mandibular advancement splints traditionally used to treat OSA in adult patients increases the pharyngeal airway space and prevents the physical blockage.[28] However there have also been frequent reports of permanent dental changes of the occlusion after long term use.[29-31] Enhancing the effects of a functional appliance such as a twin block in a growing child, we have capitalised on this side-effect in this particular case. The permanent advancement of the mandible in this case has ensured patency of the airways in the prevention of the OSA as set off from the beginning in our treatment objectives.

Full comprehensive orthodontic treatment with Invisalign has been previously criticized.[32] Its inability to control bodily movements, especially so in extraction cases, have made it an inferior treatment of choice. However with careful understanding and thorough planning, superior results can be obtained. As Invisalign

is removable appliance, the degree of 'play' between the appliance and the dentition dictates the true tracking of the appliance. Compensatory movements need to be planned within the ClinCheck set up to allow these intended movements to occur (Fig. 5b). In this extraction case, the teeth distal to the extraction sites had 8 degrees more mesial root tip placed on them. The lower canines (tooth immediately mesial to the extraction site) had 8 degrees more distal root tip placed on them.

During the lower space closure, the lower curve of Spee inevitably deepened. Compensatory movements with the further intrusion of the lower incisors during the first ClinCheck set up should have been planned. This has resulted in an anterior interference and posterior open bite at the end of the first lot of aligners. During the refinement stage, posterior triangular box elastics had to be worn to allow the occlusion to settle into a normalised bite. The recent launch of Invisalign G6 has made attempt to counter this said effect for extraction cases with modified staging patterns as well as new optimised attachment designs.

Conclusions

Effective and efficient treatment was rendered for this growing patient using an alternative treatment technique with a great treatment outcome.

A growing child with a family history of OSA, skeletal Class II pattern but with a Class I dental occlusion required orthodontic treatment. The mandibular advancement with the twin block functional appliance brought his lower jaw forwards, improving his dento facial profile. His optimal growth pattern allowed the improvement of his vertical facial ratios. With the correction of his Class II skeletal pattern, we also maintained a patent pharyngeal airway in order to reduce the chances of developing OSA in the future.

The reverse overjet was corrected by the extraction of 2 lower 1st premolar teeth. This effectively corrected the lower dental crowding while maintaining a good lower incisor angulation with the mandibular plane as well as its position in space. A therapeutic Class III dental occlusion with a full unit Class III molar and Class I canine relationship achieved was stable and functional.

The upper and lower fixed retainers were also prescribed with night time removable vacuum formed retainers to maintain treatment stability. The patient will be monitored periodically post-treatment for repairs, relapse and erupting wisdom teeth. The patient was referred back to his regular dentist for routine checks.

References

1. Ast DB, Carlos JP, Cons NC. The prevalence and characteristics of malocclusion among senior high school students in upstate New York. ***Am J Orthod*** 1965;51: 437-45.
2. Massler M, Frankel JM. Prevalence of malocclusion in children aged 14 to 18 years. ***Am J Orthod*** 1951:37: 751-68.
3. Thilander B, Myrberg N. The prevalence of malocclusion in Swedish school children. ***Scand J Dent Res*** 1973;81: 12-20.
4. de Almeida MR, Pereira ALP, de Almeida, de Almeida-Pedrin RR, da Silva Filho OG. Prevalence of malocclusion in children aged 7 to 12 years. ***Dent Press J Orthod*** 2011;16. no.4 July/Aug.
5. Pancherz H, Malmgren O, Hagg U, Omblus J, Hansen K. Class II correction in Herbst and Bass therapy. ***Eur J Orthod*** 1989;11): 17-30.
6. Clark W J. The Twin Block traction technique. ***Eur J Orthod*** 1982;4:129-38

7. Clark W J. The twin block technique. A functional orthopaedic appliance system. ***Am J Orthod Dentofacial Orthop*** 1988;93:1-18.
8. Pfeiffer JP, Grobety D: The Class II malocclusion: differential diagnosis and clinical application of activators, extraoral traction, and fixed appliances. ***Am J Orthod*** 1975;68: 499-544.
9. Pfeiffer JP, Grobety D: Simultaneous use of cervical appliance and activator: an orthodontic approach to fixed appliance therapy. ***Am J Orthod*** 1972;61: 353-73.
10. Ohashi E, Pecho OE, Moron M, Lagravere MO. Implant vs screw loading protocols in orthodontics. ***Angle Orthod*** 2006;76: 721-7.
11. Class II correction in patients treated with class II elastics and with fixed functional appliances: a comparative study. ***Am J Orthod Dentofacial Orthop*** 2000;118:142-9.
12. Kanomi R. Mini-implant for orthodontic anchorage. ***J Clin Orthod*** 1997;31:763-7.
13. Hamilton CF; Saltaji H; Preston CB; Flores-Mir C; Tabbaa S. Adolescent patients' experience with the Carriere distalizer appliance. ***Eur J of Paed Dent*** 2013;14:219-24.
14. McNamara JA. Skeletal and dental changes following functional regulator therapy on Class II patients. ***Am J Orthod*** 1985;88:91-110.
15. Yao CC, Lai HH, Chang ZC, Chen I, Chen Y. A Comparison of treatment outcomes between skeletal anchorage and extraoral anchorage in adults with maxillary dentoalveolar protrusion. ***Am J Orthod Dentofac Orthop*** 2008;134: 615-24.
16. Obstructive sleep apnea syndrome. Mannarino MR, Di Filippo F, Pirro M. ***Eur J of Int Med*** 2012;23:586-93.
17. Yagg HKi, Concato J, Kernan WN, Lichtman JH, Brass LM, Mohsenin V. Obstructive sleep apnea as a risk factor for stroke and death. ***N Eng J of Med*** 2005;353:2034-41.
18. Neil JD, Marion L, Rajat M. Is the sleep apnoea/hypopnoea syndrome inherited? ***Thorax*** 1993;48:719-21.
19. Ferguson KA, Love LL, Ryan CF. Effect of mandibular and tongue protrusion on upper airway size during wakefulness. ***Am J Respir Crit Care Med*** 1997;155:1748-54.
20. Menn SJ, Loube DI, Morgan TD, Mitler MM, Berger JS, Erman MK. The mandibular repositioning device: role in the treatment of obstructive sleep apnea. ***Sleep*** 1996;19: 794-800.
21. Caldwell S, Cook P. Predicting the outcome of twin block functional appliance treatment: a prospective study. ***Eur J Orthod*** 1999;21:533-9.
22. Turpin DL. Clinical trials needed to answer questions about Invisalign. ***Am J Orthod Dentofacial Orthop*** 2005;127:157-8.
23. Kuncio D, Maganzini A, Shelton C, Freeman K. Invisalign and traditional orthodontic treatment postretention outcomes using the American Board of Orthodontics objective grading system. ***Angle Orthod*** 2007;77:864-9.
24. Baccetti T, Franchi L, McNamara JA Jr. The cervical vertebral maturation (CVM) method for the assessment of optimal treatment timing in dentofacial orthopedics. ***Semin Orthod*** 2005;11:119-29.
25. Nagasaka H, Sugawara J, Kawamura H, et al: 'Surgery first' skeletal Class III correction using the Skeletal Anchorage System. ***J Clin Orthod*** 2009;43:97-105.
26. Liou EJ, Chen PH, Wang YC, et al: Surgery-first accelerated orthognathic surgery: Orthodontic guidelines and setup for model surgery. ***J Oral Maxillofac Surg*** 2011;69:781-5.
27. Villegas C, Uribe F, Sugawara J, et al: Expedited correction of significant dentofacial asymmetry using a 'surgery first' approach. ***J Clin Orthod*** 2010;44:97-103.
28. Schendel SA, Broujerdi JA, Jacobson RL. Three-dimensional upper-airway changes with maxillomandibular advancement for obstructive sleep apnea treatment. ***Am J Orthod Dentofacial Orthop*** 2014;146:385-93.
29. Marklund M. Predictors of long-term orthodontic side effects from mandibular advancement devices in patients with snoring and obstructive sleep apnea. ***Am J Orthod Dentofacial Orthop*** 2006;129:214-21.
30. Almeida FR, Lowe AA, Sung JO, Tsuiki S, Otsuka R. Long-term sequellae of oral appliance therapy in obstructive sleep apnea patients: part 1. Cephalometric analysis. ***Am J Orthod Dentofacial Orthop*** 2006;129:195-204.
31. Almeida FR, Lowe AA, Otsuka R, Fastlicht S, Farbood M, Tsuiki S. Long-term sequellae of oral appliance therapy in obstructive sleep apnea patients: part 2. Study-model analysis. ***Am J Orthod Dentofacial Orthop*** 2006;129: 205-13.
32. Djeu G, Shelton C, Maganzini A. Outcome assessment of Invisalign and traditional orthodontic treatment compared with the American Board of Orthodontics objective grading system. ***Am J Orthod Dentofacial Orthop*** 2005;128: 292-8.

บทความปริทัศน์
Review Article

การใช้เลเซอร์เพิ่มความเร็วในการเคลื่อนฟัน

ทัศนีย์ วังศรีมงคล* มนเทียร มโนสุดประสิทธิ์* พูนศักดิ์ ภิเศก* ปฐมพร จงจรวยสกุล**

บทคัดย่อ

ในช่วงไม่กี่ปีที่ผ่านมามีการคิดค้นวิธีต่างๆ ที่ช่วยเร่งความเร็วในการเคลื่อนฟันทางทันตกรรมจัดฟัน การฉายเลเซอร์ระดับต่ำเป็นวิธีหนึ่งที่ช่วยกระตุ้นให้มีการเปลี่ยนแปลงทางสรีรชีววิทยาของการปรับรูปกระดูกเบ้าฟัน วิธีนี้เป็นที่สนใจเนื่องจากประสบความสำเร็จในการทดลองในมนุษย์ การฉายเลเซอร์ระดับต่ำเป็นเทคนิคที่ไม่ก่อให้เกิดความเจ็บปวด ทำได้ง่ายด้วยเครื่องมือราคาไม่แพง และยังช่วยลดความเจ็บปวดหลังจากปรับเครื่องมือจัดฟันอีกด้วย มีหลายการศึกษาที่ทำการทดลองในสัตว์และมนุษย์เพื่ออธิบายกลไกการทำงานของเลเซอร์ในการเร่งความเร็วการเคลื่อนที่ของฟัน แต่อย่างไรก็ตามยังมีการศึกษาที่รายงานว่าเลเซอร์ระดับต่ำไม่มีผลในการเร่งความเร็วการเคลื่อนที่ของฟัน

บทความนี้มีวัตถุประสงค์เพื่ออธิบายโดยสรุปเกี่ยวกับเลเซอร์ระดับต่ำ ประสิทธิผลในการเร่งการเคลื่อนที่ของฟัน ปฏิกิริยาการตอบสนองทางชีววิทยาหลังได้รับเลเซอร์ วิธีการฉายเลเซอร์ ข้อดีและข้อจำกัดของการใช้เลเซอร์ระดับต่ำ

คำสำคัญ: การใช้เลเซอร์เพิ่มความเร็วในการเคลื่อนฟัน การรักษาทางทันตกรรมจัดฟัน

Laser Accelerated Tooth Movement

Tasanee Wangsrimonkol* Montian Manosudprasit*
Poonsak Pisek* Pathomporn Chongcharueyskul**

Abstract

In recent years, methods of accelerating orthodontic tooth movement have been intensively investigated. Low-level laser therapy is an irradiation method of biostimulation of human alveolar bone remodeling. This method is of interest due to its reported success in experimental human studies[1, 2, 3]. However, there are also studies reporting no positive effect of low-level laser[4, 5]. Low-level laser therapy is also a non-invasive technique[6], easy to perform with inexpensive instruments[1] and can be used for reducing post-orthodontic adjustment pain[7]. There have been several in vitro and in vivo studies that have attempted to explain the mechanism of action of low-level laser therapy to speed up tooth movement[8, 9, 10].

The purpose of this paper is to provide an overview of low-level laser's effectiveness in accelerating tooth movement, biological response to low-level laser, protocol of how low-level laser should be used, and advantages and disadvantages of low-level laser therapy.

Keywords: *laser accelerated tooth movement, orthodontic treatment*

* คณะทันตแพทยศาสตร์ มหาวิทยาลัย ขอนแก่น
** นักศึกษาปริญญาโทสาขาทันตกรรมจัดฟัน มหาวิทยาลัย ขอนแก่น
* Faculty of Dentistry, Khon Kaen University
** Postgraduate student in Master of Science (Orthodontics), Khon Kaen University

Introduction

Orthodontic treatment is long duration[11, 12] and may result in increased risk of root resorption[13], dental caries[14] and periodontal health problems[4]. Any method that will shorten the duration of tooth movement is desirable[1]. Many attempts have been made to reduce orthodontic treatment duration including the development of new biomechanics techniques to move teeth efficiently and find methods of accelerating tooth movement. Suggested accelerating tooth movement methods are local injections with prostaglandins[15], osteocalcin[16], corticotomy[17, 18, 19], electric current stimulation[20] and pulsed electromagnetic field[21].

Low-level laser irradiation has gained interest for speeding up tooth movement due to reports of successful results[2, 3, 6, 22]; even though controversy still exists[4, 5, 23].

The purpose of this paper is to provide an overview of low-level laser therapy (LLLT) in accelerating tooth movement. The topics include effectiveness, biological responses to low-level laser, application protocol, advantages, disadvantages and risks or adverse effects. Readers can gain better understanding of LLLT before deciding to use it in the future or to conduct further study on this topic.

Basic laser

"Laser" is an abbreviation of Light Amplification by Stimulated Emission of Radiation[24]. Lasers have specific characteristics. They are produced in only a single wavelength (monochromatic), all in one phase (coherency) lead to a very high intensity light and with rays parallel to each other (collimation)[24]. Lasers are named according to their active mediums, which are used for producing different types of light; for example, CO_2 laser is produced from CO_2 gas.

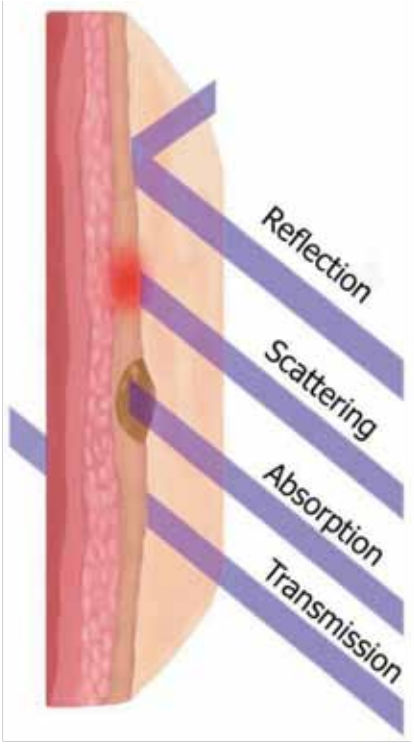


Fig. 1 Laser tissue interaction (modified from Olivi G, Crippa R, Iaria G, Kaitsas V, DiVito E, Benedicenti S., Laser in endodontics (Part I). Available from: URL:http://www.biolasecampus.com/laser-in-endodontics-part-i/)

When irradiating tissue with a laser, four types of tissue interaction may occur, depending on the optical properties of the particular tissue and laser wavelength used, including transmission, reflection, absorption and scattering (Fig. 1). The desirable interaction is the absorption of laser energy by target tissues. Reflection and transmission have no effects on the target tissue. Scattered laser weakens the energy, possibly producing inadequate biologic effect[24].

When tissue receives a laser beam, there are three photobiological effects which can occur[24] including photothermal interactions, photochemical interactions, photoacoustic interactions. Photothermal

interactions, or photoablation which means the laser energy is converted into heat, leading to the removal of tissue by vaporization and super heating of tissue fluids, coagulation and hemostasis. Photochemical interactions including biostimulation are enhancement of biochemical and molecular processes that normally occur in tissues, such as healing and repair. Photoacoustic interactions occur when a pulse of laser energy produces a shockwave on hard tissue. Then hard tissue explodes and pulverized, results in an abraded crater.

What are Lasers commonly used to accelerate tooth movement?

Lasers potentially useful for accelerating tooth movement are low-level lasers or "low intensity level laser" (LILL). The therapy performed with these lasers is called low-level lasers therapy (LLLT)[25]. LLLT operates in the range of power output 1-500 milliwatts (mW)[25] which is low enough so as not to increases the temperature of the treated tissue over normal body temperature[9], because biostimulation and not photoablation is desirable[25]. The objectives of most clinical applications of biostimulation are to reduce pain by mechanism of anti-inflammatory and neuronal effects, and to stimulate blood flow and increase cell activity[26].

Low-level laser units are small and inexpensive[27]. They are available as a self-contained, handheld device, which is portable and easy to move, or as a bench-top master unit (Fig. 2). There is no need for any cooling system and no specific safety rules such as apply to surgical laser units due to their low power output levels[27].

Fig. 2 Low-level laser units

Application protocol for accelerating orthodontic tooth movement

Wavelength

Infrared light used for biostimulatory effect involves lasers of HeNe (630-660 nanometer or nm.)[28], GaAlAs (780-890 nm.)[27] or GaAs (904-950 nm.)[28] because these provide laser wavelengths which stimulate bone cells beneath soft tissues and deeper in the alveolus[29]. Infrared light is minimally absorbed by hemoglobin and water; consequently, it could potentially penetrate tissues deeply by several millimeters[29].

More recent studies have found that diode lasers of GaAlAs and GaAs have more effectiveness in higher depth of tissue penetration than HeNe[30]; therefore, they should be suitable for accelerating orthodontic treatment[31].

Energy density or dose

Energy density is the amount of energy received by a target tissue. It is calculated by dividing energy output by the area of tissue receiving laser application in centimeters squared (cm^2). This parameter is reported in joules (J) of energy as J/cm^2. Energy density is the most important factor for biostimulation effect[32]. Biostimulatory effect is dose-dependent[27].

Small doses stimulate living systems, medium doses inhibit and large doses destroy[27]. Besides energy dose, another important point is scattering, which reduces the effectiveness of laser. Luger *et al.*[33] reported that the energy amount at the target area was 3-6% of its original intensity due to scattering while transmitting through the tissue. Moreover, a later study reported that only 50% of a 60 mW diode laser could penetrate 1.0 mm depth in human mandibular cortical bone[34].

The optimal dose or energy density for speeding up orthodontic tooth movement remains undetermined[6, 22]. Previously used energy densities have ranged from 0.71-25 J/cm^2. Genc[6] found that relatively lower energy density (2.5, 5, and 8 J/cm^2) was more effective than 20 J/cm^2 and 25 J/cm^2. However, study of Kansal *et al.*[5] showed the energy density of 4.2 J/cm^2 was unable to speed tooth movement. Therefore, further studies are required to determine optimal effective energy density.

Intervention schedule

The intervention schedule of low-level laser for accelerating orthodontic tooth movement has not yet been established[22]. However, there is information from animal studies that irradiation should be done at the start of tooth movement[35, 36] and be multiple rather than a single application[37] because cells are more sensitive to laser treatment during the proliferative and early phase of differentiation which is a specific period of the cell life cycle[37]. Therefore, multiple applications of low-level laser increase the possibility of cellular stimulation during a short window of susceptibility[37] . It has been observed that all previous clinical studies irradiated low-level laser frequently during the first week (Day 0, 3 and 7) after orthodontic force activation[1, 2, 3, 4, 5, 8, 31], then on Day 14[1, 2, 3, 5, 31]. After Day 14, different studies continued irradiation with lower frequency, applying laser weekly[5, 31] or every 15 days[3]. Some studies also repeated the cycle of Day 0, 3, 7, 14 every 3 weeks[2] or every month[1]. Detailed protocols can be found in Table 1.

Table 1 Protocol and efficacy of each study

Study	Laser type and Wavelength	Energy or power density	Power output	Total time/ tooth	Frequency	Duration	Outcome
Cruz[1] 2004	GaAlAs 780 nm	5 J/cm^2	20 mW	100 s/ tooth	Day 0,3,7,14 of each month	2 months	34% faster than control group
Limpanichkul[8] 2006	GaAlAs 860 nm	25 J/cm^2	100 mW	184 s/ tooth	Day 0,1,2 of each month	3 months	No significant difference
Youssef[2]2007	GaAlAs 809 nm	8 J/cm^2	100 mW	80 s/ tooth	Day 0,3,7,14 every stage (3 weeks)	9 weeks (3 stages)	1.98 fold in LLLT group

Study	Laser type and Wavelength	Energy or power density	Power output	Total time/ tooth	Frequency	Duration	Outcome
Doshi-Mehta[3] 2012	GaAlAs 810 nm	Not mention	80 mW	100 s/ tooth	Day 0,3,7,14 in first month, then every 15 days	4.5 months	30% faster than control side
Genc[31]2013	GaAlAs 808 nm	0.71 J/cm^2	20 mW	10 s	Day 0,3,7,14,21,28	35 days	Laser group faster than control group
Kansal[9]2014	GaAs 904 nm	4.2 J/cm^2	12 mW	100 s	Day 1,3,7,14,21,28, 35,42,49,56	63 days	No significant difference
Dominquez[38] 2015	Laser diode 670 nm	6.73 W/cm^2	200 mW	9 min	Day 0, 1,2,3,4	45 days	Total retraction Laser group =3.73±1.08 mm Control group = 2.71±0.90 mm

nm = nanometer, J/cm^2 = Joules/centimeters squared, W/cm^2 = Watt/centimeters squared, mW = milliwatt, s = second, min = minute,

Site of application

The laser device should contact the mucosa and be perpendicular to the target area in order to achieve the maximum penetration and absorption and minimize laser beam irradiating of an unintentional area[38]. Irradiation should cover as much of the periodontal support of the tooth as possible. A typical recommendation would be two irradiations on the cervical third (one medial and one distal), one level with the center of the root, and two at the apical third (one medial and one distal) all on both buccal and palatal/lingual mucosal surfaces[1, 3, 5, 31] or labial, palatal/lingual and distal mucosal surfaces[4, 8].

Mechanism of action of LLLT to accelerate orthodontic tooth movement

When irradiating low-level laser to living tissue, the light energy is absorbed by cellular photoreceptors, e.g. cytochromophores. The electromagnetic energy is converted by cellular mitochondria into ATP (adenosine tri-phosphate) leading to increased local cellular activity such as stimulation of DNA and RNA synthesis, increased protein production, modulation of enzymatic activity, variation of intra- and extra-cellular pH and elevated cellular metabolism[39]. Some energy is converted into heat, resulting in an increase in local micro-circulation through vasodilation[27].

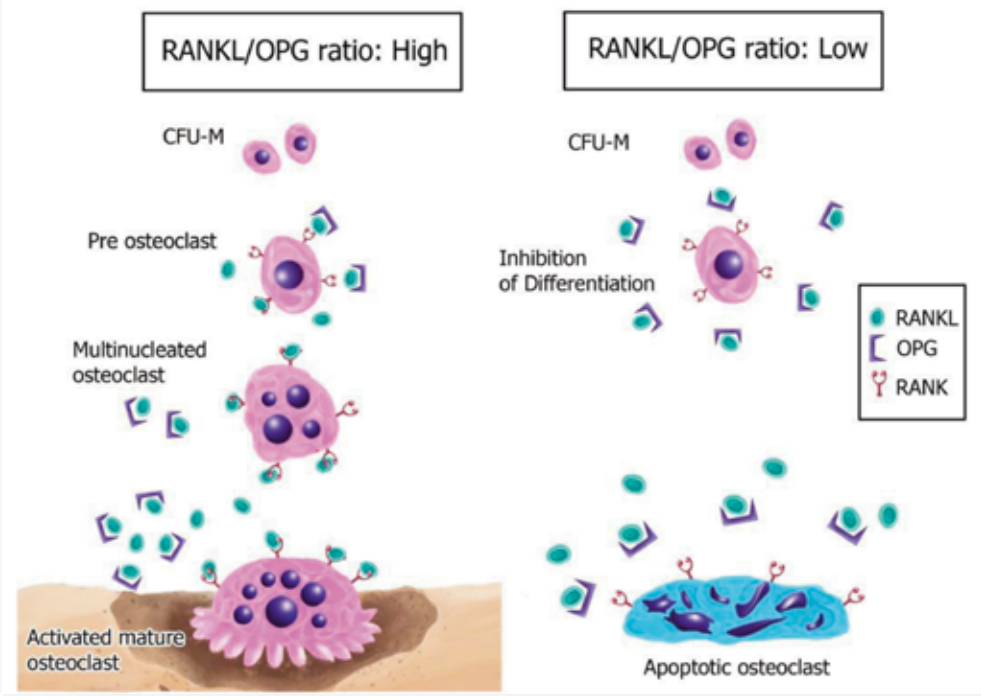


Fig. 3 RANK, RANKL and OPG system in controlling osteoclastogenesis (modified from Kajiya M, Giro G, Taubman MA, Han X, Mayer MP, Kawai T. Role of periodontal pathogenic bacteria in RANKL-mediated bone destruction in periodontal disease. J Oral Microbiol 2010; 2: 5532. - DOI: 10.3402/jom.v2i0.5532)

Osteoclasts are multinucleated cells with high activity of mitochondria; therefore, they are promptly affected by laser irradiation[9]. The result from animal studies showed that amount of osteoclasts in irradiation group significantly increased about 1.6-2.1 fold compared with non-irradiation group[10, 34]. The receptor activator of nuclear factor-kappa ligand/receptor activator of nuclear factor kappa B/ osteoprotegrin (RANKL/RANK/OPG) system plays a role in osteoclasts differentiation and function. When the RANK in the osteoclast precursor binds with RANKL, the process of differentiation, formation, and activation of osteoclast will occur[9] (Figure 3). OPG competes for the binding of RANKL result in decreasing differentiation and activation of preosteoclast. Therefore, OPG plays a role in protecting bone resorption (Fig. 3).

Gingival crevice fluid (GCF) is an inflammatory exudate that can be collected in the gingival sulcus. Mechanisms of bone resorption are relevant to the release of inflammatory mediators present in GCF. Therefore, biochemical analyses of GCF related to bone resorption have been done to study the cellular secretory responses of LLLT. After low-energy laser irradiation, a concentration of RANKL in human gingival crevicular fluid was increased greater than control group[8] whereas amount of OPG were not different[8]. The study of Fujita *et al.*[10] which investigated effect of LLLT on expression of RANK/ RANKL/ OPG in rat, also reported no difference of OPG level between laser group and control group.

Therefore, it could be implied that LLLT accelerate tooth movement by stimulating osteoclastic cell proliferation, differentiation and function on pressure side via increasing level of RANKL and RANKL/ OPG ratio.

Indication and contraindications of low-level laser therapy

Indication

LLLT is suggested in patients who are willing to attend multiple times with short intervals between laser applications[40].

Contraindications

Contraindications of LLLT are cancerous and pre-cancerous lesion in the oral cavity[39]. Irradiation laser to patient with coagulation disorders should be avoided due to its effect on blood flow[41]. Patients with epilepsy are also contraindicated because they may have a seizure during irradiation[39]. For patients who may have hyper- or hypothyroid conditions, irradiation over the thyroid gland should be avoided to prevent undesirable effects[41].

Adverse effects and risks of low-level laser therapy

Adverse effects of LLLT

There have been no reports that LLLT could cause root resorption[1], alveolar bone loss[3], and any adverse effects on oral mucosa[6], gingiva[6], periodontal ligament[1, 2, 3] and vitality of the dental pulp[42]. However, most studies were have been of relatively short duration, only a few weeks, which may be of too short duration to detect any adverse effect[40]. LLLT can be used safely without mutation effect[43] because low-level lasers are in infrared range which have no potential to ionize molecule or atom.

Risks of LLLT

LLLT are classified as a class IIIb hazard which can damage the retina. If viewed directly or from reflective light, these lasers can be dangerous to unprotected eyes for any duration[28]. Therefore, patient, practitioner and any person within the controlled area should wear appropriate eye protection. All protective glasses or goggles should be labeled with the wavelength, for which protection is given (Fig. 4).

Fig. 4 Wavelength specific protective eyewear

Advantages

1. Non-invasive technique[6] and reduction of post adjustment pain[7]

Methods of accelerating orthodontic tooth movement include local administration of exogenous substance, vibration technique, electrical current and surgical methods such as corticotomy or periodontal ligament distraction. However, most of these methods are invasive and painful in contrast to LLLT which does not cause pain or any discomfort during irradiation. LLLT is also claimed to offer a benefit of reducing pain that a patient experiences during orthodontic treatment[7]. Mechanism of reducing pain is by reducing the secretion of prostaglandin E2 and interleukin-1[42].

2. No adverse effects to irradiated tooth and periodontium[2, 3, 6, 42].

LLLT has no detectable adverse effect on tooth structure and periodontal health. However, it has so far been applied over such relatively short duration that there may not have been time for any accumulation of adverse effects[40]. This is in contrast to corticotomy which causes, or at least risks, interdental bone loss and loss of attached gingiva[44].

3. Easy to perform[45]

Application of LLLT is not a complex intervention and does not require any surgical skills. However, the practitioner must have a fundamental understanding of laser physics and ability to deliver optimal treatment to patient.

Efficacy of laser accelerated tooth movement

Efficacy of laser accelerated tooth movement is still controversial. Most studies have shown positive effects[1, 2, 3, 8, 31] with rate of tooth movement 30 - 99 % faster than control group. However, the studies of Limpanichkul *et al.*[4] and Kansal *et al.*[5] reported no effect of low-level laser. This contradictory result can be attributed to the differences in energy dose, duration of application and experimental design[40]. Protocol and efficacy of each study is shown in Table 1.

When comparing protocol and the outcome between non-effectively reported studies with studies which found that laser was effective[1, 2, 3, 8, 31]. The study which reports non-effective result had different experimental design as follows; 1) Use high energy density (25 J/cm^2) and too high output power (100 mW) which might cause a decrease or no effect on accelerating tooth movement[6] 2) Low frequency of laser exposure and time lapse between serial laser applications[22, 46] 3) Irradiation only first three days after reactivating retraction force, while other studies extended at least seven days[22, 46] 4) Different site of irradiation[4]: applied three points on both buccal and palatal/lingual surfaces (one at gingival margin, one at 4 mm. from gingival margin and one at 8 mm. from gingival margin) and two points on distal mucosal surfaces (distobuccal and distolingual line angle). While most studies[1, 3, 5, 31] irradiated 5 points on both buccal and palatal/lingual mucosal surfaces of retracting tooth (two at cervical third, one at center and two at apical third of root). 5) Choose different type of laser (GaAs laser) in the study[5] which had lower depth of tissue penetration than GaAlAs[47].

Data from systematic review showed that there is some evidence that LLLT is effective[6, 22]. But such a conclusion should be interpreted with caution[22] because the included studies have too small sample sizes[6, 22], high degree of heterogeneity[22, 46] and moderate to high risks of bias[6, 22].

Some reportedly effective studies contained a few biases such as start time of canine retraction after extraction first premolar[46, 48]. Start of canine retraction two weeks after the premolar extraction [2, 8] may produce more rapid movement because some studies[49, 50] claimed that tooth movement towards an extraction site would be accelerated if done immediately following extraction. Therefore, this period of waiting before starting may have affected the effectiveness of the studies.

Disadvantages

1. Controversial in its effectiveness

Some studies have reported that LLLT cannot accelerate tooth movement[4, 5] which result in weakening evidence for positive meta-analysis finding [6, 22].

2. High frequency of appointments

Even the optimal frequency of laser application for speeding up tooth movement is still undetermined. Nevertheless, the data from previous successful studies show that at least three applications per month were required for the treatment to be effective[51]. While surgical methods are performed once[52].

3. Optimal application protocol or parameter still not established

Further studies are required to determine the best protocols include optimal dose or frequency[6, 22].

Discussion

Since the outcome of laser treatment subsequently cause from multifactor i.e. wavelength, power output, energy density, timing and frequency of irradiation. Each study used different protocol. Therefore, the specific reasons that led to effectiveness or ineffectiveness of laser treatment in each study are still unable to be concluded. Even though many studies identify energy density as the most critical factor for the success of laser treatment, there are other important factors, such as wavelength which allows tissue to absorb laser optimally, appropriate timing and frequency and total period of laser application which provide sufficient energy for stimulating cells.

Note that clinical orthodontics involves a large number of interaction and random variables that can affect outcomes of controlled clinical trials in often unpredictable ways. Thus, LLLT, being one of the most recent orthodontic strategies, requires more studies and long time frame to be considered as a routine application. Also, clinicians should consider its value to patients by comparing benefits from accelerating tooth movement with added financial costs from additional equipment and increased number of visits so that the laser can be routinely applied.

Conclusions

LLLT is a non-invasive technique with potential to accelerate tooth movement. Its effectiveness is still controversial due to differences in application protocol and experimental design. It requires further studies which have more sample size and high quality to test effectiveness. LLLT is claimed to increases velocity of tooth movement via stimulation of RANK/RANKL/OPG system which results in greater osteoclast differentiation and function. Adverse effect of LLLT is not reported but may be because of short time of reported clinical trials[6]. Before routine application, it requires more studies about application protocol, adverse effects and cost-benefit analysis.

Acknowledgement

The authors would like to express our great appreciation to Associate Professor Keith Godfrey for his valuable and constructive suggestions. His willingness to give his time so generously has been very much appreciated. We would also like to thank the faculties and staff of the Faculty of Dentistry, Khon Kaen University

References

1. Cruz DR, Kohara EK, Ribeiro MS, Wetter NU. Effects of low-intensity laser therapy on the orthodontic movement velocity of human teeth: a preliminary study. ***Lasers Surg Med*** 2004; 35: 117-20.
2. Youssef M, Ashkar S, Hamade E, Gutknecht N, Lampert F, Mir M. The effect of low-level laser therapy during orthodontic movement: a preliminary study. ***Lasers Med Sci*** 2008; 23: 27-33.
3. Doshi-Mehta G, Bhad-Patil WA. Efficacy of low-intensity laser therapy in reducing treatment time and orthodontic pain: a clinical investigation. ***Am J Orthod Dentofacial Orthop*** 2012; 141: 289-97.
4. Limpanichkul W, Godfrey K, Srisuk N, Rattanayatikul C. Effects of low-level laser therapy on the rate of orthodontic tooth movement. ***Orthod Craniofac Res*** 2006; 9: 38-43.
5. Kansal A, Kittur N, Kumbhojkar V, Keluskar KM, Dahiya P. Effects of low-intensity laser therapy on the rate of orthodontic tooth movement: A clinical trial. ***Dent Res J (Isfahan)*** 2014; 11: 481-8.

6. Ge MK, He WL, Chen J, Wen C, Yin X, Hu ZA, et al. Efficacy of low-level laser therapy for accelerating tooth movement during orthodontic treatment: a systematic review and meta-analysis. ***Lasers Med Sci*** 2015; 30: 1609-18.
7. Tortamano A, Lenzi DC, Haddad AC, Bottino MC, Dominguez GC, Vigorito JW. Low-level laser therapy for pain caused by placement of the first orthodontic archwire: A randomized clinical trial. ***Am J Orthod Dentofacial Orthop*** 2009; 136: 662-7.
8. Dominguez A, Gomez C, Palma JC. Effects of low-level laser therapy on orthodontics: rate of tooth movement, pain, and release of RANKL and OPG in GCF. ***Lasers Med Sci*** 2015; 30: 915-23.
9. Altan BA, Sokucu O, Ozkut MM, Inan S. Metrical and histological investigation of the effects of low-level laser therapy on orthodontic tooth movement. ***Lasers Med Sci*** 2012; 27: 131-40.
10. Fujita S, Yamaguchi M, Utsunomiya T, Yamamoto H, Kasai K. Low-energy laser stimulates tooth movement velocity via expression of RANK and RANKL. ***Orthod Craniofac Res*** 2008; 11: 143-55.
11. Fisher MA, Wenger RM, Hans MG. Pretreatment characteristics associated with orthodontic treatment duration. ***Am J Orthod Dentofacial Orthop*** 2010; 137: 178-86.
12. Fink DF, Smith RJ. The duration of orthodontic treatment. ***Am J Orthod Dentofacial Orthop*** 1992; 102: 45-51.
13. Segal GR, Schiffman PH, Tuncay OC. Meta analysis of the treatment-related factors of external apical root resorption. ***Orthod Craniofac Res*** 2004; 7: 71-8.
14. Richter AE, Arruda AO, Peters MC, Sohn W. Incidence of caries lesions among patients treated with comprehensive orthodontics. ***Am J Orthod Dentofacial Orthop*** 2011; 139: 657-64.
15. Patil AK, Keluskar KM, Gaitonde SD. The Clinical application of prostaglandin E1 on orthodontic tooth movement. ***J ind orthod Soc*** 2005; 38: 91-8.
16. Kobayashi Y, Takagi H, Sakai H, Hashimoto F, Mataki S, Kobayashi K, et al. Effects of local administration of osteocalcin on experimental tooth movement. ***Angle Orthod*** 1998; 68: 259-66.
17. Hassan AH, Al-Fraidi AA, Al-Saeed SH. Corticotomy-assisted orthodontic treatment: review. ***Open Dent J*** 2010; 4: 159-64.
18. Wilcko MT, Wilcko WM, Pulver JJ, Bissada NF, Bouquot JE. Accelerated osteogenic orthodontics technique: a 1-stage surgically facilitated rapid orthodontic technique with alveolar augmentation. ***J Oral Maxillofac Surg*** 2009; 67: 2149-59.
19. Murphy KG, Wilcko MT, Wilcko WM, Ferguson DJ. Periodontal accelerated osteogenic orthodontics: a description of the surgical technique. ***J Oral Maxillofac Surg*** 2009; 67: 2160-6.
20. Kim DH, Park YG, Kang SG. The effcets of electrical current from a micro-electrical device on tooth movement. ***Korean J Orthod*** 2008; 38: 337-46.
21. Stark TM, Sinclair PM. Effect of pulsed electromagnetic fields on orthodontic tooth movement. ***Am J Orthod Dentofacial Orthop*** 1987; 91: 91-104.
22. Gkantidis N, Mistakidis I, Kouskoura T, Pandis N. Effectiveness of non-conventional methods for accelerated orthodontic tooth movement: a systematic review and meta-analysis. ***J Dent*** 2014; 42: 1300-19.
23. Abdallah MN, Flores-Mir C. Are interventions for accelerating orthodontic tooth movement effective? ***Evid Based Dent*** 2014; 15: 116-7.
24. Coluzzi DJ. An overview of laser wavelengths used in dentistry. ***Dent Clin North Am*** 2000; 44: 753-65.
25. Sun G, Tuner J. Low-level laser therapy in dentistry. ***Dent Clin North Am*** 2004; 48: 1061-76, viii.
26. Lim HM, Lew KK , Tay DK. A clinical investigation of the efficacy laser therapy in reducing orthodontic postadjustment pain. ***Am J Orthod Dentofacial Orthop*** 1995; 108: 614-22.
27. Parker S. Low-level laser use in dentistry. ***Br Dent J*** 2007; 202: 131-8.
28. Sattayut S. Lasers in oral and maxillofacial surgery. Khon Kaen: Khon Kaen university; 2013. p. 194.
29. Hillenkam F. Interaction between laser radiation and biological system In: Hillenkam F, Pratesi R, Scassi CA, editors. Lasers in biology and medicine. Newyork: Plenum Press; 1990. p. 37-68.

30. Ohshiro T, Calderhead RG. Development of low reactive-level laser therapy and its present status. ***J Clin Laser Med Surg*** 1991; 9: 267-75.
31. Genc G, Kocadereli I, Tasar F, Kilinc K, El S, Sarkarati B. Effect of low-level laser therapy (LLLT) on orthodontic tooth movement. ***Lasers Med Sci*** 2013; 28: 41-7.
32. Sommer AP, Pinheiro AL, Mester AR, Franke RP, Whelan HT. Biostimulatory windows in low-intensity laser activation: lasers, scanners, and NASA's light-emitting diode array system. ***J Clin Laser Med Surg*** 2001; 19: 29-33.
33. Luger EJ, Rochkind S, Wollman Y, Kogan G, Dekel S. Effect of low-power laser irradiation on the mechanical properties of bone fracture healing in rats. ***Laser Surg Med*** 1998; 22: 97-102.
34. Kawasaki K, Shimizu N. Effects of low-energy laser irradiation on bone remodeling during experimental tooth movement in rats. ***Lasers Surg Med*** 2000; 26: 282-91.
35. Ozawa Y, Shimizu N, Kariya G, Abiko Y. Low-energy laser irradiation stimulates bone nodule formation at early stages of cell culture in rat calvarial cell. ***Bone*** 1998; 22: 347-54.
36. Saito S, Shimizu N. Stimulatory effects of low-power laser irradiation on bone regeneration in midpalatal suture during expansion in the rat. ***Am J Orthod Dentofacial Orthop*** 1997; 111: 525-32.
37. Ng GY, Fung DT, Leung MC, Guo X. Comparison of single and multiple applications of GaAlAs laser on rat medial collateral ligament repair. ***Lasers Surg Med*** 2004; 34: 285-9.
38. Simunovic Z, Simunovic X. Laser in dentistry. In: Simunovic Z, editor. Lasers in medicine and dentistry: Basic science and up to date clinical application of low energy-level laser therapy (LLLT) Switzerland: Zlatko Simunovic; 2000. p. 477-91.
39. Simunovic Z, Simunovic K. Laser in surgery and dentistry. Slovenia: Eurotradeprint; 2001. p. 792.
40. Nimeri G, Kau CH, Abou-Kheir NS, Corona R. Acceleration of tooth movement during orthodontic treatment--a frontier in orthodontics. ***Prog Orthod*** 2013; 14: 42.
41. Tuner J, Kristensen PH. Low-level lasers in dentistry. In: Convissar RA, editor. Principles and practice of Laser dentistry. China: Mosby; 2011. p. 266.
42. Walsh LJ. The current status of low level laser therapy in dentistry. Part 2. Hard tissue applications. ***Aust Dent J*** 1997; 42: 302-6.
43. Hode L. Risks and side effects associated with LLLT. In: Simunovic Z, editor. Laser medicine and dentistry: basic science and up to date clincal application of low energy-level laser therapy (LLLT). Switzerland: Zlatko Simunovic; 2000. p. 513-27.
44. Alghamdi AS. Corticotomy facilitated orthodontics: Review of a technique. ***Saudi Dent J*** 2010; 22: 1-5.
45. Parker S. Low-level laser use in dentistry. ***Br Dent J*** 2007; 202: 131-8.
46. Long H, Zhou Y, Xue J, Liao L, Ye N, Jian F, et al. The effectiveness of low-level laser therapy in accelerating orthodontic tooth movement: a meta-analysis. ***Laser Med Sci*** 2015; 30: 1161-70
47. Ohshiro T, Calderhead RG. Development of low reactive-level laser therapy and its present status. ***J Clin Laser Med Surg*** 1991; 9: 267-75.
48. Long H, Pyakurel U, Wang Y, Liao L, Zhou Y, Lai W. Interventions for accelerating orthodontic tooth movement: a systematic review. ***Angle Orthod*** 2013; 83: 164-71.
49. Liou EJ, Huang CS. Rapid canine retraction through distraction of the periodontal ligament. ***Am J Orthod Dentofacial Orthop*** 1998; 114: 372-82.
50. Lv T, Kang N, Wang C, Han X, Chen Y, Bai D. Biologic response of rapid tooth movement with periodontal ligament distraction. ***Am J Orthod Dentofacial Orthop*** 2009; 136: 401-11.
51. Sousa MV, Scanavini MA, Sannomiya EK, Velasco LG, Angelieri F. Influence of low-level laser on the speed of orthodontic movement (abstract). ***Photomed Laser Surg*** 2011; 29: 191-6.
52. Cano J, Campo J, Bonilla E, Colmenero C. Corticotomy-assisted orthodontics. ***J Clin Exp Dent*** 2012; 4: e54-9.

บทความปริทัศน์
Review Article

บิสฟีนอล-เอ (Bisphenol-A) และไดออกซินส์ (Dioxins) กับทันตกรรมจัดฟัน

วราพร เด่นศิริอักษร* นิตา วิวัฒนทีปะ** พีรพงศ์ สันติวงศ์** สมชัย มโนพัฒนกุล*** สุรชัย เดชคุณากร**

บทคัดย่อ

บิสฟีนอล-เอ (bisphenol-A or BPA) และไดออกซิน (Dioxins) เป็นสารพิษที่เป็นอันตรายร้ายแรงต่อร่างกายมนุษย์ โดยบีพีเอทำให้เกิดฤทธิ์ที่ทำให้แสดงลักษณะของฮอร์โมนเอสโทรเจน และไดออกซินเป็นหนึ่งในสารเคมีที่มีความเป็นพิษมากที่สุดและเป็นอันตรายต่อระบบร่างกายโดยรวม จึงเป็นที่น่าสนใจว่าสารพิษทั้งสองนี้มีส่วนเกี่ยวข้องกับงานทางทันตกรรมจัดฟันหรือไม่ บทความปริทัศน์ฉบับนี้เป็นการรวบรวมข้อมูลรายละเอียดของสารพิษทั้งสองนี้ ในแง่ของสูตรทางเคมี และผลเสียต่อสุขภาพร่างกายและช่องปาก ซึ่งจากการศึกษาพบว่าทันตวัสดุทางทันตกรรมจัดฟันปลดปล่อยสารบีพีเอเข้าสู่ร่างกายบ้าง ส่วนไดออกซินอาจทำให้ฟันกรามและฟันตัดมีการพร่องของการสะสมแร่ธาตุ (เอ็มไอเอช) [Molar-incisor hypomineralization (MIH)] ถึงแม้ว่าปริมาณสารบีพีเอที่ปล่อยจากทันตวัสดุทางทันตกรรมจัดฟันจะต่ำกว่าระดับมาตรฐานที่จะเป็นอันตรายต่อร่างกายมาก บทความนี้นำเสนอข้อแนะนำสำหรับทันตแพทย์จัดฟันเพื่อลดปริมาณสารบีพีเอ นอกจากนี้ยังได้นำเสนอบทบาทของทันตแพทย์จัดฟันในการจัดการปัญหา หากพบมีการเกิดเป็นพิษของสารไดออกซินต่อสุขภาพฟันของเด็กในชุมชน

คำสำคัญ: บิสฟีนอล-เอ ไดออกซินส์ ทันตกรรมจัดฟัน

Bisphenol-A, Dioxins and Orthodontics

Warabhorn Densiri-agsorn* Nita Vitwatanatipa Peerapong Santiwong****

Somchai Manopatanakul* Surachai Dechkunakorn****

Abstract

Bisphenol-A(BPA) and Dioxins pose a serious threat to human health. Estrogenicity is the main effect to human being caused by BPA overdose. In addition, Dioxins are ones of the most hazardous chemicals to the systemic health. This review gathered the detail information of these two toxic products in terms of chemical formulas and hazard to human general and oral health. Orthodontic aspect of these two chemicals is intriguing and included. While BPA releases from orthodontic material, Dioxins may cause dental defect especially permanent molar and incisor [Molar-incisor hypomineralization (MIH)]. Although the amount of BPA released from orthodontic material is well below the standard, recommendations for orthodontist of how to minimize this BPA release were given. The suggested role of orthodontist to manage the Dioxins toxicity to the dental health in the community was also versed.

Keywords: *Bisphenol-A, BPA, Dioxins, Orthodontics*

* นักศึกษาหลักสูตรการฝึกอบรมทันตแพทย์เฉพาะทาง สาขาทันตกรรมจัดฟัน คณะทันตแพทยศาสตร์ มหาวิทยาลัยมหิดล
** ภาควิชาทันตกรรมจัดฟัน คณะทันตแพทยศาสตร์ มหาวิทยาลัยมหิดล
*** ภาควิชาทันตกรรมทั่วไปขั้นสูง คณะทันตแพทยศาสตร์ มหาวิทยาลัยมหิดล
* Specialty training student in Orthodontics, Faculty of Dentistry, Mahidol University
** Department of Orthodontics, Faculty of Dentistry, Mahidol University
*** Department of Advanced General Dentistry, Faculty of Dentistry, Mahidol University

Introduction

Bisphenol A (BPA) and Dioxins, these two toxic chemicals are commonly found in the environment. BPA has been used in plastic manufacturing, i.e., baby bottle, water bottle, food storage containers, and other food containers. In addition, BPA can be found in food and beverage cans lining. Heating food in plastic container may increase the amount of BPA leaching. More importantly, BPA can also leach out from some dental materials. These include dental sealant, plastic bracket and clear aligner. While invisalign appliances are recommended to be worn in patients' mouth for more than 20 hours a day, it is interesting to know whether these appliances leach any toxic product to human body. Dioxins are the by-products of polyvinyl chloride (PVC) plastic production and medical waste incineration. They are also found in plastic container, bleach and food. Foods that contain the highest amount of Dioxins are meat, milk, daily product and egg. Agent Orange which is the chemical used in Indo-china countries during Vietnam War also releases Dioxins. Dioxins are also formed by natural processes such as volcano eruption.[1-4] It is intriguing to know the detail information of these two toxic chemicals in relation to Orthodontic treatment.

BPA

Polycarbonate plastic contains a significant amount of BPA. While polycarbonate plastic is strong, durable and highly transparent, studies reported that BPA leached from polycarbonate containers into food and drink. In developed countries, polycarbonate plastics which contain BPA are classified in the group stamped with 3-arrow folded or a triangle with number 7 inside with the letters "PC", letter "O", or the word "other" underneath as recycling code (Fig. 1). However, Japan and Taiwan may use their own individual recycling code.[1-3, 5-8]

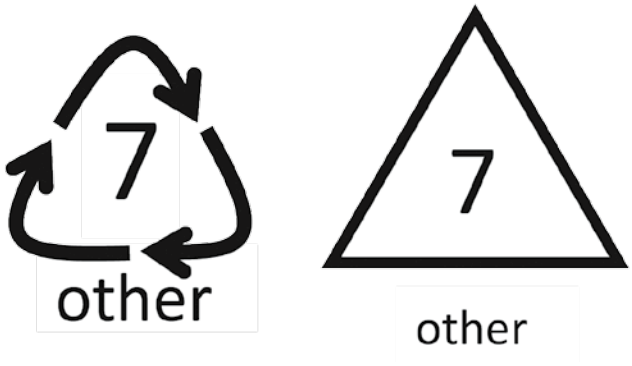


Fig. 1 Three-arrow folded or triangle with number 7 inside with the letters "PC", letter "O" or word "other" inside as recycling codes indicating that this plastic may contain BPA. (Figure redrawn from references[4-5])

European Food Safety Authority (EFSA) ascertained that the amount of BPA leached into human body both from food and non-food was well below the tolerable daily intake [TDI, 4 microgram per kilogram body weight per day (µg/kgBW/day)]. This TDI is far lower than maximum safety dose of the United States Environmental Protection Agency (US EPA, 50 µg/kgBW/day). Despite all these announcements, the Food and Drug Administration (FDA) of Thailand, the Ministry of Public Health, had issued the law banning BPA in the baby bottle, milk container for baby and children (No. 369, 2558 B.E.). It was legislated and reported in the Royal Thai Government Gazette in February 3rd, 2015. The enactment was on the first of August, 2015. As such, baby bottles, milk containers for babies and children in Thailand do not contain BPA. This means that BPA must be below the laboratory detectable level [limit of detection, (LOD) = 0.25 microgram per liter]. In addition, more than 15 states in the US have

limited the use of BPA and most plastic manufacturers change to produce BPA free products.[1-3, 9-10]

BPA can be detected from urine analysis, blood tests, or the test of placenta and breast milk. The FDA of the US found that most studies reported the blood and urine BPA tests and there was a higher urine BPA level in female than male. Children urinary BPA level was higher than adult's. Moreover, it was reported that BPA transmitted from mother to the child via breast feeding, placenta and blood transfusion.[1-3]

BPA and its effects to human

The double benzoic rings chemical when leached out, can convert to BPA. This leached BPA mimics the action of estradiol which is a hormone in the estrogen group. This leads to the estrogenicity property of BPA.[11]

Although EFSA stated that the amount of BPA under a certain level was safe for human, studies in rats showed that if BPA was administered in low dose for a long period of time (0.025-2.5 µg/kgBW/day), it could cause reproductive system abnormalities, breast cancer, prostate cancer, neurobehavioral problem, obesity, diabetes, and immunological deficiency. Moreover, human toxicity studies showed that high dose of BPA caused weak estrogenicity, increase human breast cancer cells, disruptive effect to human pancreatic cell, obesity, behavioral change, and cardiovascular diseases. In addition, a low dose of BPA was mentioned to affect human cells. Conversely, some reports contradicted that BPA showed no effect to human beings. The authors speculated that there were no precise or standardization of how low is low dose. Therefore, this topic, at present, still is open to investigation.[3, 12-24]

BPA and dentistry

In dentistry, pure BPA has not been used as the raw material to produce dental materials, however, some dental materials may contain

- Bisphenol A diglycidyl methacrylate (BisGMA)] and
- Bisphenol A ethoxylated glycol dimethacrylate (BisEMA)

Insignificant amount of pure BPA can be detected in dental materials (2ppm or 2µg/g of material) in the form of contaminant from reagent. In contrast, BPA leaches out of dental material that contains Bisphenol A dimethacrylate (BisDMA) (Fig. 2). Degradation and hydrolysis turn 89 to 100% of BisDMA into BPA. BisDMA is also used in the production of some dental resin composite and sealant. It was speculated that the ester linkage of BisDMA caused this BPA release (fig 3).[25-27]

Leaching of BPA from dental resin and dental adhesive occurs when they are not completely cured. It leaches out even more when these conditions take place intraorally. These conditions involve the raise in temperature, wear of dental material, increasing in bacterial and salivary enzymes. Dropping of pH also initiates leaching of BPA from dental resin composite and adhesives.[28-30]

For Modern dental filling material, average amount for one filling (approximately 0.25 g) contains less than 0.5 µg of BPA comparing to traditional filling material which contains 5 µg BPA. After being used for 1 year, accumulated BPA release was still less than EFSA and US EPA standard for one day. To be specific, when BisGMA and BisEMA were aged for 24 hours, BPA cannot be detected from the samples' saliva. On the other hand, 89-100 percent of BisDMA converted to BPA up to the type of dissolving solution.[1, 25-26, 31]

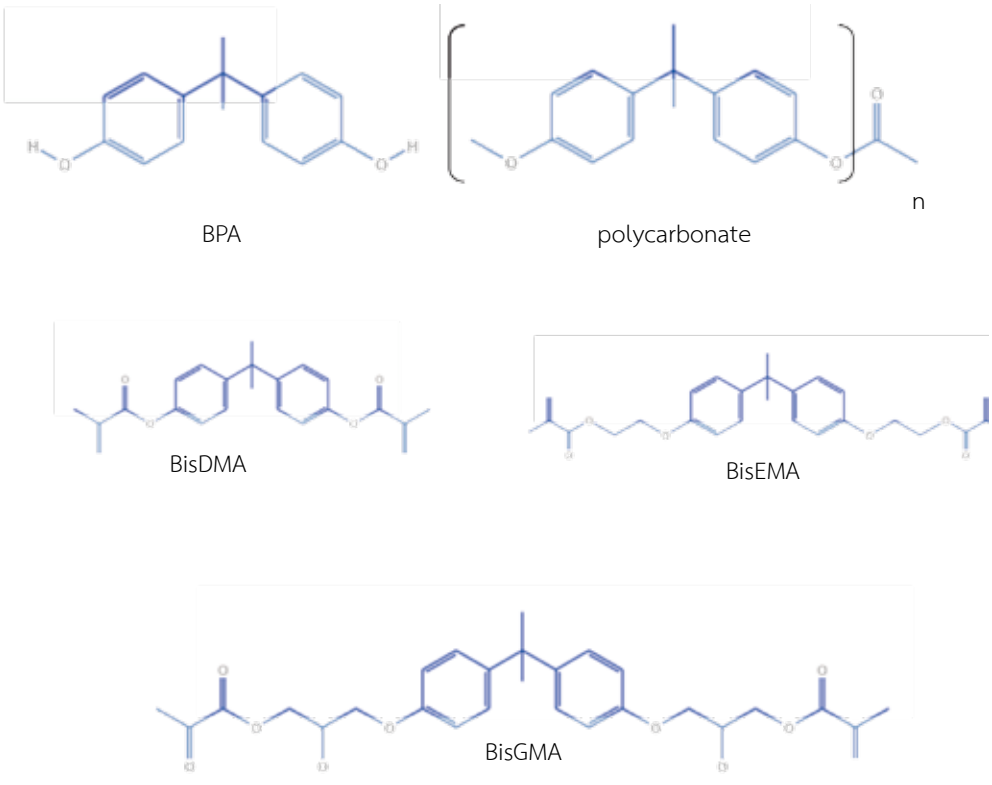


Fig. 2 Chemical structures of BPA, polycarbonate and BPA-derivatives found in dental materials, i.e. BisGMA, BisEMA and BisDMA. (Figure redrawn from Chen and Suh 2013[1])

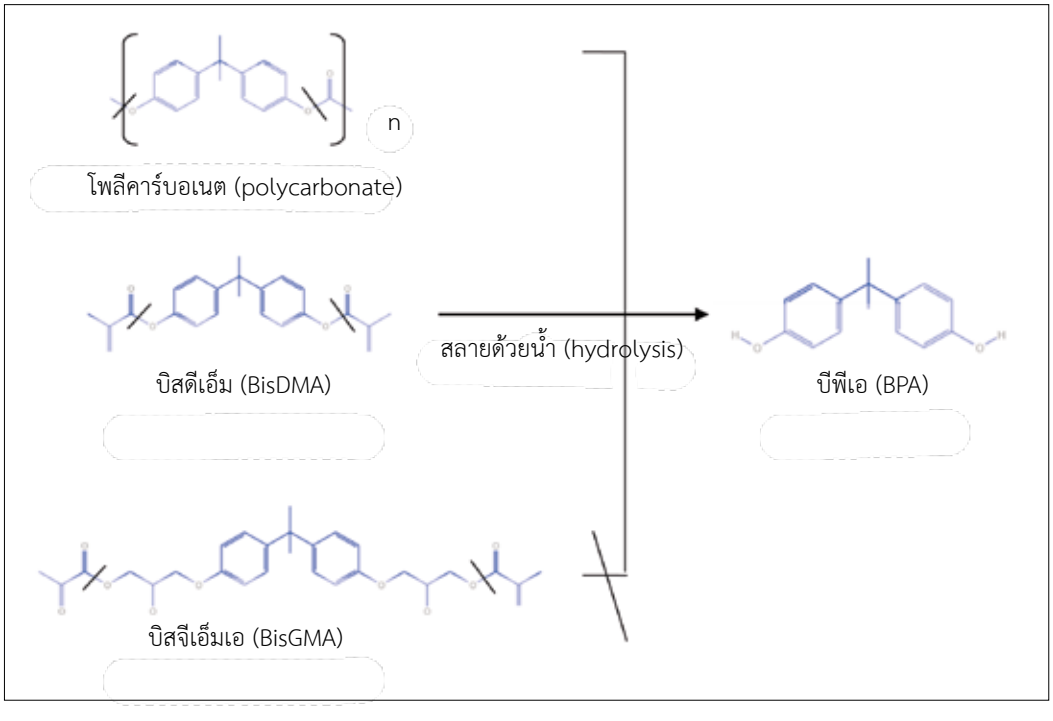


Fig. 3 Hydrolysis at the ester linkage of polycarbonate and BisDMA resulted in BPA. While ether linkage is resistance to hydrolysis, this explain why BisGMA does not hydrolyze to BPA. (Figure redrawn from Chen and Suh 2013[1])

BPA and Orthodontics

In orthodontics, BPA release was noticed from the use of polycarbonate bracket and polycarbonate clear aligner and it was also found in resin composite and dental adhesive. As mentioned earlier, there is no use of pure BPA in the manufacturing of orthodontic products. However, BPA derivatives such as BisGMA, BisEMA, and BISDMA, are used to manufacture orthodontic produces.[32-34]

As previously described, insignificant amount or only 2 ppm of BPA were identified from BisGMA and BisEMA. Nevertheless, to minimize the amount of BPA released from orthodontic material, the bracket adhesion material should be best possibly completely cured. When chemically cured orthodontic adhesive was used as manufacturers' recommendations, it will leach only 0.35 ppm BPA. Placing the tip of light-curing device close to the bracket adhesive significantly reduces the amount of BPA leaching. Try to completely light-cured orthodontic adhesive by placing tip of light-emitting diode device (LED) for 20 seconds close to the bracket yielded less than 9.5 ppm of BPA. BPA release when using this method was also reported to be less than when using halogen light for 40 seconds.[35]

BPA released from orthodontic and orthodontic-related materials was concluded in table 1. This table summarizes information from both *in vitro* and *in vivo* studies. It should be noted here that, among orthodontic materials, polycarbonate bracket released the highest amount of BPA.[1]

Dental materials	The amount of BPA released	
	in vitro studies	in vivo studies
Orthodontic adhesive	less than detection limit 0.1 ppm (in ethanol)[61] less than detection limit to 65.67 ppm[36]	0.8-20.88 ng/mL36
Polycarbonate bracket	697 µg/g material (40 months in water)[62] 37.4 µg/g material (34 months in water)[63] 0.01-0.4 µg/g material (1 hr in water)[64]	324-697 µg/g material (40 months in saliva)[63] 38-60 µg/g material (18 months in saliva)[63]
Polycarbonate denture base	2.2 µg/gram material (34 months in water)[63]	
Adhesive to bond fixed lingual retainer	Transbond XT (3M ESPE) 2.9 µg/mL (1 month in water)[65]	Filtek Z250 (3M Unitek) 20.9 ng/mL (30 minutes after placement)[66]

Table 1 The amount of BPA released from dental materials (in vivo and in vitro studies, this table was adapted from Chen and Suh 2013[1])

A Study was also conducted to test BPA leached from orthodontic materials under extreme physical and thermal conditions. After soaking the material in artificial saliva for 2 weeks, no BPA was traceable from nearly all orthodontic materials including clear thermoformed Biocryl retainer, polycarbonate bracket, bracket adhesive, invisalign appliance, elastic band, elastomeric ligature, elastomeric chain and Band-lok. However, bracket adhesive (Transbond XT) released 2.75 µg/1 g of material on day 3 of soaking. From the estimation, to be lower than the EFSA standard, the maximal allowance of using bracket adhesive will be 1.5 g per patient. (The average amount of bracket adhesive is 2-4 g/tube.) Moreover, on day one of soaking in artificial saliva, heated orange thermoformed Biocryl retainer (2 mm; 125-mm, round, Great Lakes Orthodontics, Ltd., item number 021-086, lot number 11-08) leached BPA of 7.63 µg/1 g of material. This retainer weight was estimated to be 4-5 g per piece depending on the size and outline of the appliance.[28, 36] To maximize the amount of the toxic product, the 5 g per piece is elected for this rough calculation. Therefore, estimation of this BPA leaching will be 38.15 µg per piece; finally resulting in 76.30 µg of BPA per person (This calculation includes 2 pieces of retainers per person). The average weight of Thai teenager boys and girls who are the majority of orthodontic patients are reported to be 64.24 and 52.70 kg respectively.[37] Therefore, the amount of BPA leached to average Thai teenager boys and girls will be 1.19 and 1.45 µg/kgBW/day respectively. Consequently, the amount of BPA from this orange retainer is still lower than the EFSA standard (4µg/kgBW/day) for both genders. Hence, they must be far lower than the US EPA recommendations (50µg/kg BW/day). One study particularly conducted the test of invisalign soaking in saline for 2 months. The authors revealed no cytotoxicity, nor the estrogenicity from invisalign appliance.[38]

The effect to human of BPA from orthodontic material

There are basically three routes for chemical from resin-based material entering human body. These three routes are breathing, absorption through digestive system and through dentin into pulpal tissue. Modern dental materials leach out minimal amount of BPA. Absorption of BPA through digestive system is considered to be minimal when compared to BPA from food intake and environment. Although the *in vitro* study indicated that particulate of bracket adhesive showed positive result on estrogenicity assay[31], to our knowledge, no study to date shows the BPA toxicity from breathing in this adhesive particulate. Moreover, orthodontists can prevent this mishap by using suction system carefully. Therefore, at this stage, there is no strong evidence showing that BPA from clinically used orthodontic material poses any serious threat to human health in general.

To summarize, based on present documents, the minimal risk of BPA leaching in orthodontics might be up to[36]

- The duration and surface area of the adhesive-saliva contact. These factors determine the amount of BPA release, i.e.,
  - Bonded lingual retainer leaching more BPA than adhesive around bracket rim,
  - Bracket adhesive left intra-orally due to long-term orthodontic treatment,
  - Long-term bonded lingual retainers (especially in case with increase bacterial plaque enzyme, esterase),
  - The amount of the adhesive left after bracket debonding.
- The degree of conversion of the adhesive affects the BPA leaching. Hence, the distance of the light curing tip to the adhesive influences the BPA release.

- The thickness of bracket adhesive. (This excessively thick adhesive shows both more adhesive-saliva surface contact and difficulty in light-activated polymerization process of the adhesive.)

It is the effort of this article to formulate the clinical guideline for orthodontists to minimize the amount of BPA from the information reviewed. They were listed as followings:

- Avoid the use of polycarbonate bracket.
- Use only the clear retainer, when thermoformed Biocryl retainer is the choice.[28]
- Opt for bracket adhesive without or with the least amount of BPA release.[31]
- Whenever possible, opt for chemically cured bracket adhesive and follow the manufacturer's recommendations rigidly.[35]
- Completely eliminate the bracket adhesive excess from the bracket margin during bonding.[31]
- If light-cured adhesive is used, to fully cure the adhesive, place the light curing tip closed to the adhesive in every direction. Opt for LED curing machine and cure for at least 20 seconds.[35, 36] LED curing machines mostly are lighter, smaller and produce less heat.
- Use pumice prophylaxis to minimize the excess adhesive.[36]
- Soak retainer in 60°C water for 2-3 hours before delivery.[28]

In dealing with other toxic substances in dentistry, clinical guidelines are recommended. The adaptations of these clinical guidelines to minimize BPA are[28, 36, 39-41]

- Using high-power vacuum directly to the adhesive when abrading the adhesive out, and
- Using the suction on the floor of the mouth to evacuate the remaining saliva that may contain the adhesive.
- Right after orthodontic procedures using adhesive, for example, bracket and fixed retainer bonding and adhesive removal, rinse mouth out as much as possible, especially during the first hour after such procedures.

Dioxins

Dioxins are organic compounds classified as halogenated aromatic hydrocarbons. The most common forms of Dioxins are

- Polychlorinated dibenzodioxins (PCDDs)
- Polychlorinated dibenzofurans (PCDFs)
- Polychlorinated biphenyls (PCBs)
- 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)

To human, TCDD is the most toxic chemical and carcinogenic (fig. 4).[4, 42]

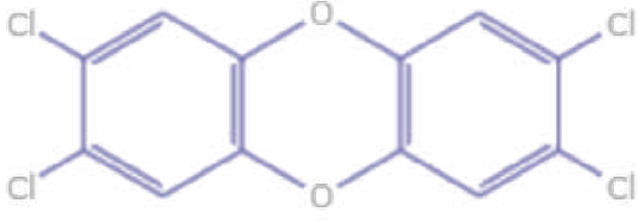


Fig. 4 Chemical structure of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)

Dioxins can be absorbed into human body through the environment because they may be found in our common daily live. Dioxins occur as by-products in the manufacture of polyvinyl chloride (PVC) plastic, food container, chlorine bleach, chlorinated phenol, and from natural sources such as volcanoes and forest fires. There have been many incidents of the incineration of chlorinated organic compound yielding Dioxins pollutants. Moreover, Dioxins formation can occur from Agent Orange used in Indo-china countries

during Vietnam War. Further, there were possibilities that Agent Orange was transported, stored or used in Thailand during the time. This is due to the fact that there is the announcement for compensation to veterans who were involved with Agent Orange in Thailand.[42-44]

To relatively demonstrate the toxicity of Dioxins, toxicities of PCDDs, PCDFs, PCBs are compared to the toxicity of TCDD using Toxicity Equivalency Factors (TEF). In other words, to indicate the level of the toxicity of dioxin-like compound, TEF is used as the equivalent toxicity to TCDD. For example, TEF of 1,2,3,7,8 PCDD is 0.5. This means that the toxicity of 1,2,3,7,8 PCDD is half of TCDD's toxicity.

When more than one type of Dioxins are mixed together, to attain the total toxicity of that mixture, the International Toxicity equivalents (I-TEQ) can be calculated using the summation from this equation.

I-TEQ = TEF x the amount of each Dioxin.[45]

World health organization (WHO) announced the tolerable daily intake (TDI) of TCDD at 1-4 picogram I-TEQ/kgBW/day. It was also demonstrated that population in some industrialized countries might absorb 2-6 picogram I-TEQ/kgBW/day.[46] It should be mentioned here that this TDI of TCDD (picogram, 10^{-12} g) is far less than that of BPA (microgram, 10^{-6} g).

At present, there is no evidence-based information showing that there are Dioxins in any dental material. However, there might be some possibilities that picogram level of Dioxins may be the result of dental procedures. This is due to the fact that Dioxins are chemicals occurred from the burning of the organocompound in the presence of chlorine. These chemicals are common in dentistry. Moreover, new chemical, i.e., Triclosan, is introduced into the dental material. It was documented that Triclosan phototransforms to Dioxins. More recently, chlorine organocompound was also added into resin composite and bracket adhesive. To be more specific, extensive dental procedures such as preparation and cut of the tooth may accidentally initiate very high heat. The combination of all these possibilities may lead to Dioxins formation.[47-51]

Some Dioxins show a very long half-life of elimination and persist in the environment for a very long period of time. Dioxins accumulate in the fat tissue of living animals; therefore, they accumulate among animals through the food chain. When they reach the toxicity dose, the poisoning results in animals are reduced body weight and toxicity to alimentary, immune, skin, liver and reproductive systems. They also trigger the developmental defect of the fetus. For human, they disturb endocrine, cardiovascular and immune system. They affect the pulmonary function and increase the prevalence of orofacial cleft. They also increase susceptibility to infection and cause neuromotoric disturbance in children. Chloracne is the apparent result of Dioxins as well.[4, 42-43] Since 1997, the International Agency for Research on Cancer (1997) classified TCDD as Class I carcinogen: known human carcinogen.[52]

In general, human exposure to Dioxins occurs through environment and food chain. Moreover, breast milk can transmit Dioxins to baby. Therefore, pregnant and breast feeding women should be cautious about this Dioxins contamination to minimize their accumulation. During a prolonged breast feeding period, a mother can secrete up to 20-30% of her Dioxins body burden to the infant.[42] To summarize Dioxins poisonous severity, Dioxins toxicity is very strong even in a miniscule level. Moreover, the half-life of elimination also is very long. They can be very long lasting as environmental pollutants. In addition, they can be accumulated in animal and passed on by food

chain and breast feeding. Therefore, they are very hazardous chemicals.

Among all several toxicities of Dioxins, effect to developing tooth bud is the most evident and documented in dentistry aspect. This toxic effect is related to the concentration of Dioxins in the breast milk. The most frequently affected tooth is the first permanent molar. This is due to its mineralization period that takes place in the first 1-2 years of age. The result is enamel hypomineralization of this affected tooth. Studies done in healthy Finns who lived in Dioxins highly contaminated environment also showed that children in the area showed mineralization defects.[42, 53-54]

The distinct clinical manifestation of MIH is demarcated opacity defect of the enamel involving at least one permanent molar. MIH opacity may also affect other molars and incisors, however, the first permanent molar is the most obvious. These demarcated opacities show the yellow to brown colors. The affected teeth will be susceptible to caries and hypersensitivity. From the critical review, while other factors showed weak to very weak evidences, Dioxins showed moderate evidences to be implicated as an etiology of MIH.[53]

Moreover, the high amount of Dioxins may cause hypodontia, agenesis, delayed tooth eruption, abnormal root development and cleft palate.[53-55] It is generally accepted that almost all cleft palate patients require a transfer for orthodontic treatment.[56] Developmental defects of the tooth and palate also affect orthodontic treatment planning. Moreover, orthodontist's documentation of radiographs, photographs, dental casts and patient history are relatively good. This led to the researches and publications of developmental defects of tooth and palate in orthodontic journals.[57-58] In addition, when severe MIH especially that of the first molar occurs, extraction of this severely defected tooth in young patients is not uncommon. When denture is not the best option for young patients, dentists will also transfer these young patients to orthodontists.[59-60] Implemented with the opportunity of orthodontists' seeing so many patients during clinical visits, when incorporated with careful comprehensive inspection and documentation, this will lead to the better chance of having systematic diagnoses of these developmental defects of the tooth and oral tissue.

In case there are many children with MIH seen in one community simultaneously, not only orthodontic treatment should be provided, orthodontists should keep in mind that there might be the chance of Dioxins contamination causing MIH in the area. These children might have exposed themselves to Dioxins when their teeth were developing. This high level of Dioxins intake may come from food and environment in the area. Care should be taken to report this possibility to the local environment agency for further investigation and control. Warning to other dentists in the areas may also warrant the best possible patient examination and treatment. Furthermore, introduction of chlorinated organocompound into dental material should also be circumspect. Ingredients of dental materials must be showed clearly, so new additives are noticeable. Dioxins reagent must be well controlled or banned from innovative orthodontic and dental material. In addition, updating Dioxins information will warrant patient's safety. This stimulates orthodontists to be cautious and play an important role in managing the Dioxins control in their community.

Summary

BPA and Dioxins are toxic to human. They are absorbed to human body from the environment and general consumptions. To reduce this contamination,

the recommendations include eating fresh food and fruit instead of canned produce. Avoid plastic container or replace them with glass, ceramic or stainless steel especially to contain hot food. Baby bottles must be BPA free. The use of BPA leachable plastic in microwave oven should not be allowed. Also when dealing with hot food (higher than 60 ºC), heat resistance glass or ceramic should be opted.

Orthodontists should warn the local environmental agency immediately, whenever noticing MIH in many children from the same community. This may be the result of Dioxins pollutant both from food and from environment.

Although BPA and Dioxins levels are considered relatively safe in dental material when they are wisely and cautiously dealt with, to maximize patients' wellbeing, orthodontists should control their toxic chemical used to protect their patients. Some clinical guidelines are therefore established. These guidelines include using BPA free orthodontic materials, avoidance of the use of new material with staple releasing BPA or Dioxins, keeping abreast of BPA and Dioxins information and technology. This is to guarantee that there will be no harm to the patients and people in the community.

References

1. Chen L, Suh BI. Bisphenol A in dental materials: A review. ***JSM Dent*** 2013;1:1004-8.
2. https://ntp.niehs.nih.gov/ntp/ohat/bisphenol/bisphenol.pdf
3. http://www.efsa.europa.eu/en/topics/topic/bisphenol.htm
4. http://www.who.int/mediacentre/factsheets/fs225/en/
5. http://www.plasticsindustry.org/AboutPlastics/content.cfm?ItemNumber=823
6. http://www.astm.org/Standards/D7611.htm
7. http://www.jcpra.or.jp/Portals/0/resource/association/pamph/pdf/law2003_eng09.pdf
8. http://www2.epa.gov/sites/production/files/2014-05/documents/handout-4-awareness.pdf
9. http://203.157.72.106/fulltext2/กฎหมาย/กองควบคุมอาหาร/ประกาศกระทรวงสาธารณสุข/58/ขวดนม.pdf
10. http://www.ncsl.org/research/environment-and-natural-resources/policy-update-on-state-restrictions-on-bisphenol-a.aspx#juris
11. Eliades T, Pratsinis H, Athanasiou AE, Eliades G, and Kletsase D. Cytotoxicity and estrogenicity of Invisalign appliances. ***Am J Orthod Dentofacial Orthop*** 2009;136:100-3.
12. Olea N, Pulgar R, Pérez P, Olea-Serrano F, Rivas A, Novillo-Fertrell A, Estrogenicity of resin-based composites and sealants used in dentistry. ***Environ Health Perspect*** 1996;104:298-305.
13. Gaido KW, Leonard LS, Lovell S, Gould JC, Babaï D, Portier CJ, et al. Evaluation of chemicals with endocrine modulating activity in a yeast-based steroid hormone receptor gene transcription assay. ***Toxicol Appl Pharmacol*** 1997;143:205-12.
14. Pupo M, Pisano A, Lappano R, Santolla MF, De Francesco EM, Abonante S, et al. Bisphenol A induces gene expression changes and proliferative effects through GPER in breast cancer cells and cancer-associated fibroblasts. ***Environ Health Perspect*** 2012;120:1177-82.
15. Soriano S, Alonso-Magdalena P, García-Arévalo M, Novials A, Muhammed SJ, Salehi A, et al. Rapid insulinotropic action of low doses of bisphenol-A on mouse and human islets of Langerhans: role of estrogen receptor ß. ***PLoS One*** 2012;7:e31109.
16. Ropero AB, Alonso-Magdalena P, García-García E, Ripoll C, Fuentes E, Nadal A. Bisphenol-A disruption of the endocrine pancreas and blood glucose homeostasis. ***Int J Androl*** 2008 ;31:194-200. Review.
17. Carwile JL, Michels KB., Urinary bisphenol A and obesity: NHANES 2003-2006. ***Environ Res*** 2011;111:825-30.
18. Wang T, Li M, Chen B, Xu M, Xu Y, Huang Y, et al. Urinary bisphenol A (BPA) concentration associates with obesity and insulin resistance. ***J Clin Endocrinol Metab*** 2012;97:E223-7.
19. Braun JM, Kalkbrenner AE, Calafat AM, Yolton K, Ye X, Dietrich KN, et al. Impact of early-life bisphenol A exposure

on behavior and executive function in children. ***Pediatrics*** 2011;128:873-82.

20. Melzer D, Rice NE, Lewis C, Henley WE, Galloway TS. Association of urinary bisphenol a concentration with heart disease: evidence from NHANES 2003/06. ***PLoS One*** 2010;13:e8673.
21. Lakind JS, Levesque J, Dumas P, Bryan S, Clarke J, Naiman DQ. Comparing United States and Canadian population exposures from National Biomonitoring Surveys: bisphenol A intake as a case study. ***J Expo Sci Environ Epidemiol*** 2012;22:219-26.
22. Teeguarden JG, Hanson-Drury S. A systematic review of Bisphenol A "low dose" studies in the context of human exposure: a case for establishing standards for reporting "low-dose" effects of chemicals. ***Food Chem Toxicol*** 2013;62:935-48.
23. Newbold RR, Jefferson WN, Padilla-Banks E. Long-term adverse effects of neonatal exposure to bisphenol A on the murine female reproductive tract. ***Reprod Toxicol*** 2007;24:253-8.
24. Dairkee SH, Seok J, Champion S, Sayeed A, Mindrinos M, Xiao W, et al. Bisphenol A induces a profile of tumor aggressiveness in high-risk cells from breast cancer patients. ***Cancer Res*** 2008;68:2076-80.
25. Schmalz G, Preiss A, Arenholt-Bindslev D. Bisphenol-A content of resin monomers and related degradation products. ***Clin Oral Investig*** 1999;3:114-9.
26. Atkinson JC, Diamond F, Eichmiller F, Selwitz R, Jones G. Stability of bisphenol A, triethylene-glycol dimethacrylate, and bisphenol A dimethacrylate in whole saliva. ***Dent Mater*** 2002;18:128-35
27. Lewis JB, Rueggeberg FA, Lapp CA, Ergle JW, Schuster GS. Identification and characterization of estrogen-like components in commercial resin-based dental restorative materials. ***Clin Oral Investig*** 1999;3:107-13.
28. Kotyk MW, Wiltshire WA. An investigation into bisphenol-A leaching from orthodontic materials. ***Angle Orthod*** 2014;84:516–20.
29. Gioka C, Eliades T, Zinelis S, Pratsinis H, Athanasiou AE, Eliades G, et al. Characterization and in vitro estrogenicity of orthodontic adhesive particulates produced by simulated debonding. ***Dent Mater*** 2009;25: 376-82.
30. Sajiki J, Yonekubo J. Leaching of bisphenol A (BPA) from polycarbonate plastic to water containing amino acids and its degradation by radical oxygen species. ***Chemosphere*** 2004;55:861-7.
31. Kingman A, Hyman J, Masten SA, Jayaram B, Smith C, Eichmiller F, et al. Bisphenol A and other compounds in human saliva and urine associated with the placement of composite restorations. ***J Am Dent Assoc*** 2012;143:1292–302.
32. Suzuki K, Ishikawa K, Sugiyama K, Furuta H, Nishimura F. Content and release of bisphenol A from polycarbonate dental products. ***Dent Mater J*** 2000;19:389-95.
33. Watanabe M, Hase T, Imai Y. Change in the bisphenol A content in a polycarbonate orthodontic bracket and its leaching characteristics in water. ***Dent Mater J*** 2001;20:353-8.
34. Sunitha C, Kailasam V, Padmanabhan S, Chitharanjan AB. Bisphenol A release from an orthodontic adhesive and its correlation with the degree of conversion on varying light-curing tip distances. ***Am J Orthod Dentofacial Orthop*** 2011;140:239-44.
35. Purushothaman D, Kailasam V, Chitharanjan AB. Bisphenol A release from orthodontic adhesives and its correlation with the degree of conversion. ***Am J Orthod Dentofacial Orthop*** 2015;147:29-36.
36. Kloukos D, Pandis N, Eliades T. Bisphenol-A and residual monomer leaching from orthodontic adhesive resins and polycarbonate brackets: A systematic review. ***Am J Orthod Dentofacial Orthop*** 2013;143:S104-12.
37. http://www.sizethailand.org/region_all.html
38. Eliades T, Pratsinis H, Athanasiou AE, Eliades G, and Kletsase D. Cytotoxicity and estrogenicity of Invisalign appliances. ***Am J Orthod Dentofacial Orthop*** 2009;136:100-3.
39. Warwick R, O'Connor A, Lamey B. Mercury vapour exposure during dental student training in amalgam removal. ***J Occup Med Toxicol*** 2013;3:27.
40. Eisen JJ, LeCompte EJ. A comparison of oral fluoride retention following topical treatments with APF gels of varying viscosities. ***Pediatr Dent*** 1985;7:175-9.

41. Arenholt-Bindslev D. Environmental aspects of dental filling materials. ***Eur J Oral Sci*** 1998;106:713-20. Review
42. Kiukkonen A. Toxicity of dioxin to developing teeth and salivary glands: An experimental study [thesis], Helsinki: University of Helsinki; 2006.
43. Boffetta P, Mundt KA, Adami HO, Cole P, Mandel JS. TCDD and cancer: a critical review of epidemiologic studies. ***Crit Rev Toxicol*** 2011; 41:622-36.
44. https://www.publichealth.va.gov/exposures/agentorange/locations/thailand.asp
45. http://en.wikipedia.org/wiki/Toxic_equivalency_factor
46. http://www.who.int/ipcs/publications/en/exe-sum-final.pdf
47. Altarawneh M, Dlugogorski BZ. Formation of dibenzofuran, dibenzo-p-dioxin and their hydroxylated derivatives from catechol. ***Phys Chem Chem Phys*** 2015;17:1822-30.
48. Chang YC, Tai KW, Huang FM, Huang MF. Cytotoxic and nongenotoxic effects of phenolic compounds in human pulp cell cultures. ***J Endod*** 2000 Aug;26(8):440-3.
49. Sehgal V, Shetty VS, Mogra S, Bhat G, Eipe M, Jacob S, Prabu L. Evaluation of antimicrobial and physical properties of orthodontic composite resin modified by addition of antimicrobial agents--an in-vitro study. ***Am J Orthod Dentofacial Orthop*** 2007;131:525-9.
50. Othman HF, Wu CD, Evans CA, Drummond JL, Matasa CG. Evaluation of antimicrobial properties of orthodontic composite resins combined with benzalkonium chloride. ***Am J Orthod Dentofacial Orthop*** 2002;122:288-94.
51. Al-Musallam TA, Evans CA, Drummond JL, Matasa C, Wu CD. Antimicrobial properties of an orthodontic adhesive combined with cetylpyridinium chloride. ***Am J Orthod Dentofacial Orthop*** 2006;129:245-51.
52. http://monographs.iarc.fr/ENG/Monographs/vol100F/mono100F-27.pdf
53. Crombie F, Manton D, Kilpatrick N. Aetiology of molar-incisor hypomineralization: a critical review. ***Int J Paediatr Dent*** 2009;19:73-83.
54. Alaluusua S, Calderara P, Gerthoux PM, Lukinmaa PL, Kovero O, Needham L, et al. Developmental dental aberrations after the dioxin accident in Seveso. ***Environ Health Perspect*** 2004;112:1313-8.
55. Imura H, Yamada T, Mishima K, Fujiwara K, Kawaki H, Hirata A, et al. Effect of 2,3,7,8-tetrachlorodibenzo-p-dioxin suggests abnormal palate development after palatal fusion. ***Congenit Anom (Kyoto)*** 2010;50:77-84.
56. Freer TJ. Who says that child needs orthodontic treatment? ***Aust Orthod J*** 1975;4:64-70.
57. Al-Amiri A, Tabbaa S, Preston CB, Al-Jewair T. The prevalence of dental anomalies in orthodontic patients at the State University of New York at Buffalo. ***J Contemp Dent Pract*** 2013;14:518-23.
58. Wangsrimongkol T, Manosudprasit M, Pisek P, Chittiwatanapong N. Prevalence and types of dental anomaly in a Thai non-syndromic oral cleft sample. ***J Med Assoc Thai*** 2013;96 Suppl 4:S25-35.
59. Williams JK, Gowans AJ. Hypomineralised first permanent molars and the orthodontist. ***Eur J Paediatr Dent*** 2003;4:129-32.
60. Cobourne MT, Williams A, Harrison M. National clinical guidelines for the extraction of first permanent molars in children. ***Br Dent J*** 2014;217:643-8.
61. Eliades T, Hiskia A, Eliades G, Athanasiou AE. Assessment of bisphenol-A release from orthodontic adhesives. ***Am J Orthod Dentofacial Orthop*** 2007;131:72-5.
62. Arenholt-Bindslev D, Breinholt V, Preiss A, Schmalz G. Time-related bisphenol-A content and estrogenic activity in saliva samples collected in relation to placement of fissure sealants. ***Clin Oral Investig*** 1999; 3:120-5.
63. Watanabe M. Degradation and formation of bisphenol A in polycarbonate used in dentistry. ***J Med Dent Sci*** 2004;51:1-6.
64. Suzuki K, Ishikawa K, Sugiyama K, Furuta H, Nishimura F. Content and release of bisphenol A from polycarbonate dental products. ***Dent Mater J*** 2000;19:389-95.
65. Eliades T, Voutsa D, Sifakakis I, Makou M, Katsaros C. Release of bisphenol-A from a light-cured adhesive bonded to lingual fixed retainers. ***Am J Orthod Dentofacial Orthop*** 2011;139: 192-5.
66. Kang YG, Kim JY, Kim J, Won PJ, Nam JH. Release of bisphenol A from resin composite used to bond orthodontic lingual retainers. ***Am J Orthod Dentofacial Orthop*** 2011;140:779-89.

บทสัมภาษณ์พิเศษ

Special Interview

Interview with Professor Kee-Joon Lee

by Dr. Sasipa Thiradilok

Dr. Kee-Joon Lee is a professor of the Department of Orthodontics, Yonsei University College of Dentistry, Seoul, Korea. He has contributed to many book chapters on biomechanics of miniscrew-driven orthodontics, non-extraction treatment in adults, up-to-date lingual orthodontic mechanics and surgery-first approached using temporary anchorage devices (TADs). He is the first who demonstrated the miniscrew-assisted rapid palatal expander (MARPE) for adults in American Journal of Orthodontics and Dentofacial Orthopedics (AJO-DO), which was cited by many other authors. He has published many articles and case reports regarding the treatment of non-eruption, and total arch movement for hyperdivergent face in orthodontic journals including two cover issues in AJO-DO. His fields in research include clinical biomechanics regarding TADs application and the suture and bone responses to orthodontic stimulus. He has served as a reviewer in major orthodontic journals, including AJO-DO, Angle Orthodontists and European Journal of Orthodontics.

In the occasion of his visit as an invited speaker at the Thai Association of Orthodontists scientific meeting on July 6, 2015, we had an honor to have a conversation with Professor Kee-Joon Lee about interesting topics from his presentation.

Question: Could you please introduce your expertise in orthodontics?

Prof KJL: I'm interested in applied biomechanics (using TADs), lingual orthodontics and related biomechanics, surgery-first approach and related to predictability-based treatment planning, non-surgical treatment related to applied biomechanics, transverse expansion in adults related to suture biology, and

treatment for non-eruption teeth related to eruption biology. I think that the combination of the known biomechanics rules and the underlying tissue biology may be the key to the success in the clinical orthodontic practice. Hence effective translation of the known rules to the orthodontic practice is I think one of the crucial things.

Question How do you conceptualize temporary anchorage devices (TADs)?

Prof KJL: TADs is a stable point of force application apart from tooth segment to secure the anchorage in clinical orthodontics. Variation in the position of the stable anchorage unit allows predictable movement of target segment, ranging from a single tooth to an entire dental arch. Hence in terms of biomechanics, predictable orthodontic treatment without the mini-screw-type TADs is hardly imaginable.

Question: How do you combine mini implants to distalize the whole maxilla?

Prof KJL: I use the Interradicular mini-screws between molars. Since the displacement of the entire arch is dictated by a direct relationship between the center of resistance of the whole arch and the line of action

generated between the mini-screws and force application points at the archwire, I apply the horizontal force vectors on the main archwire.

Question: What is your approach in treating Class III patients?

Prof KJL: The skeletal Class III patients characterized by mandibular prognathism, maxillary deficiency, or both. The growth and development of the mandible should be monitored and the treatment timing should be carefully adjusted.

At the early treatment stage, the maxillary expansion is needed for the eruption guidance especially in the premaxilla area. Hence the rationale of early treatment has more to do with the eruption issue than the actual anteroposterior relation. Then minimal but intensive maxillary protraction should be applied before the patients reach their growth peak. Overcorrection of the maxillary protraction is also recommended. Considering the proliferation potential of the suture cells, extension of the protraction time is not recommended. Instead, six-month intensive protraction may be good enough. Before the growth peak, protraction may be repeated for another six months depending on the residual growth.

After the patients reach their growth spurt, the growth is monitored. The treatment planning either the camouflage treatment, or orthognathic surgery have to be reassessed. Therefore, proper eruption of permanent teeth and the reduction of the possibility of orthognathic surgery are main rationale of the early treatment of Class III.

Question: Would you recommend the use of maxillary expansion in treating Class III patients?

Prof KJL: Yes. I will suggest the maxillary expansion in the case of skeleton Class III patients with retrognathic maxilla. There are two reasons--facilitation of the maxillary anterior eruption and maxillary protraction.

Question: How different is your Kee's bone expander from other expanders?

Prof KJL: Kee's bone expander consists of 4 miniscrews and an expander at the palate. The insertion of Kee's bone expander is direct method. The bone plate is easily contoured and can be customized for individual patient's palate. It is much less time-consuming than the conventional expanders because there is no extra laboratory work. The bone screws fit properly on the surgical plate without additional composite reinforcement. It eliminates the need for consolidation after expansion. Simultaneous leveling and alignment during and/or after palatal expansion is possible, which greatly reduces the treatment time for overall arch development. For instance, in case of moderate canine space deficiency, palatal expansion and space regain can be finished in three to four months of time.

Question: Would you also recommend the use of Kee's bone expander in treating Class III patients?

Prof KJL: Kee's bone expansion is indicated for correcting the transversal maxillary discrepancy. I think that it is not always necessarily to use the Kee's bone expander for every Class III patients since protraction would need additional tooth-borne appliance. Class III patient with maxillary transverse discrepancy is recommended.

Question: Would you please describe "Asymmetrical distalization"?

Prof KJL: Distalization is the orthodontics mechanic means distal movement of the teeth segment and we do know that majority of our cases need certain level of asymmetrical distalization related to asymmetric molar relation. Using the interradicular miniscrews, instead of midpalatal ones, asymmetric distalization is always possible and predictable, using a main arch wire bypass the anterior segment. One may use a segmental archwire instead.

Question: You mentioned in your lecture about "The biological clues concept". Please explain more.

Prof KJL: The concept includes

- The assessment of eruption potential of each tooth
- Early correction of the patients in the proper timing
- Induction of normal eruption by eliminating of mechanical obstacles
- Formation of intrabony environment for maximum root and bone formation within the soft and hard tissue coverage
- The understanding of the behavior of the stem cells is the key

Especially the eruption and jaw growth take place as a consequence of biological programming. 'Reading' the potential biological activity should be the first step of treatment, similar to the treatment of prepubertal patients. By understanding the biological status of each case, conversion from 'challenging case' to an 'easy case' may be the greatest benefit to the orthodontists. Moreover, periodontal ligament, tooth follicles, and the open root apices are known reservoir of abundant stem cells, with which formation of bone, root and periodontal structure is possible. Relocating the cell-rich area within the biologically favorable area (scaffold) at the right time point is the core idea of the biological clues concept.

Question: What is your secret to success in treating embedded tooth?

Prof KJL: We should evaluate the following factors

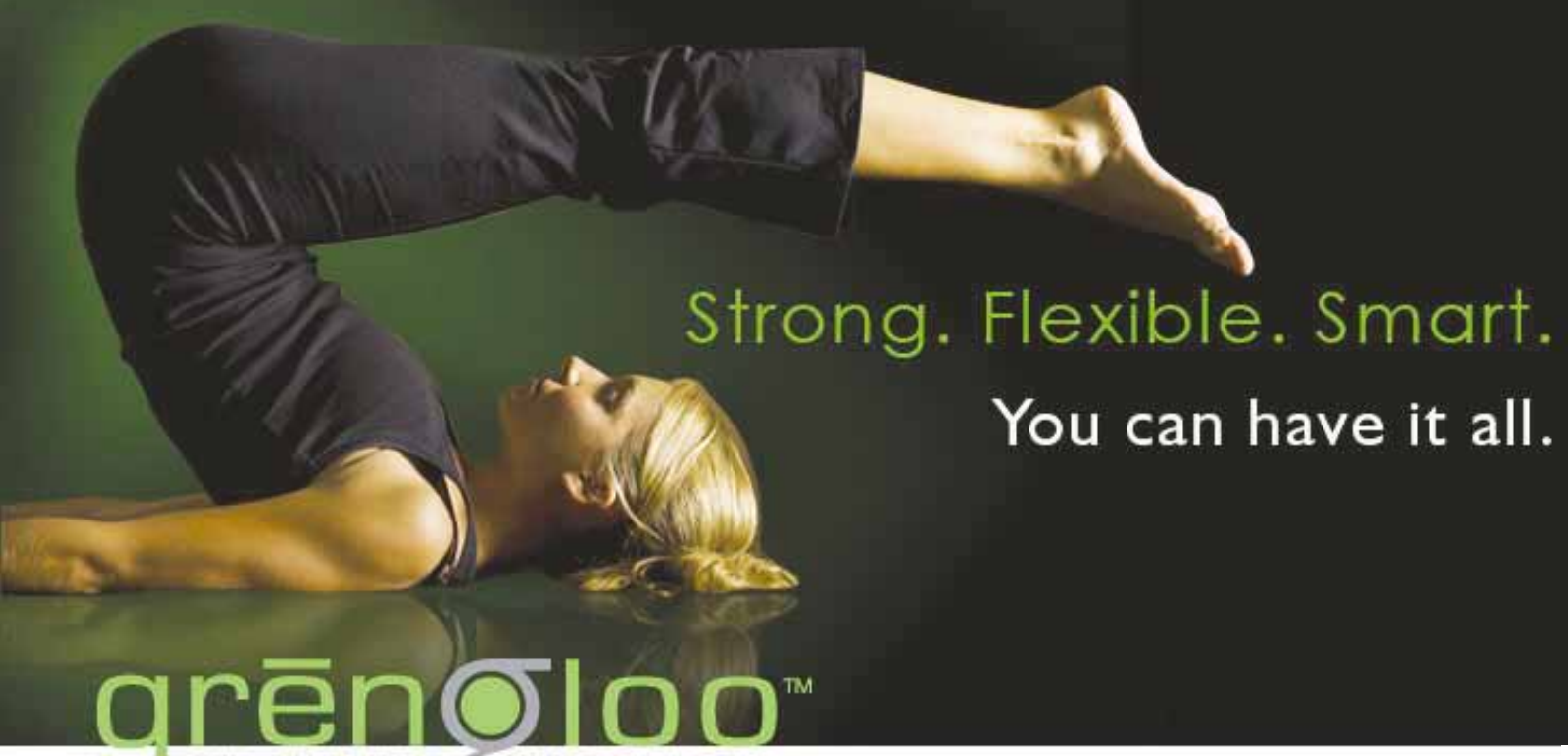
- The eruption potential of each tooth
- The good timing for early correction
- Induction of normal eruption by eliminating of mechanical obstacles
- Formation of intrabony environment for maximum root and bone formation within the soft and hard tissue coverage

Question: What is your future prospective of the 3D digital treatment plan?

Prof KJL: In the future, the 3D treatment planning can help the orthodontists in many ways:

- The final treatment goal should be set from 3D models and the information of every tooth movement will be planned, so as to assess the first 'major step' of the treatment i.e. distalization or major expansion prior to overall alignment.
- The remote-controlled diagnosis can become practical.
- The fabrication of indirect bonding jig, wire and other tools (most are already available in the market).
- The interactive patient and doctor communication in orthodontic diagnosis: between doctors, between doctor and patient.
- Digital archiving of entire orthodontic records.
- Establishment of standardized treatment protocol by visualizing the treatment objectives.
- Finally, quality-controlled treatment 'worldwide' can be realistic, regardless of the location of the orthodontic clinic.

The Online Jounal of the Thai Association of Orthodontists would like to express our sincere appreciation to Professor Kee-Joon Lee for sharing his valuable knowledges and experiences in this interview.



Grengloo Two-Way Color Change Adhesive

Grengloo is the two-way color change adhesive that revolutionizes metal bracket bonding by combining the strength, flexibility and intelligent features you want for the reliability you demand.

Quick cure when you want it, not when you don't

- Provides great working time for better placement under ambient conditions, affording you more flexibility to place and position brackets, remove flash and begin light curing.
- Quick-cure polymerization provides greater shear bond strength at initial force loading. When you're ready to cure, Grengloo's quick-cure initiator cures in as little as 5 seconds with a broad-spectrum curing light (e.g., halogen or CoolBeam™ LED) to quickly deliver remarkable strength while reducing chairtime.
- Fast polymerization, providing a high percent of total bond strength at initial force loading.

Greater impact resistance

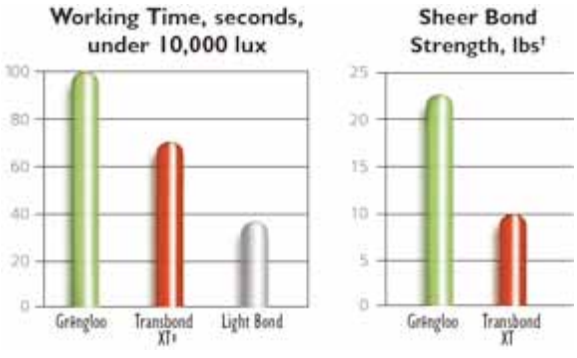
- Provides high level of impact resistance for reduced emergency visits as a result of bond failures caused by traumatic impacts.
- Chemical affinity for metal brackets such as Damon® 3MX and Titanium Orthos™ ensures reliable bond strength, even in the most challenging cases.

Superb handling characteristics

Formula includes a unique hybrid filler material that provides excellent handling characteristics, making cleanup easy and virtually eliminating bracket drift.

Color on demand

- "On-demand" color contrast feature aids in fast and accurate cleanup at bonding and debonding.
- As Grengloo warms to body temperature, the color disappears, remaining clear throughout treatment.
- When debonding, simply introduce a short blast of cool air or water to lower the bonding surface temperature, and Grengloo turns green again for easy and thorough cleanup.



ACCORD CORPORATION LIMITED
A HENRY SCHEIN® COMPANY

จัดจำหน่ายโดย
บริษัท แอคคอร์ด คอร์ปอเรชั่น จำกัด
33/2-8 ซ.รองเมือง 4 แขวงรองเมือง เขตปทุมวัน กรุงเทพฯ 10330 โทร. 0 2119 4900 แฟกซ์: 0 2216 3235
www.accordhenryschein.com

Syringe Kit contents — 740-0320

- 2 Syringes Grengloo (4g), 740-0321
- 1 Bottle Ortho Solo™ (5ml), 740-0271
- 1 Bottle Etching Solution (10ml), 740-0038
- 38 Sealant Applicator Brushes, 740-0041*
- 75 Spatulas, 740-0075