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Gujarat State Branch

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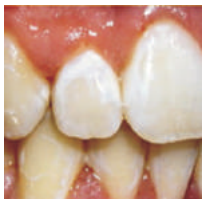
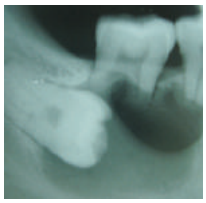
VOLUME -17 (ISSUE : 4 - OCTOBER, NOVEMBER, DECEMBER - 2012)

DENTIMEDIA

JOURNAL OF DENTISTRY

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Guest Editorial

Ethics in dentistry

Skill and Care are two essential requirements of a Good Dental Surgeon. In addition to these qualities Dental Surgeons need have to maintain the highest achievable standard and also update themselves on the technological innovations. If they are involved in academics; they also need to do some Research also to achieve professional success. In our country large number of graduates pass out each year and there is mushrooming of Dental Clinics in Cities whereas large populations in Rural areas are grossly underserved. In this highly competitive scenario where in each dentist strives to ensure that his practice improves, it is very easy to forget the Ethical aspect of dentistry. It is very easy to forget ethics in this competition and we tend to indulge in fraudulent practises and unethical marketing gimmicks. Let us pause and introspect into our professional practise, into ourselves so that we recognise our deficiencies, address our inadequacies and correct ourselves. For those of us involved in academic policy making it is our duty to inculcate classes on ethics into our dental curriculum and ensure that the dentists of the future generation step into this bright world with not just good skills and patient care but also with Good Ethics.

I would also like to wish all the readers a happy new year

Prof.(Dr.)U.S.Krishna Nayak

Past President – Indian Dental Association(H.O.)

Greetings from IDA GUJARAT STATE BRANCH

Dear colleagues,

We thank you for the ever increasing support for the endeavour for the last couple of months. In this editorial we introduce the Doyen of Dentistry Dr. U. S. Krishna Nayak for the guest editorial.

Happy reading & wishing you all Khushi wala New Year.

Jai Hind. Jai IDA.

Yours in fraternity,

Dr. Hemant I. Patel

President

Dr. Nitin Parikh

Hon. State Secretary

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Interactive studies of proteins involved in controlling expression of DSPP gene responsible for Dentinogenesis Imperfecta using bioinformatics tools.

Tanvi Patel[a], Dr Hetalkumar Panchal[b],Dr Jimis Patel[c], Dr. Amish Mehta[d], Dr. Tapan Rawal[e]

Abstract :

Dentinogenesis Imperfecta (hereditary Opalescent Dentin) is a genetic disorder of tooth development. This condition is inherited as an autosomal dominant pattern, which results from mutation in DSPP (Dentin sialophosphoprotein) gene. The product of this gene DSPP is involved in Wnt canonical signaling pathway. DSPP encodes two principal proteins of the dentin extracellular matrix of the tooth, dentin sialoprotein and dentin phosphoprotein. Mutations in this gene have been associated with Dentinogenesis Imperfecta-1 in some individuals. Dentinogenesis Imperfecta occurs in combination with an autosomal dominant form of deafness. From literature, it was clear that mutation in this gene leads to improper formation of proteins required for the normal growth of teeth. We also found, that not only mutations but improper regulation of genes can also lead to its haploinsufficiency which can lead to the disorder. So we have mainly focused on Dspp mRNA expression that is potentiated by the activation of the Wnt canonical signaling pathway. In addition, pharmacological interference with Heparan sulfate sulfation promotes DSPP mRNA expression through activation of Wnt signaling. We also found that Wnt10A (wingless-type MMTV integration site family, member 10A) protein binds to cell surface HSPGs (heparan sulfate proteoglycans) in odontoblasts and interference with HS sulfation decreases the binding affinity of Wnt10A for HSPGs, which facilitates the binding of Wnt10A to its receptor FZD1 (Frizzled-1) and potentiates the Wnt signaling pathway, thereby upregulating DSPP mRNA expression. Thus Sulf (Sulfatase 1/2, enzyme) mediated desulfation of cellular HSPGs is an important modification for the activation of the Wnt signaling in odontoblasts and for production of the dentin matrix to overcome from Dentinogenesis imperfecta.

Key Words : Dentinogenesis Imperfecta, DSPP, WNT, FZD1.

INTRODUCTION

Dentinogenesis imperfecta (hereditary Opalescent Dentin) is a genetic disorder of tooth development. This condition causes teeth to be discolored (most often a blue-gray or yellow-brown color) and translucent. Teeth are also weaker than normal, making them prone to rapid wear, breakage and loss. These problems can affect both primary (baby) teeth and permanent teeth. This condition is inherited in an autosomal dominant pattern, which means one copy of the altered gene in each cell is sufficient to cause the disorder.

Mutations in the DSPP gene have been identified in people with type II and type III dentinogenesis imperfecta. Type I occurs as a part of osteogenesis imperfecta.(http://ghr.nlm.nih.gov/condition/dentinogenesis-imperfecta).

DSPP gene encodes two principal proteins of the dentin extracellular matrix of the tooth. The preproprotein is secreted by odontoblasts and cleaved into dentin sialoprotein and dentin phosphoprotein. Dentin phosphoprotein is thought to be involved in the biomineralization process of dentin. Mutations in this gene have been associated with dentinogenesis imperfecta-1. In some individuals, dentinogenesis imperfecta occurs in combination with an autosomal dominant form of deafness. Allelic differences due to repeated polymorphisms have been found for this gene. We studied the articles, from which we could find out that the gene responsible for Dentinogenesis imperfecta is DSPP. The dentin sialophosphoprotein (DSPP) gene (4q21.3) encodes two major noncollagenous dentin matrix proteins: dentin sialoprotein (DSP) and dentin phosphoprotein (DPP). Defects in the human gene encoding DSPP can cause inherited dentin defects, and these defects can be associated with bilateral progressive high-frequency sensorineural hearing loss. Clinically, five different patterns of inherited dentin defects are distinguished and are classified as

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Submitted, September,2012 ; revised and accepted, October, 2012.

dentinogenesis imperfecta (DGI) types I, II, and III, and dentin dysplasia types I and II. The genetic basis for this clinical heterogeneity is unknown. Among the 11 members recruited from the studied kindred, five were affected with autosomal dominant DGI type II. The mutation (g.1188C G, IVS2-3C G) lies in the third from the last nucleotide of intron 2 and changed its sequence from CAG to GAG. The mutation was correlated with the affection status and was absent in 104 unaffected individuals (208 alleles) with the same ethnic and geological background. The proband was in the primary dentition stage and presented with multiple pulp exposures. The occlusal surface of the dental enamel was generally abraded, and the dentin was heavily worn and uniformly shaded brown. From the literature we studied about the mutations found in DSPP gene that could lead to Dentinogenesis Imperfecta. (Kim. et al).

We found the article which gave us information through which we found that, not only mutations but improper regulation of gene can also lead to its haploinsufficiency, which can lead to the disorder. This study provides molecular evidence for the functional roles of HSPG sulfation and desulfation in dentinogenesis. We saw that odontogenic cells are highly sulfated on the cell surface and become desulfated during their differentiation to odontoblasts, which produce tooth dentin. Sulf1/Sulf2 double null mutant mice exhibit a thin dentin matrix and short roots combined with reduced expression of dentin sialophosphoprotein (Dspp) mRNA, encoding a dentin-specific extracellular matrix precursor protein, while single Sulf mutants do not show such defective phenotypes. In odontoblast cell lines, Dspp mRNA expression is potentiated by the activation of the Wnt canonical signaling pathway. In addition, pharmacological interference with HS sulfation promotes Dspp mRNA expression through activation of Wnt signaling. On the contrary, the silencing of Sulf suppresses the Wnt signaling pathway and subsequently Dspp mRNA expression. We also saw that Wnt10a protein binds to cell surface HSPGs of odontoblasts and interferes with HS sulfation decreasing the binding affinity of Wnt10a for HSPGs, which facilitates the binding of Wnt10a to its receptor and potentiates the Wnt signaling pathway, thereby upregulating Dspp mRNA expression. These results demonstrate that Sulf-mediated desulfation of cellular HSPGs is an important modification which is critical for the activation of the Wnt signaling in

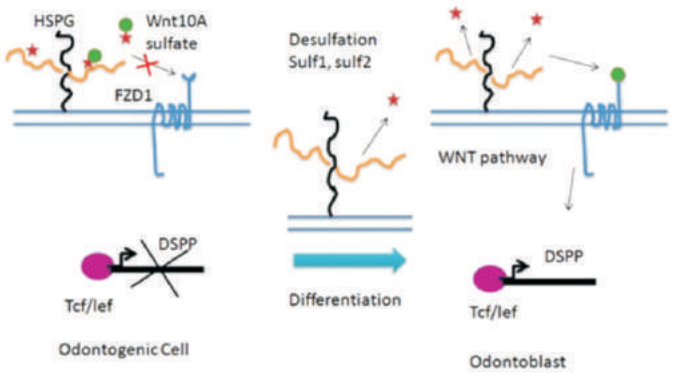


Figure 1: WNT pathway and regulation of DSPP gene.

odontoblasts and for production of the dentin matrix. (Satoru et al).

Materials and Methods :

Sequence retrieval :

The protein sequences of DSPP, FZD1 and Wnt10A were retrieved from one of the largest sequence repository NCBI with sequence ID: NP_055023.2, NP_003496.1, NP_079492.2 respectively.

Protein Structure retrieval:

The Protein 3-D structures of HSPGs was retrieved from the Protein DataBank (PDB) with PDB ID: 1GL4. PDB archive is a repository of atomic coordinates and other information describing proteins.

Protein Structure modeling & Validation

Structure of HSPG2 is obtained from the Protein Databank but structural search of FZD1and Wnt10A proteins suggested that 3-D structures of both the proteins was not present in Protein Databank (PDB). So we modeled the structures of FZD1 and Wnt10A by using Bhageerath H server. The modeled structures of FZD1and Wnt10A are deposited in

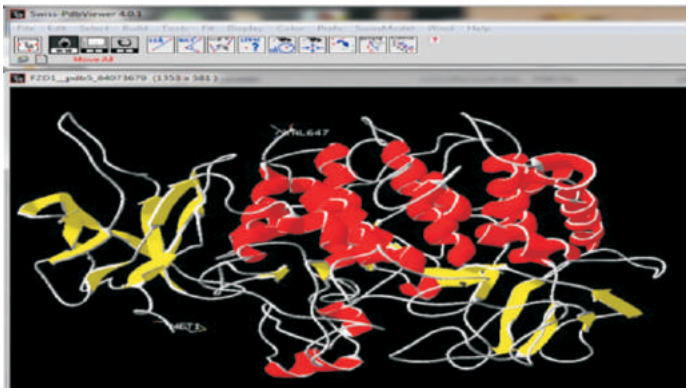


Figure 2: Modeled structure of FZD1 viewed in Swiss PDB viewer with PMID: PM0078031.

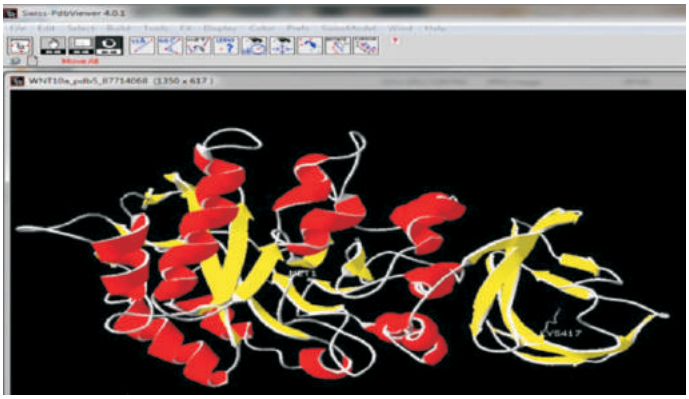
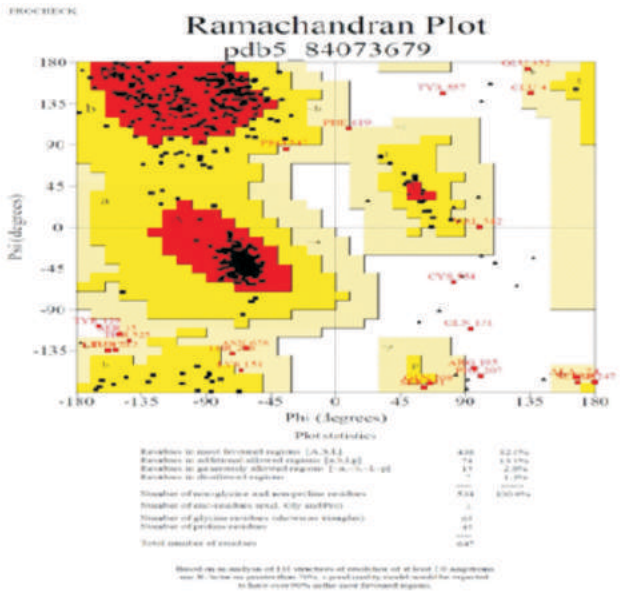


Figure 3: Modeled structure of Wnt10A viewed in Swiss PDB viewer with PMID: PM0078030.

international modeled protein structure repository named Protein Model DataBase (PMDB) with PMID: PM0078031 and PM0078030 respectively.

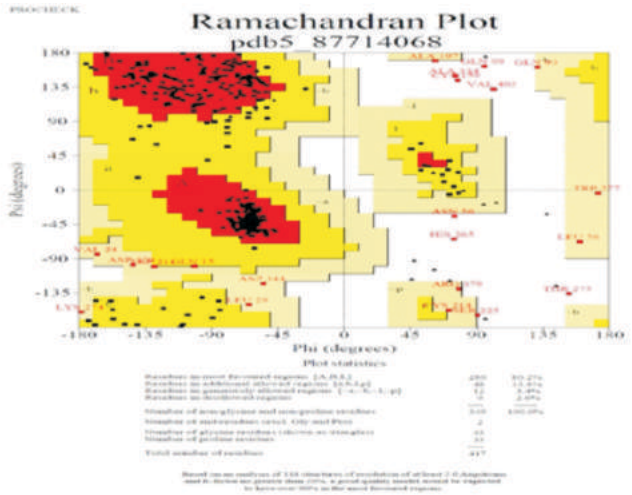
According to Ramachandran plot of modeled FZD1 and Wnt10A proteins we can say that the modeled protein may be considered as of good quality. Structural validation of both the proteins was done using structural validation program PROCHECK available at SAVES server.

Figure 4: Ramachandran plot result of FZD1 with 98.7% of



structure validity, Residues in most favoured region are 438-82%, residues in additionally allowed regions are 74-13.9%, residues in generously allowed region are 15-2.8%, residues in disallowed regions are 7-1.3%.

Figure 5: Ramachandran plot result of Wnt10A with 97.4% of



structure validity, Residues in most favoured region are 280-80.2%, residues in additionally allowed regions are 44-13.8%, residues in generously allowed region are 12-3.4%, residues in disallowed regions are 9-2.6%.

Interaction studies

Interactive studies of various proteins provide important information about the functioning of protein. Thus we have carried out protein-protein interaction of Wnt10A and FZD1 using the PatchDock server. The interaction results of PatchDock server were mined to get best pose of interaction with highest minimum energy using the FireDock program at PatchDock server.

Result

String result for DSPP gene

Dentin sialophosphoprotein; DSP may be an important factor

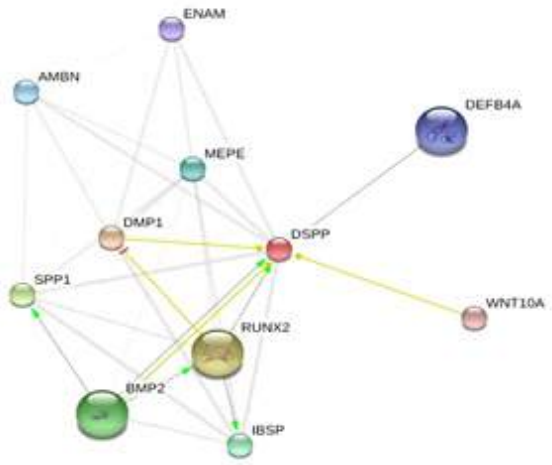


Figure 6: The figure represents the interacting protiens with DSPP from STRING.

in dentinogenesis. DPP may bind high amount of calcium and facilitate initial mineralization of dentin matrix collagen as well as regulate the size and shape of the crystals.

The interaction study of Wnt10A and FZD1 was viewed using PyMOL.

Discussion :

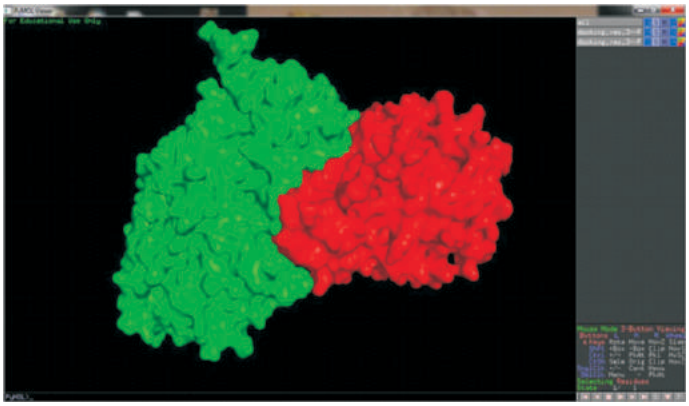


Figure 7: This figure represents interaction and the surface view of the proteins Wnt10A and FZD1.

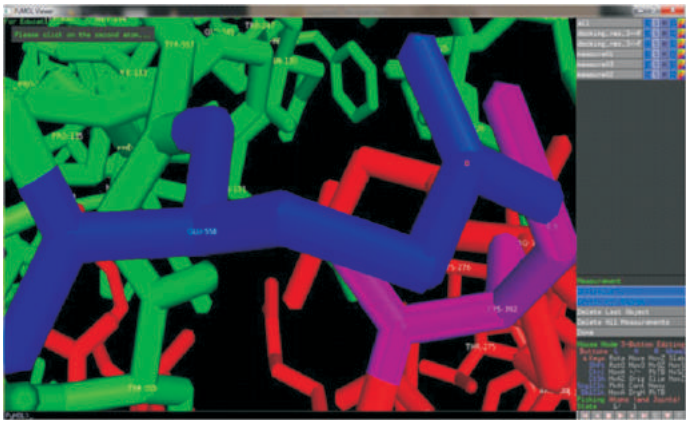


Figure 8: Protein-protein interaction result showing minimum distance by bond length 0.9A◦ between atoms GLU-558 and CYS-392.

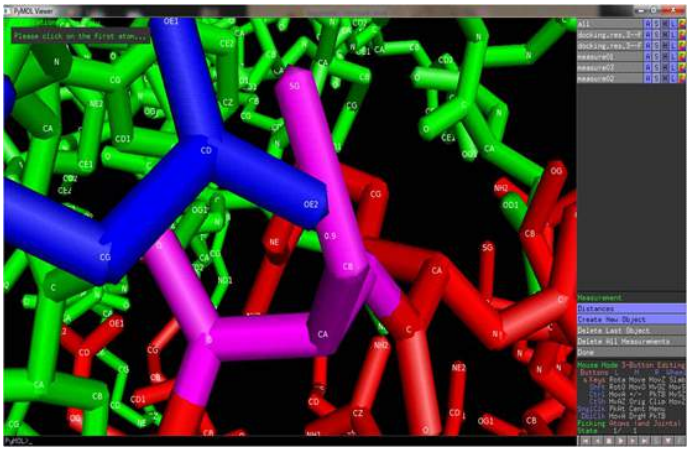


Figure 9: Protein-protein interaction result showing minimum distance by bond length 0.9A◦ between atoms OE2 and CB.

FireDock

[Web Server] [About] [Download] [FAQ] [Help] [References]

Receptor	Ligand
FZD1_84073679.pdb	WNT10a_87714068.pdb

Rank	Solution Number	Global Energy ↓	Attractive VdW	Repulsive VdW	ACE	HB	Structure show/hide
1	3	-59.95	-66.89	44.95	6.31	-7.89	☑
2	6	-45.68	-34.73	22.15	1.83	-1.64	☐
3	5	-36.75	-38.75	30.88	-12.40	-0.70	☐
4	1	-26.43	-61.57	66.00	-2.25	-6.23	☐
5	7	-9.02	-10.04	1.83	0.02	-1.54	☐
6	10	-6.91	-35.08	32.01	6.51	-4.08	☐
7	2	-3.34	-35.68	49.09	5.59	-4.35	☐
8	8	-3.17	-21.77	10.50	2.99	0.00	☐
9	9	3.51	-33.24	30.86	7.12	-2.19	☐
10	4	38.89	-54.04	22.87	7.80	-4.79	☐

Figure 10: The figure represents the global energy of the interactions performed between Wnt10A and FZD1 by FireDock.

FireDock

[Web Server] [About] [Download] [FAQ] [Help] [References]

Receptor	Ligand
1GL4.pdb	WNT10a_pdb5_87714068.pdb

Rank	Solution Number	Global Energy ↓	Attractive VdW	Repulsive VdW	ACE	HB	Structure show/hide
1	9	-26.70	-30.41	21.17	-1.33	-3.89	☑
2	2	-25.65	-31.97	9.94	0.30	-2.39	☐
3	7	-19.07	-26.21	22.78	-2.94	-2.88	☐
4	8	-17.39	-52.19	35.36	9.91	-6.22	☐
5	3	-14.15	-39.89	43.35	7.39	-3.76	☐
6	6	-12.27	-39.46	37.85	0.54	-3.78	☐
7	10	5.71	-19.31	14.97	1.04	-0.59	☐
8	5	26.60	-44.76	35.62	7.92	-4.34	☐
9	1	216.32	-55.46	336.20	11.15	-1.15	☐
10	4	514.00	-49.99	714.10	-5.59	-3.94	☐

Figure 11: The figure represents the global energy of the interactions performed between HSPG (1GL4) and Wnt10A by FireDock.

Dentinogenesis Imperfecta is a genetic disorder of tooth development, gene responsible for Dentinogenesis Imperfecta is DSPP. Many mutations have been found but we could not correct them as we have no proper 3D structure to study the mutations to improve them. From the research done we found that if DSPP gene is not transcribed properly then Dentinogenesis Imperfecta is caused. So we studied the pathway in which DSPP gene is involved to identify target

proteins that need to be modulated to up regulate the DSPP gene expression. Wnt10A and FZD1 are the proteins that can be interacted for the signaling of DSPP gene expression. Protein-protein interaction between Wnt10A and FZD1 proteins was carried out using PatchDock server and we got best pose with minimum global energy with minimum acceptable bond length between atoms OE2-CB and of amino acids GLU-CYS respectively. To check binding affinity of two protein, protein-protein interaction studies between HSPG and Wnt10A was carried out.We found that the binding energy of Wnt10A with HSPG was less than the binding energy of Wnt10A with FZD1. So we can say that the binding of Wnt10A with FZD1 is more favourable than the binding of Wnt10A with HSPG.

Conclusion

From the present insilico study we conclude that the disorder might be cured or some of its major symptoms can be cured by increasing the mRNA levels of DSPP gene by the activation of WNT signaling pathway. We have studied the interactions between the proteins from the WNT pathway and we found that affinity of Wnt10A to bind with FZD1 was higher than Wnt10A binding with HSPG. We can also go for sulfation of HSPG by sulf and increase the binding affinity of Wnt10A to bind with FZD1. Binding of Wnt10A with FZD1 will lead to further activaion of WNT pathway. Which can triger the signaling process of the pathway this will lead to proper signaling of pathway. As a result of this transcription of DSPP gene can be controlled and the mRNA levels of DSPP gene would be increased. Thus there will be increase in gene product to the required level. which is important in mineralization of dentin matrix collagen as well as regulate the size and shape of the crystals.

References

1. Satoru Hayano1, Hiroshi Kurosaka1, Takeshi Yanagita1, Ina Kalus2, Fabian Milz2, Yoshihito Ishihara1, Md. Nurul Islam1, Noriaki Kawanabe1, Masahiro Saito3, Hiroshi Kamioka1, Taiji Adachi4, Thomas Dierks2, and Takashi Yamashiro1 "Roles of Heparan Sulfate Sulfation in Dentinogenesis", February 20, 2012".

2. J.-W. Kim. S.-H. Nam. K.-T. Jang. S.-H. Lee .C.-C. Kim. S.-H. Hahn. J. C.-C. Hu. J. P. Simmer "A novel splice acceptor mutation in the DSPP gene causing dentinogenesis imperfecta type II" , Aug-2004, Human Genetics 115 (3): 248-254.

3. "Entrez Gene: DSPP dentin sialophosphoprotein".

4. Jensen LJ, Kuhn M, Stark M, Chaffron S, Creevey C, Muller J, Doerks T, Julien P, Roth A, Simonovic M, Bork P, von Mering C. "STRING 8-- a global view on proteins and their functional interactions in 630 organisms. Source European Molecular Biology Laboratory, Heidelberg, Germany."

5. Yuval Inbar, Ruth Nussinov1,2 and Haim J. Wolfson* "PatchDock and SymmDock: servers for rigid and symmetric docking Dina Schneidman-Duhovny, School of Computer Science, Raymond and Beverly Sackler Faculty of Exact Sciences and 1Sackler Institute of Molecular Medicine, Sackler Faculty of Medicine, Tel Aviv University, Tel Aviv 69978, Israel and 2Basic Research Program, SAIC-Frederick Inc., Laboratory of Experimental and Computational Biology NCI-Frederick, Building 469, Room 151, Frederick, MD 21702, USA".

Unicystic Ameloblastoma of the Mandible

A case report and review of literature

Dr Nilesh Raval[a], Dr Dhaval N. Mehta[b], Dr Vivek M. Tarsariya[c], Dr Viral Parekh[d], Dr Chintan Modi[e],Dr Sneha Udhani[f]

Abstract :

Ameloblastoma is a true neoplasm of odontogenic epithelial origin. It is one of the most common odontogenic neoplasm reported in frequency of occurrence. Its incidence, combined with its clinical behaviour, makes ameloblastoma the most significant odontogenic neoplasm. Unicystic ameloblastoma (UCA) refers to those cystic lesions that show clinical, radiographic, or gross features of a mandibular cyst, but on histologic examination show a typical ameloblastomatous epithelium lining part of the cyst cavity, with or without luminal and/or mural tumor growth. It accounts for 5-15% of all intraosseous ameloblastomas. A rare case report of unicystic ameloblastoma in a 40-year-old female treated by enucleation of lesion along with involved teeth, and review the literature is discussed here.

Key Words : Ameloblastoma, Dentigerous Cyst, Mandible, Radiolucent, Unicystic Ameloblastoma

INTRODUCTION

The most common tumour of odontogenic origin is ameloblastoma, which develops from epithelial cellular elements and dental tissues in their various phases of development. It is a slow-growing, persistent, and locally aggressive neoplasm of epithelial origin.

Ameloblastomas originate from the epithelium involved in the formation of the teeth: enamel organ, odontogenic rests of malassez, reduced enamel epithelium and odontogenic cyst lining, heterogenic epithelium in other parts, especially the pituitary gland. Presently, it is thought that it is a result of an alteration or mutation in the genetic material of cells.[1]

It is classified clinically into solid, cystic, peripheral, malignant and carcinomatous types. [1,2]

The term Unicystic Ameloblastoma refers to that lesion which shows clinical, radiographic and gross features of jaw cyst but on histologic examination shows typical ameloblastomatous lining part of the cyst cavity with or without luminal &/or mural tumor growth.(4) Unicystic Ameloblastoma also has been referred to as mural ameloblastoma, luminal ameloblastoma, and ameloblastoma arising in dentigerous cysts. The concept of this tumour was first introduced by Robinson and Martinez in 1977. The more common term used to designate these pathological entities in UCA (Unicystic Ameloblastoma).[2,3,4,5]

Here we present a case of mandibular unicystic ameloblastoma associated with impacted third molar in a 40 years female.

Historical Review

Cusack JW (1827) first published a case of an ameloblastoma. But, the detailed histopathological description was first made by Wedl (1853). He called the tumour, "Cystosarcoma or Cystosarcoma Adenoids", but suggested that it could have arisen from a tooth bud or from the dental lamina. Broca (1868) gave the first detailed description of solid/multicystic ameloblastoma, whereas the first histological drawing of ameloblastoma was made by Wagstaffe (1871). The detailed description of ameloblastoma was made by Falksson (1879). Malassez (1885) suggested the name "Epithelioma Adamantin". Derjinsky (1890) suggested the term "Adamantinoma" . Ivey and Churchill (1930) used the name "Ameloblastoma". The first case of Peripheral Ameloblastoma, was made by Stanley and Krough (1959)[6].

Vickers and Gorlin, in 1970, first described the features of the early ameloblastic change that occur within a wall of cyst. Robinson and Martinez in 1977, is reported to have a less aggressive biologic behaviour and lower recurrence rate than

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the classic solid or multicystic ameloblastoma.[5] WHO (1992) has described Ameloblastoma as "a benign, locally aggressive, polymorphic neoplasm, which is presumably derived from the intraosseous remnants of the odontogenic epithelium."(6)

In 1988, In a clinicopathologic study of 57 cases of unicystic ameloblastoma, Ackermann classified this entity into the following three histologic groups(1,2,4):

Group I: Luminal UA (tumor confined to the luminal surface of the cyst)

Group II: Intraluminal/plexiform UA (nodular proliferation into the lumen without infiltration of tumor cells into the connective tissue wall), and

Group III: Mural UA (invasive islands of ameloblastomatous epithelium in the connective tissue wall not involving the entire epithelium).

Another histologic subgrouping by Philipsen and Reichart has also been described(1):

Subgroup 1: Luminal UA

Subgroup 1.2: Luminal and intraluminal

Subgroup 1.2.3: Luminal, intraluminal and intramural

Subgroup 1.3: Luminal and intramural

Case Report

A 40 year old lady came to the Karnavati school of dentistry, Uvarsad, Gandhinagar, Gujarat with pain & slowly growing swelling on the lower right back teeth region since 8 months. There was difficulty in opening the mouth, and little difficulty in chewing.

On Extraoral examination, we noticed a swelling measuring 3x2 cm in lower 1/3rd of face which extends from right parasymphysis region to right angle of mandible with overlying normal skin. On palpation, the swelling was non-tender & firm in consistency (figure 1). Right submandibular lymphnodes were enlarged, palpable, non-tender & mobile. Intraorally, we noticed a swelling in right side posterior mandibular region originating from alveolar mucosa and obliterating buccal vestibule from mesial to 46 & upto distal to 47. The overlying mucosa of the swelling was normal. Clinically, all third molars were absent. On palpation, swelling was non tender & soft in consistency with crepitus buccally in 47 region (figure 2). No mobility noted in 46 & 47. On percussion, tenderness present with 46 & 47. No mobility noted in 46 or 47 on either inspection or palpation.

A provisional diagnosis of Dentigerous Cyst with impacted 48 was made.

Vitality test with electric pulp tester of 46 & 47 showed delayed response compared to that on the contralateral side. An IOPA & an orthopantomogram (OPG) were advised, which showed a well defined pericoronal radiolucency of approximately 3x2 cm in size with horizontally impacted third molar, well corticated borders and knife edge external root resorption of both roots of 47 & distal root of 46 (figure 3&4).

On the basis of clinical and radiographic features clinicoradiographic diagnosis of Unicystic Ameloblastoma was made with differential diagnosis of dentigerous cyst involving right mandible.

The lesion was enucleated along with removal of involved teeth 46,47,48. The specimen size was 4.5 x 3 cms which showed soft tissue attatched to cementoenamel junction of impacted third molar and knife edge external root resorption of both roots of 47 & mesial root of 46.(figure 5&6) The specimen was sent for histopathologic examination which showed classical features of unicystic Ameloblastoma (figure 7). Patient was advised for regular follow-up to avoid recurrence of lesion.

Discussion

Ameloblastoma is a true neoplasm of the enamel organ type tissue which does not undergo differentiation to the point of enamel formation. Various synonyms which are used for ameloblastoma are Adamantinoma, Adamantoblastoma, Epithelioma Adamantin, Multilocular Cyst, Adontomes embryolastiques and Epithelial odontoma(6).

Multicystic ameloblastoma is the most common variety and represent 86% of cases. The relative prevalence and incidence of UCAs have been reported as between 6-15% of all types of ameloblastomas(3). UCAs are more commonly seen in younger patients, with 50% of cases being diagnosed during the second decade of life. The average age in one large series was found to be 23 years. The gender distribution shows a slight male predilection with a male:female ratio of 1.6:1. However, when the tumor is not associated with an un-erupted tooth, the gender ratio is reversed to a male to female ratio of 1:1.8[6]. We reported a case of UCA in female patient of middle age group.

UCA can be divided into 2 categories(6,7): Dentigerous variant (Histologically verified UCAs which are associated with an unerupted tooth) & Nondentigerous variant (UCAs lacking an association with an unerupted tooth). Clinically, UCA presents as a localized swelling, with occasional pain and signs of lip numbness. In cases of secondary infection, discharge or drainage can be noted. The location of UCA within the jawbone shows a marked predominance for the mandible, irrespective of the variant. The ratio of the maxilla: mandible is 1:7 for the dentigerous variant, versus 1:4.7 for the nondentigerous type(6). In the present case, UCA present dentigerous variant associated with pain and localised swelling in posterior mandible.

Radiographically, UCAs have been divided into 2 main patterns: Unilocular and Multilocular. UCAs have clear preponderance for the uniclocular pattern(6). Our case also supports this finding.

Some investigators believe that UCA arises from pre-existing odontogenic cysts, in particular a dentigerous cyst, while others maintain that it arises de novo. Robinson and Martinez (1997) argued that as the epithelium of odontogenic cysts and ameloblastomas have a common ancestry, a transition from a nonneoplastic to a neoplastic one could be possible, even though it occurs infrequently.

Leider AS et al (1985) proposed three pathogenic mechanisms for the evolution of UCA:

1.The reduced enamel epithelium which is associated with a developing tooth undergoes ameloblastic transformation with subsequent cystic development

2. Ameloblastomas arise in dentigerous cysts or in others in which the neoplastic ameloblastic epithelium is preceded temporarily by a non-neoplastic stratified squamous epithelial lining.

3. A solid ameloblastoma undergoes cystic degeneration of the ameloblastic islands, with subsequent fusion of multiple microcysts and develops into unicystic lesions[2,6].

Various treatments are advised by different authors as per nature of lesion (agrresive & proliferative growth). They are either enucleated, if locally aggressive lesion and radical resection, if more aggressive and showing intramural growth. Chemical cauterization with carnoy's solution is also advised in some studies.[1,6] In our case, due to locally aggressive lesion, it was treated by enucleation along with removal of involved 46,47,48. Frequent follow-up advised as the recurrence rate for UCAs after conservative surgical treatment (curettage or enucleation) are generally reported to be 10-20%, and on average, less than 25%.

Conclusion

Ameloblastoma is the most common odontogenic neoplasm. It presents with a numerous variety of clinical, radiographical and histopathological features. UCA, a type of Ameloblatoma, too presents with a variety of clinical, radiological and histopathological features. Hence, it presents as a challenge both for its diagnosis and treatment. There is always an on-going debate regarding the origin of Unicystic Ameloblastoma. Immunohistochemical studies help us to know the nature of the lesion and also to differentiate the same from other cysts of odontogenic origin. Hence, it is essential that studies should be conducted on a large scale in order to know the origin and nature of the lesion.

References

1. Rakesh S Ramesh, Suraj Manjunath, Tanveer h Ustad, Saira Pais, K Shivakumar: Unicystic ameloblastoma of the mandible an unusual case report and review of literature. J of Head & Neck Oncology 2010, 2:1-5
2. Mohammad Yunus, Nahidullah Baig, Anwar Ul Haq et al: Unicystic Ameloblastoma: A distinct clinicopathologic entity. Pakistan Oral & Dental Journal Vol 29, No. 1:9-12, (June 2009)
3. Satosh ITO, Toshiko MANDAI, Kosci ISHIDA, Naoya KITAMURA, Hiroyo DEGUCHI, Tsuyoshi HATA, Isao IREI, Masaru HOSODA: Unicystic ameloblastoma of the maxilla: A case report, Kawasaki Medical Journal 35(1): 95-98, 2009
4. Paikkatt V.J., Sreedharan S., Kannan V.P.: Unicystic ameloblastoma of the maxilla:

   A case report, J Indian Soc Pedod Prev Dent- June 2007:106-110
5. Yavagal C, Anegundi R, Shetty S: Unicystic plexiform ameloblastoma: an insight for pediatric dentists, J Indian Soc Pedod Prev Dent- Jan-Mar 2009:Issue 1: Vol 27:70-74

6. Rastogi S, Nijhawan S, Modi M, Kumar A, Aslam N, Latheef F: a large mixed radiolucent-radiopaque lesion in the mandible- A diagnostic approach: Journal of Clinical and Diagnostic Research:2010 April: (4): 2300-2307

7. Anton Dunsche, Ortwin Babendererde, Jutta Luttges, Ingo N.G. Springer: Dentigerous Cyst versis Unicystic Ameloblastoma- differential diagnosis in routine histology: J Oral Path Med 32: 486-91

8. Sivapathasundharam B, Einstein A: Unicystic ameloblastoma with presence of dentin: Indian j Dent Res 18(3): 2007: 128-130

List of Figures:

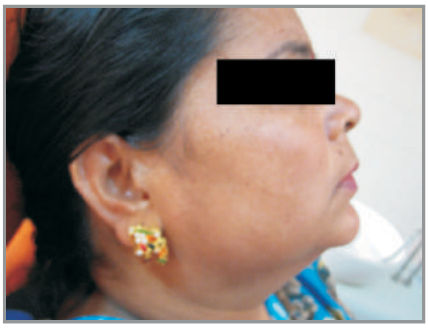

Figure 1: Photograph showing extraoral swelling on the right side of the face.

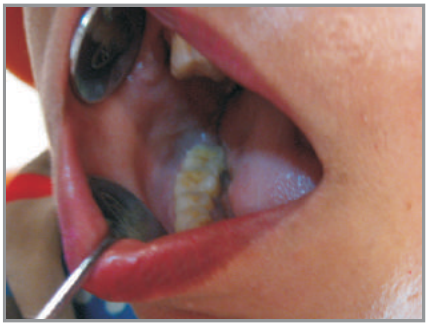

Figure 2: Photograph showing intraoral swelling which is obliterating the buccal vestibule.

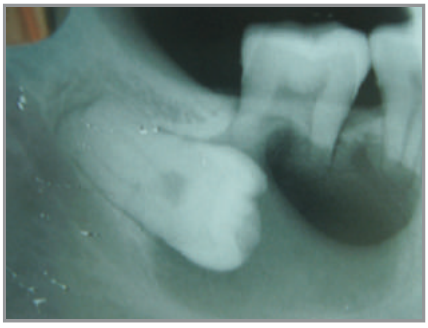

Figure 3: Photograph showing IOPA of mandibular right side showing a well defined pericoronal radiolucency of 3x2 cm in size with horizontally impacted 48 with well corticated borders and knife edge external root resorption of both roots of 47 and distal root of 46.

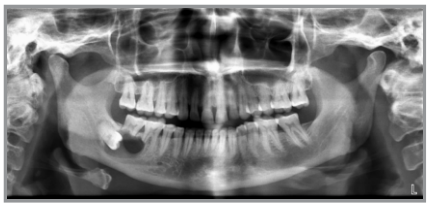

Figure 4: Photograph showing OPG showing teeth central incisor to second molar in all quadrants and a well defined pericoronal radiolucency of 3x2 cm in size with horizontally impacted 48 with well corticated borders and knife edge external root resorption of both roots of 47 and distal root of 46.

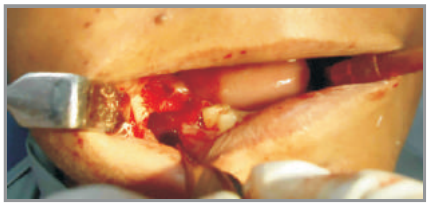

Figure 5 : Photograph showing surgical removal (enucleation) of the lesion.

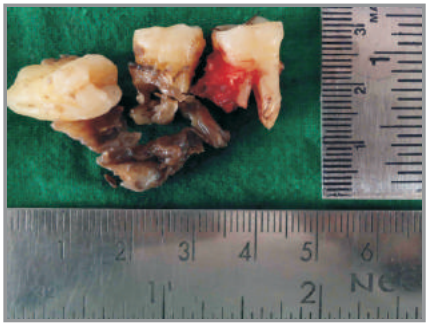

Figure 6: Photograph showing specimen of lesion.

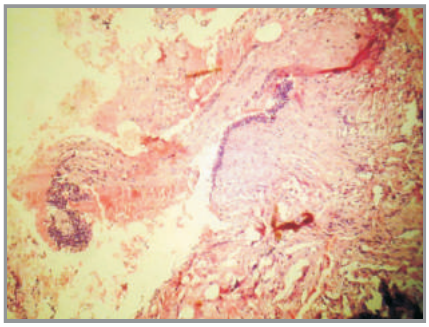

Figure 7: Photograph showing histopathologic features of unicystic ameloblastoma.

White Spot Lesion In Orthodontic Practice: A Contemporary Review

Dr Hiral Savani[a]

Abstract :

The demineralization of enamel adjacent to orthodontic brackets is a significant clinical problem. White spot lesions develop as a result of prolonged plaque accumulation on the affected surface, commonly due to inadequate oral hygiene. To prevent development of white spot lesion orthodontist should assess each patient's risk factor before and during treatment. Oral hygine instruction is important but patient might need additional measures, including fluoride varnish, chlorhexidine, xylitol. This article reviews the relevant historical and contemporary literature regarding methods available to prevent the formation of these white spot lesions during orthodontic treatment.

Key Words : White Spot Leasion (wsl), Fluoride, Chlorhexidine,

INTRODUCTION

The demineralization of enamel adjacent to orthodontic brackets is a significant clinical problem. White spot lesions develop as a result of prolonged plaque accumulation on the affected surface, commonly due to inadequate oral hygiene. It has been reported that there is a significant increase in the prevalence and severity of enamel demineralization after orthodontic treatment1. Once active orthodontic treatment has been completed, the demineralization process is normally expected to decelerate due to a change in local environmental factors. Some white spot lesions may remineralize and return either to normal or at least to a visually acceptable appearance. However, white spot lesions may also persist, resulting in an aesthetically unacceptable result.

To prevent development of white spot lesion orthodontist should assess each patient's risk factor before and during treatment. Oral hygine instruction is important but patient might need additional measures, including fluoride varnish, chlorhexidine, xylitol.

Overall management of white spot lesions involves consideration of methods of preventing demineralization and also methods of encouraging remineralization of existing lesions. In addition to regular professional oral hygiene visits and the application of appropriate preventive medicaments, successful preventive strategies involve oral health promotion, patient education and patient compliance. This article reviews the relevant historical and contemporary literature regarding methods available to prevent the formation of these white spot lesions during orthodontic treatment.

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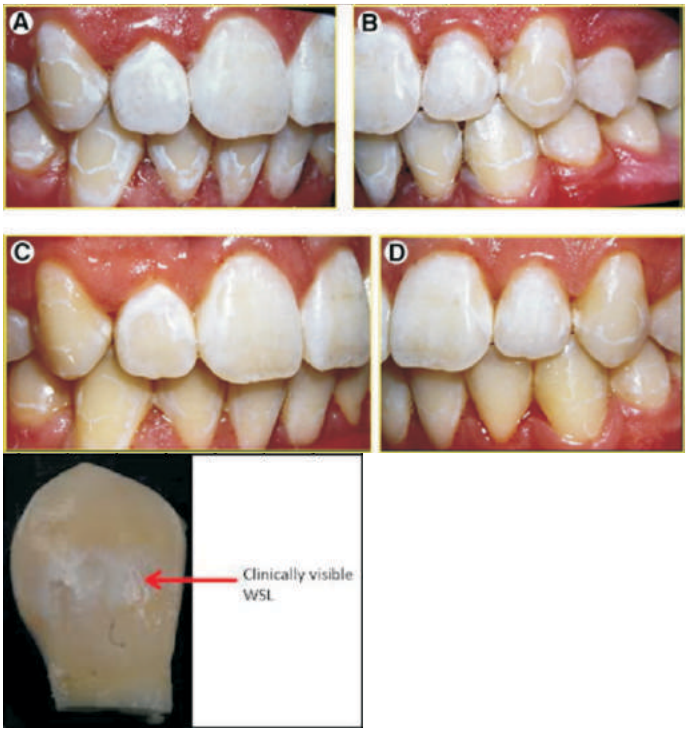
Etiology :

WSL (white spot leasion) are areas of demineralized enamel that develops because of prolonged accumulation of plaque. It develops as a result of a dietary carbohydrate and saliva modified bacterial infection, resulting in an imbalance between demineralization and remineralization of the enamel.2 The irregular surface of band and brackets limit the naturally occurring self cleansing mechanism of oral musculature and saliva and encourage plaque accumulation. Over a time this results in active white spot lesion and if not treated can develop cavitated carious lesion.

The white appearance of early enamel caries is due to an optical phenomenon which is caused by mineral loss in the surface or subsurface enamel. Enamel crystal dissolution begins with subsurface demineralization, creating pores between the enamel rods. The resultant alteration of the refractive index in the affected area is then a consequence of both surface roughness and loss of surface shine and alterations in internal reflection, all resulting in greater visual enamel opacity, as porous enamel scatters more light than sound enamel[3].

Strategies to prevent white spot lesion during orthodontic treatment:

Depending upon patient's risk factors number of agents and therapy can be applied: fluoride tooth paste, varnishes, mouth rinse, antimicrobial, diet counseling, xylitol gums and casein derivatives.



1) Fluoride administration:

The fact that fluoride can be integrated into the crystalline lattice of dental enamel resulting in a structure that is more resistant to the onset of dissolution provides the scientific basis for its use in caries prevention.[4] Fluoride ions can be incorporated into the hydroxyapatite structure of tooth enamel by the replacement of hydroxy groups or by the redeposition of dissolved hydroxyapatite as less soluble fluoridated forms, such as fluorapatite or fluorhydroxyapatite. Calcium fluoride is the major reaction product of topical fluoride treatment of enamel, and it has been found to play a significant role in the cariostatic mechanism of fluoride. Calcium fluoride may persist in dental plaque as calcospherites on the enamel surface for several weeks after a topical application, having the potential to be incorporated into the crystal lattice as fluorapatite during pH cycling within the plaque.[3] During orthodontic treatment, fluoride can be administered to the teeth in various ways, including topical (fluoridated toothpaste, mouthrinse, gel and varnish) and adhesive (fluoride-releasing cements and elastomeric modules and chains) methods.

a) Fluoride tooth paste, mouth rinse and gel :

In a recent systematic literature review evaluating the effectiveness of fluoride in preventing white spot lesion development during orthodontic treatment, it was shown that the use of daily sodium fluoride mouthrinse or the use of glass ionomer cement for bonding brackets can reduce the severity of enamel demineralization surrounding orthodontic appliances.[5] Other fluoride delivery methods which have been reported to reduce the demineralization of enamel surrounding orthodontic brackets include the daily use of toothpastes and/or gels with a high fluoride concentration (15005000ppm) or fluoride toothpaste in combination with chlorhexidine mouthwash.

Recently, it was suggested that individuals undergoing orthodontic treatment should brush twice daily with a 5000ppm fluoride dentrifice. This regime was reported to provide much greater prevention than the daily use of 1000ppm fluoride toothpaste in combination with the daily use of a 500ppm sodium fluoride rinse.[6]

Fluoride mouth rinse can add an extra exposer of fluoride and might be beneficial to some patient, but there is no strong evidence that fluoride mouth rinse can effectively prevent or reduce severity of white spotlesion during orthodontic treatment. The method of fluoride delivery is important, mouth rinse will work best if they are used daily by patient during treatment.

b) Fluoride varnishes:

The professional application of fluoride varnish (5% sodium fluoride in an alcohol suspension of natural resins, approximately 22,000ppm) is a preventive method requiring little patient compliance only attendance at the dental practice. In addition to the fluoride mechanisms mentioned previously, the application of a fluoride varnish provides a protective coating over the tooth surface which decreases enamel solubility[7]. Fluoride varnish adheres to the enamel surface longer than other topical fluoride products and has been shown to be superior to the use of sodium fluoride and monofluorophosphate toothpastes. Overall, the efficacy of regular application of fluoride varnish appears to reduce lesion formation on bracketed maxillary incisor teeth.[7,8]

In case of poor patient compliance with using preventive

protocols at home, it would be advantageous to apply fluoride varnish more than 2 times a year.

2) Casein phosphopeptide-amorphous calcium phosphate (CPP-ACP):

The application of product containg CPP-ACPmight help to prevent enamel demineralization. The role of CPP-ACP in decreasing the incidence of dental caries in the community is anticipated to be additive to the beneficial effects of topical fluoride.

The proposed anticariogenic mechanism of CPPACP involves the incorporation of the nanocomplexes into dental plaque and onto the tooth surface, thereby acting as a calcium and phosphate reservoir. Studies have shown that CPP-ACP incorporated into dental plaque can significantly increase the levels of plaque calcium and phosphate ions.[9,10] This mechanism is ideal for the prevention of enamel demineralization as

there appears to be an inverse association between plaque calcium and phosphate levels and measured caries experience.[10] The localized CPP-ACP nanocomplexes subsequently act to buffer free calcium and phosphate ions in the plaque fluid, in order to maintain a state of supersaturation of ACP with respect to enamel mineral, thereby limiting enamel demineralization and enhancing remineralization. In addition, immunolocalization studies have revealed that CPPACP can be incorporated into supragingival dental plaque by binding to the surfaces of bacterial cells, to components of the intercellular plaque matrix and to adsorbed macromolecules on the tooth surface. All these interactions may then lead to the formation of a less cariogenic plaque.

CPP-ACP has been incorporated into various products in order to exert a topical effect. These products include commercially available sugar-free chewing gum (Recaldent™; Japan and Trident White), mints (Recaldent Mints™; Cadbury Japan Ltd., Japan), topical gels (Tooth Mousse™; GC Corp.,Japan) and experimentally-tested sports drinks and glass ionomer cements.[9,10]

3) Argon-laser enamel surface attenuation:

The results of recent studies would suggest that argon laser may be used to prevent enamel decalcification by altering the crystalline structure of enamel.[11,12] It has been reported that enamel exposure to argon laser irradiation results in the alteration of the surface characteristics of the enamel by creating microspaces that stabilize ions during an acid attack rather than allowing them to be lost from the enamel.[12] The available calcium, phosphate and fluoride ions in saliva may then precipitate into these microspaces, increasing the resistance of the enamel to demineralization and increasing the uptake of minerals from saliva.[53,54] Further in vivo and in vitro studies are required in order to establish the optimal fluence (energy density) for argon laser administration in order to simultaneously prevent enamel decalcification and achieve curing of bonding cements.[12]

Prognosis of white spot leasion:

White spot lesion that develop during fixed appliance orthodontic treatment appear slightly supragingivally or surrounding brackets. Usually the surface is dull, irregular and pitted where the demineralization process has occurred, and plaque signifies an active lesion. A flat or shiny white and sometimes brown surface occurs when the remineralization process has started or completed, signifying arrested lesion.

Active WSL have a better prognosis to recover the translucency of the enamel than arrested WSL because of their porosity and therefore easier incorporation of calcium ions. Arrested lesions have a tendency to result in less favorable esthetic result, because of lack of enamel porosity.

Treatment protocol after orthodontic treatment:

If white spot lesion occur during treatment, it is advisable to first allow the teeth to remineralized naturally. Within 1st week after debonding, there is usually a significant natural reduction of WSL size by remineralization. Fluoride must not be used in high concentration because it can arrest reminaralization and lead to staining. Low concentration of fluoride might assist in reminaralization.

After the natural reminaralization process is allowed to happen, external bleaching might be option to help camouflage WSL and obtain better esthetic result for the patient. For severe cases, acid micro abrasion is recommended when the esthetic result after external bleaching therapy are not satisfactory.

Lastly, aggressive restorative treatment such as direct or indirect veener can be consider if patient still sees the need for further esthetic improvement. No studies addresses, when to determine more aggressive restorative treatment only for

esthetic purpose to cover white spot lesion.

CONCLUSIONS

The development of white spot lesions during fixed appliance orthodontic treatment is preventable. The chosen method or methods for prevention will be largely dependent on the individual needs of each patient and the opinion of the clinician. It is therefore necessary to universally promote the need to maintain a high standard of oral hygiene and to reduce daily exposure to refined carbohydrates throughout the treatment period. In addition, the continuous presence of fluoride in both saliva and plaque, even in low concentrations, is necessary for maximum caries inhibition. The need to prescribe an additional topical fluoride will be dependent upon the needs of the individual patient and clinical judgment.

The ability of CPP-ACP to prevent white spot lesion formation has not, as yet, been proven.

References:

1. Australian dental journal (2006)Prevention of white spot lesions in orthodontic practice: a contemporary review. TR Sudjalim,* MG Woods,† DJ Manton‡
2. Chang HS, Walsh LJ, Freer TJ. Enamel demineralization during orthodontic treatment. Aetiology and prevention. Aust Dent J 1997;42:322-327.
3. Ogaard B. Prevalence of white spot lesions in 19-year-olds: A study on untreated and orthodontically treated persons 5 years after treatment. Am J Orthod Dentofacial Orthop 1989;96:423-427.
4. Ten Cate JM. Current concepts on the theories of the mechanism of action of fluoride. Acta Odontol Scand 1999;57:325-329.
5. Benson PE, Shah AA, Millett DT, Dyer F, Parkin N, Vine RS.

   Fluorides, orthodontics and demineralization: a systematic review. J Orthod 2005;32:102-114.
6. Alexander SA, Ripa LW. Effects of self-applied topical fluoride preparations in orthodontic patients. Angle Orthod 2000;70:424-430.
7. Demito CF, Vivaldi-Rodrigues G, Ramos AL, Bowman SJ. The efficacy of a fluoride varnish in reducing enamel demineralizationadjacent to orthodontic brackets: an in vitro study. Orthod Craniofac Res 2004;7:205-210.
8. Ogaard B, Larsson E, Henriksson T, et al. Effects of combined application of antimicrobial and fluoride varnishes in orthodontic patients. Am J Orthod Dentofacial Orthop 2001;120:28-35.
9. Reynolds EC. Remineralization of enamel subsurface lesions by casein phosphopeptide-stabilized calcium phosphate solutions. J Dent Res 1997;76:1587-1595.
10. Shen P, Cai F, Nowicki A, Vincent J, Reynolds EC. Remineralization of enamel subsurface lesions by sugar-free chewing gum containing casein phosphopeptide-amorphous calcium phosphate. J Dent Res 2001;80:2066-2070.
11. Elaut J, Wehrbein H. The effects of argon laser curing of a resin adhesive on bracket retention and enamel decalcification: a prospective clinical trial. Eur J Orthod 2004;26:553-560.
12. Anderson AM, Kao E, Gladwin M, Benli O, Ngan P. The effects of argon laser irradiation on enamel decalcification: An in vivo study. Am J Orthod Dentofacial Orthop 2002;122:251-259.

LASER IN ORTHODONTICS : An Overview

Dr. Kaushal Shah[a], Dr. Amish Mehta[b], Dr Parul Gupta[c]

Abstract :

It has been emphasized that one of the most valuable treatment objectives in dental practice is to afford the patient a pain-free treatment. With the evolution of the laser applications, the dental committee aimed to achieve this goal without analgesic drugs and painful methods. Orthodontic treatment shows these concerns, that one of the major reason of patient to reject this treatment is the pain accompanied during the different treatment phases. Another great concern of the patient is not being able to get through prolonged periods of treatment. The (GaAlAs) low-level laser is considered to be an effective tool during orthodontic treatment, as the rate of tooth movement is raised significantly, and the pain level is reduced significantly.



Key-words: Low level laser, orthodontic tooth movement

INTRODUCTION

L*aser* is an acronym for **"light amplification by stimulated emission of radiation."** A laser is a single wavelength (or color) of light traveling through a collimated tube delivering a concentrated source of energy.

Most elements in the periodic system (atoms, gases, organic molecules, diodes, chemicals, or electrons)can be used as a media to develop a laser beam.[1]

In 1960, the first laser to use visible light (using a ruby medium) was developed by physicist Theodore H.Maiman,[2] after the theoretical work of Einstein, Basov, Prokhorov, and Townes.[1] In 1968, carbon dioxide was used to perform the first soft-tissue surgery. In 1997, the US Food and Drug Administration approved the erbium laser for hard-tissue surgery. The next year, the first diode laser with a medium of gallium, aluminum, and arsenide was approved for soft-tissue surgery.[3]

A laser offers numerous advantages over a traditional scalpel surgery. Soft-tissue excision is more precise with a laser than a scalpel.[4]

A laser coagulates blood vessels, seals lymphatic, and sterilizes the wound during ablation, maintaining a clear and clean surgical field.[5] Laser surgery is routinely performed by using only topical anesthetic, which is particularly beneficial in an open orthodontic clinic.[6]

There is markedly less bleeding (particularly for frenal surgery), minimal swelling and no need for irritating sutures or unsightly periodontal dressing.[7] A report suggested that laser excisions produce less scar tissue than conventional scalpel surgery,[8] although contrary evidence also exists.[9,10]

Post surgically, patients report less discomfort and fewer functional complications (speaking and chewing), and require fewer analgesics than do patients treated with conventional scalpel surgery.[7]

The benefits of laser surgery are best summarized by Sarver and Yanosky[5]: "Soft-tissue lasers result in a shorter operative time and faster postoperative recuperation."

The primary disadvantage of laser surgery is the operatory and upkeep expense. Some clinicians have reported greater tactile sense with a scalpel (which might be particularly true for noncontact soft-tissue lasers such as the erbium laser), tissue desiccation, and poor wound healing.[11]

Lasers cut by thermal ablationdecomposition of tissue through an instantaneous process of absorption, melting, and vaporization. Essentially, the cells of the target tissue absorb the concentrated light energy, rapidly rise in temperature, and produce a micro-explosion known as spallation.[1]

Thermal ablation depends on the amount of light energy absorbed.[4] The degree of absorption is determined by the wavelength (measured in nanometers [nm]) of the laser, the

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electrical power of the surgical unit (measured in watts [W]), the time of exposure and the composition of the tissues.[1,4,6]

The optical fiber, or cutting end of the laser, is protected with an insulated layer that helps to collimate the light energy.[4]

Thus, ablation occurs only at the tip of the optical fiber. Attempting to cut from the sides of the laser will only drag the optical fiber against the gingival tissue, impeding tissue excision and damaging the laser tip.

Soft-tissues lasers deliver light energy in either a pulsed (gated) or a continuous mode. In the pulsed mode, periodic alternations of energy are created by a mechanical shutter that permits intermittent cooling of the tissues between pulses of light energy. Pulse energy is measured in millijoules (mJ) and can be adjusted on the laser display system. In the continuous mode, thermal relaxation does not occur, resulting in greater heat to the tissue. When greater coagulation is needed, either continuous energy or longer pulsed durations are desired to increase residual heat and seal open vessels.[1]

DIODE AND ERBIUM LASERS

Currently, the 2 most popular types of lasers used in dentistry are the diode and the erbium lasers. Diode lasers (ie, Odyssey, Ivoclar Vivadent, Amherst, NY) are almost exclusively used for soft-tissue surgery. Erbium lasers (ie WaterlaseMD, Biolase, San Clemente, Calif) can be used for hard- and soft-tissue surgeries. Each laser produces a different wavelength and has advantages and risks.

Diode lasers are semiconductors that use solid-state elements (ie, gallium, arsenide, aluminum, and indium) to change electrical energy in to light energy. Diode laser wavelengths (810980 nm) approximate the absorption coefficient of soft-tissue pigmentation (melanin).

Therefore, the light energy from the diode is highly absorbed by the soft tissues and poorly absorbed by teeth and bone.

Diode lasers are packaged in small, portable units (typically weighing less than 10 lbs). Connecting to the main unit is a thin, pencil-size hand piece containing a 400-**μm lasing fiber.**

Before surgery, some diode lasers must first be conditioned or primed. Priming is the process of concentrating heat energy at the tip of the laser fiber. This is done by simply taping the fiber on articulating paper while the laser is energized.[3] After the surgery, the end of the fiber (23 mm) is cleaved to expose a fresh tip. The glass fiber optic is scored and removed to prevent cross-contamination.[12]

During laser surgery with a diode, the fiber tip should be held in light contact with the tissue. Excision is performed with gentle, sweeping brush strokes. Highspeed suction is helpful to reduce the slight charred odor and remove the laser plume.[3] The tissues should have a light brown trim with minimal bleeding.

The **advantages of the diode laser** include the following:

(1) they have excellent soft-tissue absorption and hemostasis;

(2) it is difficult to damage hard tissues;

(3) they can be used in contact mode, which provides tactile feedback;

(4) they can be used for tooth bleaching; and

(5) they are compact and low-cost.

Erbium lasers are solid-state lasers based on the erbium ion (Er3+). The ion is incorporated into a crystal matrix, which offers favorable mechanical and thermo optical properties.[1] The most common matrices are yttrium aluminum garnet (YAG) and yttrium scandium gadolinium garnet (YSGG).

The 2 most common erbium lasers are the erbium-YAG and the erbium-chromium- YSGG. Comparative studies have shown little difference in efficacy between them.[14] Erbium wavelengths (2780 2940 nm) can be absorbed by hydroxyapatite and water, and ablate both hard and soft tissues.[15]

Erbium lasers are packaged in larger, rolling units (typically weighing 8090 lbs). The laser hand piece resembles a high-speed hand piece, with removable fiber optic tips.

During surgery with an erbium laser, the fiber tip should be held 1 mm from the tissue.[16] Excision is performed with slow, short back-and-forth strokes. Coagulation is achieved under a different setting, with low wattage and no water. An erbium laser can effectively control hemorrhaging, but strict hemostasis can be difficult because the laser operates in the pulsed mode.[17,18]

Tissues appear slightly reddish during excision and chalky white after coagulation.

The **advantages of the erbium laser** include the following:

(1) priming is not required and

(2) the fiber-optic tips are autoclavable.

The primary **disadvantage** is the size and cost of the operating unit

The main unit requires 80 psi of air pressure provided by an

external source such as an operatory bay. Electrical power, measured in watts, influences the depth of tissue penetration. For the diode laser, soft-tissue excision generally requires less than 1W of power. For the erbium laser, soft-tissue excision can require 1.5 to 2.5 W, depending on the tissue thickness; coagulation generally requires less than 0.75 W.

An erbium laser ablates enamel at 4 to 5 W.14,18 Above 5 to 6 W, patients start to feel significant discomfort.[1] Strict adherence to the manufacturer's recommendations for unit settings should be followed.

Soft-tissue lasers both coagulate and produce a mild anesthetic effect during excision; as such, topical anesthetic to be used in place of local infiltration. The topic anesthetic should be highly viscous, include several active anesthetic agents to provide a wide spectrum of anesthetic action, and contain a vasoconstrictive agent.[19]

PATIENT SAFETY

The clinician should perform ablation with the lowest possible energy. Higher energy will produce a higher ablation rate or speed of excision, but, if the energy is too high, it can cause unnecessary collateral damage.

This is particularly true for the erbium laser, which can penetrate deeper into the dental hard tissue. Sarver and Yanosky[4] recommended using a pulse mode with low wattage for all soft-tissue procedures.

The major concerns in laser surgery are exposure to laser radiation. Laser safety is regulated according to the American National Standards Institute's (ANSI) Z136 safety standards.

ANSI laser safety standards are the basis for Occupational Safety and Health Administration (OSHA) and state occupational safety rules. All lasers sold in the United States since 1976 are classified according to their hazard potential.

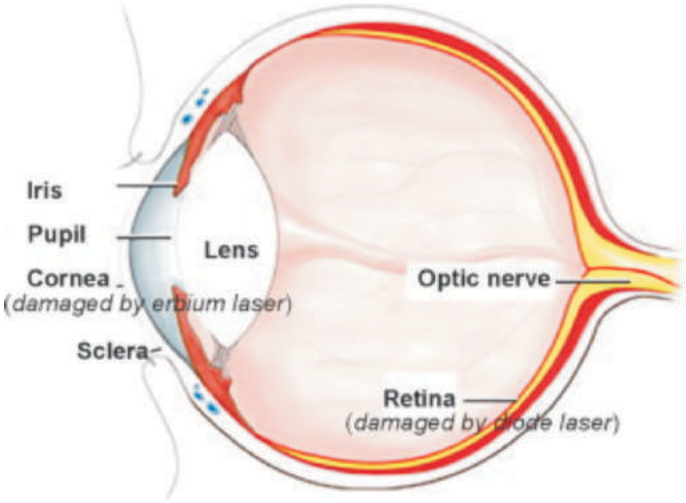
Currently, there are 6 laser hazard classifications (classes 1, 1M, 2M, 3B, 3R, and 4). Lasers used in medical therapeutic use, such as soft-tissue lasers, are class 4 products. Class 4 lasers have an output power greater than 0.5 W. At this power, eyes and skin are endangered even at diffuse reflection. Protective arrangements must include creation of a danger zone, presence of a laser safety officer (the doctor), proper training of users, and consideration of fire hazards.

The greatest risk of soft-tissue laser surgery is injury to an eye. The severity of injury depends on laser wavelength, distance from the laser, and power of the laser.

The eye is precise at focusing light, and a split second exposure to laser radiation can be sufficient to cause permanent injury. Retinal damage can occur at 400 to 1400 nm (called the retinal hazard region).

The major danger is a stray laser beam reflected from a table, jewelry, or a belt. Diode lasers risk retinal burns and cataracts. Erbium lasers risk corneal burns, aqueous flare-ups, and infra-red cataracts.

Skin is the largest organ of the body and poses a high risk of

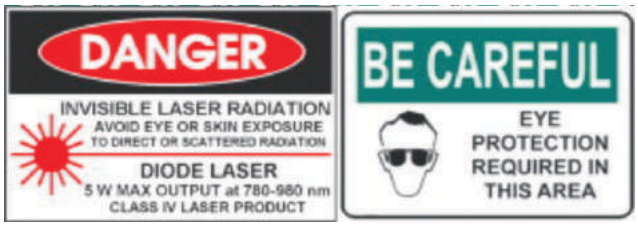


radiation exposure. Skin can be penetrated at wavelengths of 300 to 3000 nm (both diode and erbium lasers), reaching a maximum penetration at 1000 nm. Arms, hands, and head are most likely to be exposed to laser radiation.

The patient and the clinician should be fully covered and wear protective goggles at all times. The goggles must block light at the appropriate wavelength and protect all possible (reflective) paths to the eyes. All nearby reflective surfaces should be covered or removed. Class 4 laser systems pose a fire hazard if the beam contacts flammable substances, and flame-retardant materials should be available.

A discernable danger zone shoul be created around the surgical bay with a sign reading:

"Warning: visible and invisible laser radiation. Avoid eye or



skin exposure to direct scatter radiation. Class 4 laser product".

Informed consent can vary, depending on the type of laser. Consent for the diode laser might include warnings about mild bleeding, postoperative discomfort, and the need for surgical refinement. Consent for the erbium laser might include these additional risks: microcracks in the enamel, pulpal overheating, and tooth necrosis[20] (although these risks are easily minimized by operating the laser at low wattage).

Complete tissue healing takes place in 1 week. The patient should be seen for a postoperative follow-up after 2 weeks.

CONCLUSIONS

Diode and erbrium soft-tissue lasers offer many advantages in regard to esthetic finishing, practice efficiencies, and interdisciplinary treatment options.

Clinicians interested in incorporating soft-tissue lasers into their practice should obtain proficiency certification, provide proper staff training, attend continuing education courses, consider membership in the Academy of Laser Dentistry, and recognize the inherent risks of laser surgery.

REFERENCES

1. Moritz A. Oral laser application. Chicago: Quintessence; 2006.
2. Maiman TH. Stimulated optical radiation in ruby lasers. Nature 1960;187:493.
3. Tracey S. Light work. Orthod Products 2005;Apr/May:88-93.
4. Rossman JA, Cobb CM. Lasers in periodontal therapy. Periodontology 2000 1995;9:150-64.
5. Sarver DM, Yanosky M. Principles of cosmetic dentistry in orthodontics: part 2. Soft tissue laser technology and cosmetic gingival contouring. Am J Orthod Dentofacial Orthop 2005;127:85-90.
6. Sarver DM. Use of the 810 nm diode laser: soft tissue management and orthodontic applications of innovative technology. Practice Proced Aesthet Dent 2006;18: suppl 7-13.
7. Haytac MC, Ozcelik O. Evaluation of patient perceptions: a comparison of carbon dioxide laser and scalpel techniques. J Periodontol 2006;77: 1815-9.
8. Fisher SE, Frame JW, Browne RM, Tranter RMD. A comparative histological study of wound healing following CO2 laser and conventional surgical excision of canine buccal mucosa. Arch Oral Biol 1983;28:287-91.
9. Buell BR, Schuller DE. Comparison of tensile strength in CO2 laser and scalpel skin incisions. Arch Otolaryngol 1983;109:465-7.
10. Frame JW. Removal of oral soft tissue pathology with the CO2 laser. J Oral Maxillofac Surg 1985;43:850-5.
11. Baker SS, Hunnewell JM, Muenzler WS, Hunter GJ. Laser blepharoplasty: diamond laser scalpel compared to the free beam CO2 laser. Dermatol Surg 2002;28:127-31.
12. Press J. Effective use of the 810 nm diode laser within the wellness model. Pract Proced Aesthet Dent 2006;18 (suppl):18-21.
13. Hilgers JJ, Tracey SG. Clinical uses of diode lasers in orthodontics. J Clin Orthod 2004;38:266-73.
14. Harashima T, Kinoshita J, Kimura Y, Brugnera A, Zanin F, Pecora JD, et al. Morphological comparative study on ablation of dental hard tissue at cavity preparation by Er:YAG and Er,Cr: YSGG lasers. Photomed Laser Surg 2005;23:52-5.
15. Eversole LR, Rizoiu IM. Preliminary investigations on the utility of an erbium, chromium YSGG laser. J Calif Dent Assoc 1995;23:41-7.
16. Hadley J, Young DA, Eversole LR, Gornbein JA. A laserpowered hydrokinetic system for caries removal and cavity preparation. J Am Dent Assoc 2000;131:777-85.
17. Wang X, Zhang C, Matsumoto K. In vivo study of the healing processes that occur in the jaws of rabbits following perforation by Er,Cr:YSGG laser. Lasers Med Sci 2005;20:21-7.
18. Rizoiu IM, Eversole LR, Kimmel AI. Effects of erbium, chromium: yttrium, scandium, gallium, garnet laser on mucocutaneous soft tissues. Oral Surg Oral Med Oral Pathol 1996;82:386-95.
19. Graham JW. Profound, needle-free anesthesia in orthodontics. J Clin Orthod 2006;40:723-4.
20. Burkes EJ, Hoke J, Gomes E, Wolbarsht M. Wet versus dry enamel ablation by Er:YAG laser. J Prosthet Dent 1992;67:847-51.

RESEARCH METHODOLOGY

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Abstract :

Improvements in dental practice depend on the continued accumulation and synthesis of new knowledge. Research should provide sound data on which clinicians may rely when making treatment decisions. Ideally advances in clinical care should be evaluated by comparison with currently available practice. New systems should be adopted only if they achieve results that were not previously possible or reduce the failures, inconvenience, or adverse consequences of existing systems. Rigorous and powerful research methods such as randomized controlled trials have been developed for clinical research. A valid result from a scientific study would be one that accurately represents the effect of the intervention without distortion of bias.

Key Words : Randomized Controlled Trials

INTRODUCTION

Research is the method of science. It is a systematic body of procedures & techniques applied in carrying out investigation or experimentation targeted at obtaining new knowledge. In other words, Research is a quest for knowledge through diligent search or investigation or experimentation aimed at the discovery & interpretation of new knowledge.

OBJECTIVES OF RESEARCH:-

The purpose of research is to discover answers to questions through the application of scientific procedures. The main aim of research is to find out the truth which is hidden and which has not been discovered as yet[9,16]. Though each research study has its own specific purpose, we may think of research objectives as falling into a number of following broad groupings:

1. To gain familiarity with a phenomenon or to achieve new insights into it (studies with this object in view are termed as exploratory or formulative research studies);

2. To portray accurately the characteristics of a particular individual, situation or a group (studies with this object in view are known as descriptive research studies);

3. To determine the frequency with which something occurs or with which it is associated with something else (studies with this object in view are known as diagnostic research studies);

4. To test a hypothesis of a causal relationship between variables (such studies are known as hypothesis-testing research studies).

MOTIVATION IN RESEARCH:-

What makes people to undertake research? This is a question of fundamental importance. The possible motives for doing research may be either one or more of the following:[9]

1. Desire to get a research degree along with its consequential benefits;

2. Desire to face the challenge in solving the unsolved problems, i.e., concern over practical

problems initiates research;

3. Desire to get intellectual joy of doing some creative work;

4. Desire to be of service to society;

5. Desire to get respectability,etc.

SCIENTIFIC FOUNDATIONS OF RESEARCH :-

1) Order

2) Inference & chance

3) Maintenance of probability

4) Hypothesis

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Order :

Research employs an organized observation of entities or events which are classified or ordered on the basis of common properties & behaviors[11]. The commonality among the observations help in predictions which carry to the ultimate become laws.

Inference & Chance:

Reasoning or inference is the force of advance inresearch. There are two distinct approaches in the development of inferences : [12]

DEDUCTIVE – It moves from the general to the specific. Hence it does not allow for the element of chance. It is not used much in health research.

INDUCTIVE – It moves from specific to the general. There is extrapolation of results from a sample to the target population. Health research depends almost entirely upon inductive reasoning. Hence chance must be fully accounted.

Maintenance of probability:

Maintaining a very high probability & eliminating the chance occurrence is critical to ensure the validity of a research. Techniques used to maintain high probability[4,11]

1. Representative sampling
2. Randomization in selection of study groups
3. Maintenance of comparison groups as controls
4. Blinding procedures

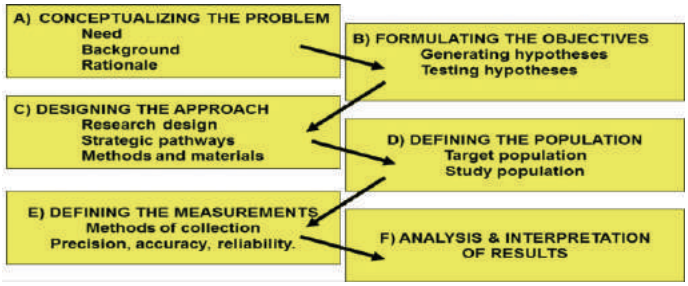
Hypothesis:

Hypothesis is defined as a presumption, supposition or assumption derived either out of observation or reflection[12,17]. Hypotheses are carefully constructed statements generated from inferences & they use argument of induction.

RESEARCH DESIGN :-

Strategies include ... [14]

- Definition of Variables
- Their level
- Relationship to one another



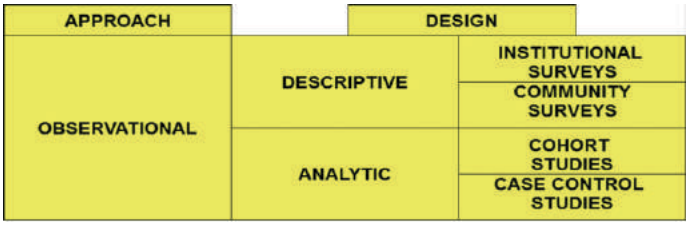
TYPES OF RESEARCH:-

The basic types of research are as follows:

Descriptive vs. Analytical:

Descriptive research includes surveys and fact-finding enquiries of different kinds. The major purpose of descriptive research is description of the state of affairs as it exists at present. The main characteristic of this method is that the researcher has no control over the variables; he can only report what has happened or what is happening.The methods of research utilized in descriptive research are survey methods of all kinds, including comparative and correlational methods. [1,2,17]

In analytical research, on the other hand, the researcher has to use facts or information already available, and analyze these to make a critical evaluation of material.



Applied vs. Fundamental:

Research can either be applied (or action) research or fundamental (to basic or pure) research.

Applied research aims at finding a solution for an immediate problem facing a society, whereas *fundamental research* is mainly concerned with generalisations and with the formulation of a theory. [1,2,13]

"Gathering knowledge for knowledge's sake is termed 'pure' or 'basic' research." Research concerning some natural

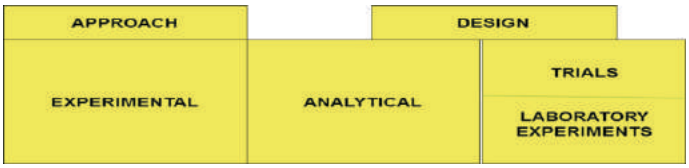
phenomenon or relating to pure mathematics are examples of fundamental research. Similarly, research studies, concerning human behaviour carried on with a view to make generalisations about human behaviour, are also examples of fundamental research.Thus, the central aim of applied research is to discover a solution for some pressing practical problem[6,13], whereas basic research is directed towards finding information that has a broad base of applications and thus, adds to the already existing organized body of scientific knowledge.

Quantitative vs. Qualitative:

Quantitative research is based on the measurement of quantity or amount. It is applicable to phenomena that can be expressed in terms of quantity. Qualitative research, on the other hand, is concerned with qualitative phenomenon, i.e., phenomena relating to or involving quality or kind[8,15]. Qualitative research is specially important in the behavioural sciences where the aim is to discover the underlying motives of human behaviour.[8] Through such research we can analyse the various factors which motivate people to behave in a particular manner or which make people like or dislike a particular thing.

Conceptual vs. Empirical:

Conceptual research is that related to some abstract idea(s) or theory. It is generally used by philosophers and thinkers to develop new concepts or to reinterpret existing ones. On the other hand, empirical research relies on experience or observation alone, often without due regard for system and theory. It is data-based research, coming up with conclusions which are capable of being verified by observation or experiment[1,2,3]. We can also call it as experimental type of research. In such a research it is necessary to get at facts firsthand, at their source, and actively to go about doing certain things to stimulate the production of desired information. In such a research, the researcher must first provide himself with a working hypothesis or guess as to the probable results. He then works to get enough facts (data) to prove or disprove his hypothesis. He then sets up experimental designs which he thinks will manipulate the persons or the materials concerned so as to bring forth the desired information. Such research is thus characterised by the experimenter's control over the variables under study and his deliberate manipulation of one of them to study its effects. Empirical research is appropriate when proof is sought that certain variables affect other variables in some way. Evidence gathered through experiments or empirical studies is today considered to be the most powerful support possible for a given hypothesis.



TYPES OF STUDY:-

Case control study design:

Choose groups with and without disease, look back at what different exposures they may have had. Attempt are made to make inference from existing observations (retrospective) [1,2,7].Compares patients with outcome/disease with those without and attempts to identify factors that influenced that outcome (or caused that disease).

Important concept: **start with the result (disease) and work backwards for the cause**.[1,2,7]

Strength of this study:-

- Best study when have rare disease or outcome
- Relatively quick and inexpensive

Weakness of this study:-

- *Selection (confounding) bias*: controls must be as similar to cases as possible
- Berkinsonian bias
- *Recall bias*: cases may be able to remember events better because of its significance or may be prompted to remember by investigators
- *Survival bias:* dead people don't make it into many case-cohort studies; and if they do, they don't remember things very well

Interviewer's bias

Ways to combat the weakness:-

- *Matching:* for each case, find a control that looks just like him/her in all other possible ways except for the disease (same age, race, economic class, etc.)
- *Blinding:* individual assessing exposures should be blinded to whether the person is a case or control.

Cohort Studies/Prospective study/ Longitudinal/ Incidence/Forward-looking study:

Studies whether exposure to a "risk factor" is associated with a subsequent "outcome". Select two populations who

seem the same except for the hypothesized risk factor. Follow them and see how many have the outcome or disease[1,2,7]. Start with two groups of people who are exposed and unexposed, follow them to see who gets disease.

Important concept: Start with the risk, then look for the outcome.[1,2,7]

Strength of this study:

- Not only can you look at risk, you can calculate how many people actually get the disease (incidence rates).
- Since you enroll subjects before the outcome, you can measure multiple exposures without recall bias.
- Best for rare exposures.

Weakness of this study:

- Cohort studies may take a long time
- Cohort studies may require a large number of people especially if the outcome is uncommon
- Both of these make cohort studies expensive
- Attrition of study group

Cross-sectional study:

- Observation of all of population at one point of time. It is used to setermine prevalence of illness.[1,2,7]

Longitudinal study:

- Series of observation is done more than one on the study population over a period of time.[1,2,7]

BIAS:-

- A valid result from a scientific study would be one that accurately represents the effect of the intervention without distortion of bias.

Types of Biases:[5,14]

- Selection bias
- Information bias
- Confounding bias

Selection bias:

Results due to the way in which subjects were selected into the study. This includes the background characteristics (race, sex, age, occupation, etc.) of the study and comparison groups. It also includes problems resulting from subjects leaving or entering the study while it was in progress (migration bias) or not responding to questionnaires or telephone inquiries (nonrespondent bias).[5] Selective survival, detection bias, and Berkson's bias (hospital[5,14] admission bias). Choosing one's best cases for evaluation of orthodontic treatment is also a form of selection bias.

Information bias:

Information bias is the distortion of the study results due to measurement error or misclassification of subjects into the study and/or comparison groups[5,14]. This can arise if one uses invalid measurements (for example, a faulty sphygmomanometer for blood pressure recordings) or incorrect diagnostic criteria.Can also result from a lack of objectivity from subject or examiner. Thus, "blinding" and "double-blinding" are frequently used to keep the subject or the subject and the examiner from knowing who is in the experimental group and who is in the control group.

Confounding bias:

Study factor of interest is mixed with the effect of other extraneous variables. For example, in many epidemiologic studies age acts as a confounder.[5,14]

EHICAL CONSIDERATIONS:-

3 principles :[10]

- Beneficence
- Respect for rights
- Justice

Declaration of Helsinki[10]

- Scientific principles
- Qualified personnel
- Careful assessment of risk Vs benefit
- Rights of subject
- Accuracy of research results
- Subjects to be well informed
- Informed consent
- Subject free to abstain or withdraw any time

CONCLUSION:-

Adherence to the standards of research methods is vital if dental research is to make real progress.

REFERENCES:-

1. Textbook of preventive and social medicine.K.Park.20th edition.
2. Essentials of preventive and community dentistry.Soben Peter.5th edition.
3. Chapel Hill, N.C., Boston, Mass., and Jackson, Miss. A review of clinical research in orthodontics. AJODO.1989;95:499-504.
4. Ceib Phillips.Sample size and Power :What is enough?.Semin orthod.2002;8:67-76.
5. Guest editorial.Reasearch Biases.AJODO.1983:351-352.
6. Watson, Wayne: Orthodontic methods-Art or science, AM. J.ORTHOD. 83: 353-354, 1983.
7. Ceib Phillips,J.F.Camilla Tulloch.The Randomised Clinical Trial as powerful means to understand clinical efficiency.Seminar in orthod.1995;1(3):128-138.
8. Omar Gelo,Diana Braakman,Gerhad Benetka. Quantitative and Qualitative Research: Beyond the Debate.September 2008;42(3) 26:6-290
9. Carlos L. Lastrucci, *The Scientific Approach*: *Basic Principles of the Scientific Method,* p. 7.
10. Principles of research methodology.Supino,Phyllis G,Borer,Jeffery.S.2012.
11. Ballini A, Capodiferro S, Toia M, Cantore S, Favia G, De Frenza G, Grassi FR Evidence-based dentistry: what's new? Int J Med Sci. 2007 Jun 6;4(3):174-8.
12. DePaola DP. The revitalization of U.S. dental education. J Dent Educ. 2008 Feb;72(2 Suppl):28-42.
13. Creswell, J. W., Plano Clark, V. L., Gutmann, M., & Hanson, W. (2003). Advanced mixed methods research designs. In A. Tashakkori, & C. Teddlie (Eds.), Handbook of mixed methods in social and behavioral research (pp. 209204). Thousand Oaks, CA: Sage.
14. Michael,Robert Moyers, Thomas.Imprecision and bias in orthodontic treatment results.AJODO.1988;93:138-42
15. Cox M, Pacala JT, Vercellotti GM, Shea JA. Health care economics, financing, organization, and delivery. Fam Med. 2004 Jan;36 Suppl:S20-30
16. Iacopino AM.The influence of "new science" on dental education: current concepts, trends, and models for the future. J Dent Educ. 2007 Apr;71(4):450-62
17. Krithikadatta J. Research methodology in Dentistry: Part I - The essentials and relevance of research.J Conserv Dent. 2012 Jan;15(1):5-11

BIOSTATISTICS

Dr. Shreyash Patel[a], Dr. Urvish Modi[b]

Abstract :

Orthodontic effects are, for the most part, gross and measurable. Thus, of all of dentistry's specialists, it should be easiest for us to deliver optimal, evidence-based treatments. Unfortunately, the profusion of disparate methods and philosophies that compete for our attention argues that, for some reason, our penchant for measuring things has come to naught. A basic understanding of statistical methodology is essential, both for designing quality research projects and for evaluating the medical literature. This article deals with the basic principles involved in sample size calculations and shows the concepts and factors that determine sample sizes for comparing means, proportions, and time-to-event measures.

Key Words : SD- Standard Deviation, SEM- Standard error of the mean

INTRODUCTION

We are said, generally by the editors of professional journals, to have entered an era of evidence-based dentistry. Orthodontics, however, does not seem to be part of the trend. In the marketplace of orthodontic ideas, much that is bought and sold is little more than wishful thinking shaped by the biases of referring dentists, many of whom have a profound distrust both of orthodontics and of the people who practice it. How can this be? Orthodontics is a learned specialty peopled by the best in dentistry. The prevailing model of health care delivery emphasizes evidence-based criteria and guidelines for developing treatment options and alternatives. [1,2]

Statistics have become the second language of dental science and to be unfamiliar with its concepts and techniques is to be functionally illiterate. Professionals thus handicapped are the rightful prey of the entrepreneur.[3]It would help us to determine if investigators applied appropriate, if any, statistical techniques to the data used to address clinical and research questions. It would also guide us in verifying whether or not the conclusions were supported by the results of the analysis. This is the first step in practicing evidence-based dentistry. Our ability to assess critically the information levelled at us would be greatly enhanced by a familiarity with biomedical statistics.[4]

SAMPLING

A sample is a group of units selected from a larger group (the population). By studying the sample it is hoped to draw valid conclusions about the larger group.[5]Sampling is that part of statistical practice concerned with the selection of a subset of individuals from within a population to yield some knowledge about the whole population, especially for the purposes of making predictions based on statistical inference.

Need for sampling

A sample is generally selected for study because the population is too large to study in its entirety. The sample should be representative of the general population. Also, before collecting the sample, it is important that the researcher carefully and completely defines the population, including a description of the members to be included.

Sample size calculations are made using various assumptions about the anticipated treatment effect or differences between treatments together with realistic projections concerning patient accrual and follow-up.

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Calculating a sample size requires four things:

(1) Deciding on the design of the study;

(2) Assessing the availability of resources;

(3) Specifying distribution assumptions; and

(4) Perhaps most challengingly, defining a clinically relevant effect.

Key Questions before Sample Size Calculation

1. What is the primary outcome? Clinicians frequently include multiple outcomes in clinical studies. This creates a dilemma for calculating a sample size (n) because the n required will vary from outcome to outcome. One or two outcomes should be designated as the primary basis for sample size determinations. Other outcomes typically are considered the secondary analyses.

2. What's the scale of measurement for the outcome? Outcomes can be classified as binary or dichotomous (yes/no, present/absent), categorical (mutually exclusive categories), ordinal (ranked or rated), or continuous (measures on which arithmetic operations can be performed). In general, binary and categorical measures provide less information and thus lower power, requiring larger sample sizes.

3. What is the variability o/the outcome? For binary measures, the variability is directly related to the proportion of positives (yes or present responses). For continuous measures, the sample size required increases as the standard deviation increases.

4. What is the desired level of significance and power? Typically, level of significance is set at 0.05 or 0.01, whereas power is set at 80% or 90%. Increasing the power or decreasing the level of significance increases the sample size required.

5. Are there special features in the study design? These include multiple group comparisons, clustered outcome data (such as multiple observations of the same subject or multicenter studies), long-term follow-up when dropout or attrition is expected, and inclusion of covariates (other measures that are believed to somehow affect the relationship between the treatment and the outcome). For many studies, software packages such as nQuery Advisor or Epilnfo can be used to make preliminary T sample size determinations. However, special features often involve nonstandard calculations and a statistician should be consulted.

6. What defines a clinically relevant effect (effect size)? This is perhaps the most challenging question. The clinician must define the clinically relevant effect in terms of the primary outcome. What difference expressed in the values of the outcome measure would alter/change/ modify, the way" patients are treated)?

Hypothesis Testing

Clinical research is designed to determine if a treatment has an effect or if different treatments produce different outcomes. Suppose we believe that removable appliances increase mandibular length. We might assume that in untreated Class II children, age 7 to 10 years, mandibular length growth during a 1-year period is a normally distributed variable (bell-shaped curve) with a mean μ1 and a standard deviation (SD) σ1. The mean refers to the average amount of mandibular growth in 1 year and is represented μ1 because we do not know the true amount of growth. The SD tells us how much variation we can expect in the amount of annual growth among all children age 7 to 10 years. The SD is represented by μ1. Another special term is the standard error of the mean (SEM). SEM is a measure of variability similar to the SD. SD indicates the spread of the values for a measurement such as mandibular length changes. SEM tells us about the spread of values of the means that would occur if we drew all of the possible samples of a given size from the population and calculated the mean for each sample. It is important to understand the concept of means and SDs because their estimates are used to calculate sample sizes.

RANDOMIZATION

A key feature of a randomized control trial is the process of randomization. Through randomization, we allocate interventions to trial arms in a way that ensures that neither the investigators nor the participants know or can predict ahead of time which treatment a subject will receive. Proper randomization procedures and reporting include the following steps.[5]

1. Generation of the random allocation sequence, including details of any restrictions.

2. Allocation concealment.

3. Implementation of the random allocation sequence: information on who generated the allocation sequence, who enrolled the participants, and who assigned them to their groups.

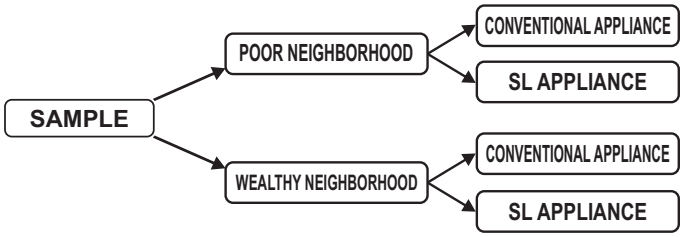


FIGURE:-Stratified Randomization to conventional or self-ligating appliances by location. A separate randomization list is generated for each location- neighbourhood.

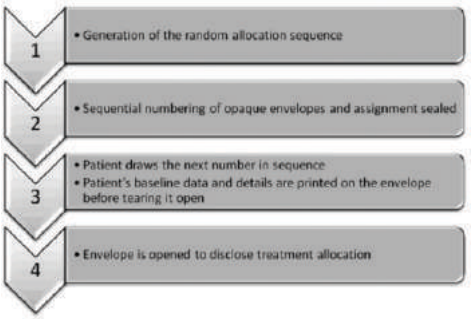
Allocation concealment is the means to guarantee that the generated randomization lists and consequently the treatment allocations of the trial participants cannot be known or predicted by all involved persons. These include patients, investigators, and other personnel engaged in the study.

The objective of allocation concealment is to reduce selection bias, and it can always be applied. Knowing or being able to predict the next treatment allocation might lead to subversion of the randomization in a way that validates the preconceptions of the investigators. For example, if we are conducting a study to compare time to alignment between conventional and self-ligating appliances, lack of allocation concealment could permit a biased investigator to undermine randomization and assign patients with more crowding to the group that he or she does not favour. In this manner, the ensuing selection bias will falsely overestimate or underestimate the effect of appliance type on the alleviation of crowding. In other words, if the investigator favours conventional appliances, he or she might randomize patients with more severe crowding to the selfligating group, thus making the conventional arm appear to reach alignment in a shorter time. Studies on allocation concealment have shown that lack of allocation concealment has been associated with greater and biased treatment effects.[5]

Advantages and Disadvantages of common randomization methods, adapted from Pandis et al[2]

	ADVANTAGES	DISADVANTAGES
Simple randomization	Simple to implement Unpredictable	Unequal sizes of trial arms are common Cannot balance on baseline characteristics, especially in small trials
Blocked randomization	Ensures balance in trial arms at all times	Assignment can be predicted if nonvarying, small blocks are used, especially when blinding is not feasible Cannot balance on baseline characteristics, especially in small trials
Stratified randomization	Ensures balance in trial arms when combined with blocking. Balances on important baseline characteristics that are potential outcome predictors	Overstratification, many subgroups, and imbalances due to incomplete blocks Assignment can be predicted if nonvarying, small blocks are used, especially when blinding is not feasible Identification of participants before group assignment is necessary
Minimization	Ensures balance in trial arms Balances on important baseline characteristics that are potential outcome predictors	Complex, especially when several predictors are considered Knowledge of previous allocations is required Not strictly random, but a random element could be included

Sequence of events when using sealed opaque envelopes



In RCTs, it is important to create treatment groups that are similar in all possible known and unknown factors except for the treatment that the trial groups will receive. If the treatment groups are similar, then we are more certain that any differences in the outcomes are related to the intervention rather than to other factors.

The control group in an RCT is required for the following reasons.[5-7]

- Participants with a certain condition might get well with time regardless of any therapy received. Therefore, if a study is conducted without a control group, we cannot be certain whether any improvements are related to the intervention or to the effect of time. For example, suspected canine impaction might resolve automatically without intervention as patients get older.
- Selection bias can cloud the results. An investigator might select participants who have the best prognosis, thus permitting overestimation of the effects of the treatment of interest. For example, investigators designing a trial to assess resolution of canine impaction after deciduous canine extraction (the intervention) without using a control group might select subjects who are more likely to experience the outcome (resolution of canine impaction) and conclude that the intervention is effective when it is not.
- The placebo effect can be powerful. Study participants might respond better to treatment just because they are included in a study and not because they are receiving active therapy. The use of a placebo in the control neutralizes the potential positive response to a therapy just because of inclusion in a study. In other words, patients receiving the active therapy might experience a response, which would be the sum of the placebo effect (invoked by inclusion in the trial) and the effect of the intervention. Adding a control allows for the comparison to be fair, since the control group will also experience the placebo effect; therefore, the difference between the 2 groups would be only the effect of the treatment.
- The Hawthorne effect can affect subjects' behaviours. Inclusion in a study might elicit behavioural changes that can predispose participants to respond in an exaggerated fashion to the therapy. For example, when subjective outcomes, such as pain levels, are used, participants might modify their response because they know that they are being tested.[8]

Blinding or Masking

The term "single blind" indicates that only patients or investigators are unaware of the intervention; "double blind" indicates that both patients and investigators are blind to the assignment. Blinding can also be extended to include other personnel and data analysts.[5,6] Bias from lack of blinding can be generated at the patient level and the investigator or staff level (Table).

Type of Bias due to lack of blinding at patient and investigator or staff levels		
Type of bias	Patient level	Investigator level
Selection bias		yes
Post-randomization bias	yes	yes
Observer bias	yes	yes
Bias in study management		yes
Biased data management and analysis		yes

- Postrand omization selection bias. A patient who is unhappy with the assigned treatment might be less cooperative, not follow directions, or drop out of the study. For example, in a trial comparing oral hygiene levels with a standard toothbrush and an electric toothbrush, patients who are enthusiastic about the potential effects of electric toothbrushing but assigned to the standard group might be less cooperative and follow the protocol less diligently.

  Observer bias. Patients who know their treatment might respond more or less favourably to the intervention, depending on their predisposition to this treatment. Observer bias is potentially worse when the outcome is subjective, because patients might consciously or unconsciously give better scores to the preferred treatment or modify their behaviour (Hawthorne effect) in a way to give the expected answer. For example, reporting of pain levels might be influenced by whether the assigned treatment is favoured by the patient receiving it.[7,8]

At the investigator or staff level:

- Selection bias. Knowledge of treatment allocation might allow investigators to guess the next allocation, thus influencing their ability to assign future allocations without bias.
- Postrandomization selection bias. Investigators and other personnel might exclude patients from the trial after randomization, applying different standards between the treatment arms.

Bias in study management and concomitant care. Investigators and other personnel might follow more closely or more frequently, or provide better care to the preferred treatment group, thus potentially overestimating the effect of the therapy assigned to that group.

Observer bias. Unblinded investigators, depending on their predisposition to the treatment, might record outcomes in a more optimistic manner for the favoured group. Investigators might round outcome values up or down, and they might repeat measurements when unexpected values are recorded. With subjective outcomes, the investigators might coerce patients consciously or unconsciously to give the desired response. Also, recording of side effects might be biased in favour of the preferred treatment.

Biased data management and analysis. Unmasked data analysts might introduce bias on the analytic strategies to be used. Analysts might select favourable time points, outcomes, and subgroups, or they might select biased handling of missing data and analyses that emphasize the desired results.5

Sample calculation for split-mouth designs

- Split-mouth designs are efficient because they require smaller sample sizes.
- Split-mouth designs are not appropriate when contamination between sites is suspected and when it is not possible to find matching sites in patients.

Split-mouth designs are not appropriate for all interventions, especially when the therapy on 1 quadrant might affect the outcome on the other quadrants, or when it is difficult to find appropriate and similar pairs of quadrants or teeth in the same patients.[9,10] For example, it would be difficult to conduct a splitmouth trial aiming to compare plaque reduction achieved by 2 types of antibacterial mouth rinses because the risk of 1 mouth rinse contaminating the other side is high.

CONCLUSION

1. Sample calculations are an important part of solid clinical trial methodology.
2. Sample calculations should balance scientific validity with feasibility.
3. Important differences between intervention arms might go undetected only because sample size and power are low.
4. Sample calculations make certain assumptions, and information from previous studies or pilots are helpful for reliable sample calculations.
5. Trial design characteristics such as clustering and pairing should be considered during sample size calculations.
6. Clustering requires a larger sample size compared with nonclustering, whereas pairing decreases the required sample size compared with nonpairing.
7. Allocation concealment is always feasible and should not be confused with blinding.
8. Careful implementation of the randomization process is as important as the generation of random allocations and allocation concealment.

REFERENCES

1. BeGole E: Interpreting the literature on the effectiveness of clinical procedures. Semin Orthod 6:67-75, 2000
2. Sackett D, Straus S, Richardson W, et al: Evidence-based Medicine: How to Practice and Teach EBM (ed 2). New York, NY: Churchill Livingstone, 2000
3. Lysle E. Johnston, Jr. Statistics as a Second Language: A Brief Overview for the Wary Clinician.Semin Orthod.2002;8:54-61
4. Rose D. Shears and V. Shane Pankratz. Common Statistical Tests.Semin Orthod.2002;8:77-86
5 Moher D, Hopewell S, Schulz KF, Montori V, Gøtzsche PC, Devereaux PJ, et al. CONSORT 2010 explanation and elaboration: updated guidelines for reporting parallel group randomised trials. BMJ 2010;340:c869.
6 Boutron I, Moher D, Altman DG, Schulz KF, Ravaud P. Extending the CONSORT statement to randomized trials of nonpharmacologic treatment: explanation and elaboration. Ann Intern Med 2008;148: 295-309.
7 Pocock SJ. Clinical trials: a practical approach. Chichester, United Kingdom: Wiley; 1993. p. 50-65.
8 Piantadosi S. Clinical trials: a methodologic perspective. 2nd ed. Hoboken, NJ: Wiley; 2005. p. 192.
9 Lesaffre E, Philstrom B, Needleman I, Worthington H. The design and analysis of split-mouth studies: what statisticians and clinicians should know. Stat Med 2009;28:3470-82.
10 Hujoel PP, DeRouen TA. Validity issues in split-mouth trials. J Clin Periodontol 1992;19:625-7.

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	Gingivitis reduction *from placebo at 6 months[1]	41%	39%
	Anti - viral activity for adenovirus and coronavirus (responsible for majority of winter colds)[2]	✓	✗
	Bactericidal activity for strict anaerobes (responsible for post - operative infections)[2]	✓	✗
Long term effect	Does not cause tooth staining[1, 7]	✓	✗
	Does not cause taste alterations[6, 7]	✓	✗
	Does not promote Calculus formation[1, 7]	✓	✗
	Efficacy not impaired by toothpaste ingredients[5,7]	✓	✗
	Approved by IDA and ADA[3]	✓	✗
	CONCLUSION	Ideal for Long term use	Not ideal for long term use

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1. Overholser CD, Meiller TF. DePaola LG, Minah GE and Niehaus C. Comparative effects of 2 chemotherapeutic mouthrinses on the development of supragingival dental plaque and gingivitis. J Chn Periodontol 1990; 17.-S7S-579. 2. Lawrence J. Walsh.Antiviral and antibacterial effects of pre-procedural mouth rinses. 3. Data on file 4. John C. Gunsolley.cA meta-analysis of six-month studies of antiplaque and antigingivitis agents. J Am Dent Assoc 2006;137;1649-1657 5. Barkvoll P, Rolla G, Svendsen AK. Interaction between chlorhexidine digluconate and sodium lauryl sulfate in vivo. J Clin Periodontol. 1989;16:593-595. 6. Peridex® (0.12% chlorhexidine gluconate) Oral Rinse [prescribing information]. Phoenix, Ariz: Zila, Inc; 2004. 7. Louis G. DePaola and Ann Eshenaur Spolarich. Safety and Efficacy of Antimicrobial Mouthrinses in Clinical Practice, J Dent Hygiene 2007, Supp: 13-25.

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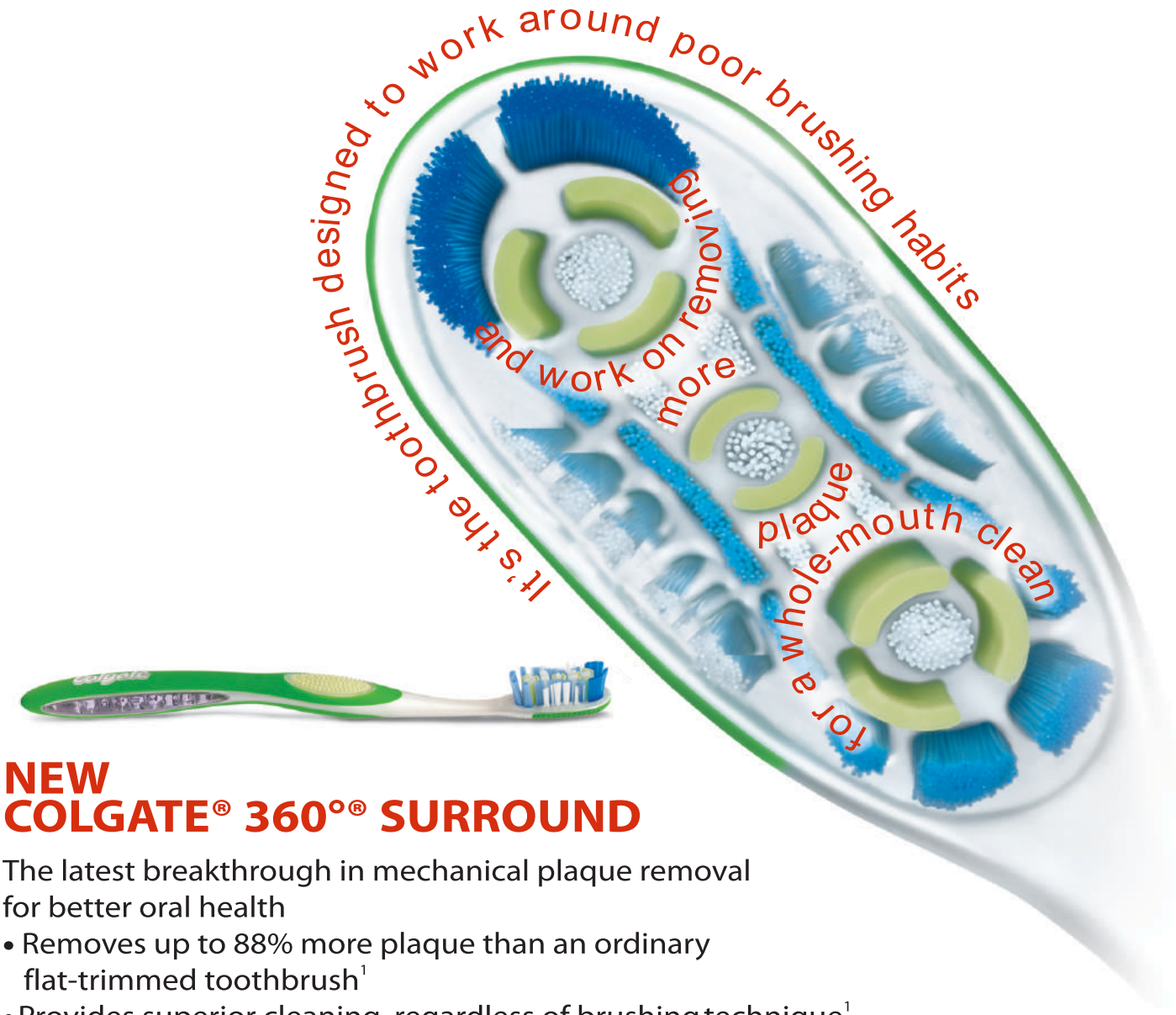
1119.2012



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Refrence: 1. Mankodi S. Data on file. July 2010. Colgate e-Palmovie company

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