Prevalence, Distribution, Caries lesion severity and Salivary Characteristics of Molar Incisor Hypomineralization

- A Cross-Sectional Study

Dissertation submitted to

THE TAMILNADU Dr M.G.R. MEDICAL UNIVERSITY

In partial fulfilment for the degree of

MASTER OF DENTAL SURGERY

BRANCH – VIII

PEDODONTICS AND PREVENTIVE DENTISTRY

APRIL 2017

KSR INSTITUTE OF DENTAL SCIENCE AND RESEARCH

DEPARTMENT OF PEDODONTICS AND PREVENTIVE DENTISTRY

CERTIFICATE

This is to certify that the dissertation titled "Prevalence, Distribution, Caries lesion severity and Salivary Characteristics of Molar Incisor Hypomineralization - A Cross Sectional Study" is a bonafide work done by Dr. ALLWYN SAMUEL.J, Postgraduate student, during the course of the study for the degree of "Master of Dental Surgery" in Department of Pedodontics and Preventive Dentistry, KSR Institute of Dental Science and Research, Tiruchengode during the period of 2014-2017.

Date: **Dr. G.S. Kumar, M.D.S.,**

Place: Tiruchengode **Principal**

KSR INSTITUTE OF DENTAL SCIENCE AND RESEARCH

DEPARTMENT OF PEDODONTICS AND PREVENTIVE DENTISTRY

CERTIFICATE

This is to certify that the dissertation titled "Prevalence, Distribution, Caries lesion severity and Salivary Characteristics of Molar Incisor Hypomineralization - A Cross Sectional Study" is a bonafide work done by Dr. ALLWYN SAMUEL.J, Postgraduate student, during the course of the study for the degree of "Master of Dental Surgery" in Department of Pedodontics and Preventive Dentistry, KSR Institute of Dental Science and Research, Tiruchengode during the period of 2014-2017.

Date:

Place: Tiruchengode

Dr. Sharath Asokan, M.D.S., Ph.D

Professor and Head

DECLARATION BY THE CANDIDATE

TITLE OF DISSERTATION	Prevalence, Distribution, Caries lesion severity, and Salivary characteristics of Molar Incisor Hypomineralization -A Cross-Sectional Study
PLACE OF STUDY	K.S.R Institute of Dental Science and Research
DURATION OF COURSE	3 Years (2014-2017)
NAME OF THE GUIDE	Dr. Geetha Priya PR
HEAD OF THE DEPARTMENT	Dr. Sharath Asokan

I hereby declare that no part of the dissertation will be utilized for gaining financial assistance for research or other promotions without obtaining prior permission from the principal, K.S.R Institute of Dental Science and Research, Tiruchengode. In addition, I declare that no part of this work will be published either in print or electronic without the guide who has been actively involved in this dissertation. The author has the rights reserved for publishing the work solely with prior permission of the principal, K.S.R Institute of Dental Science and Research, Tiruchengode.

Head of the Department **Signature of candidate**

Acknowledgement

"Thank you for coming into my life and giving me joy, thank you for loving me and receiving my love in return. Thank you for the memories I will cherish forever"

I am extremely grateful to my dear parents **Mr.Joseph Rajendran, Mrs.Annie Joseph,** my sister **Mrs.Jemima** and my brother in law **Dr.Carl lewis** for their love, prayers, caring and sacrifices for educating and preparing me for my future. Without you all, I couldn't have achieved my dream. I express my sincere thanks to **Dr. G. S. Kumar, M.D.S.,** Principal for providing the opportunity of doing post-graduation in this college.

I would like to express the deepest gratitude to my Professor and head, **Dr.Sharath Asokan**, who has shown the attitude and the substance of brilliance. My sincere thanks for his valuable guidance, suggestions and for giving me freedom throughout the course. Without his supervision and constant help this dissertation would not have been possible.

I would like to express the deepest appreciation to my guide and reader, **Dr.Geetha Priya,** for the trust, the insightful discussion, offering valuable advice, her support and guidance throughout the course. You are always my inspiration mam.

Dr.T.D.Yogesh Kumar, my senior lecturer, has been a pillar of support, encouragement, both personally and professionally. I would like to thank **Dr.G.Thiruvenkadam and Dr.Seby Thomas** my loved seniors and good friends who were along with me throughout my course. Thank you for being there to pass through my ups and downs in life. Sincere thanks to **Dr.Logeshwari** and **Dr. Lakshmi Prabha**, for their support and motivation.

I would also wish to thank my seniors **Dr.A.K.Shanmugaavel, Dr.Saravana Kumar** for their advice, motivation, and helping in my clinical cases as teachers. At this juncture, I thank my friends **Dr.Kameshwaran, Dr.Vijayasankari, Dr.Janani, Dr.Jijo Mon, Dr.Chitravadhana** for being with me throughout my study and course. Your timely help, support, never ending enthusiasm motivated me in many ways. It was like a superb dream being with you all in this entire post graduate program.

I would thank my besties **Dr.Sreedev, Dr.Ishwarya, Dr.Sai, Dr.Poojitha, Dr.Ithris, Dr.Sanjay, Dr.Jhonny** for their timely help and constant support. I am thankful to **Dr. Kal Singh M.S.C., Ph.D.,** Chennai, for the Biostatistics work. I would like to thank all the non-teaching staff, for their support and help all throughout my PG programme. A word of thanks is always reserved for all those **children** who took part in my study, without whom this study would have been only a dream. Last but not least, I thank **Team Pedo** for being a family to me. I dedicate this dissertation to Team Pedo.

CONTENTS

INTRODUCTION

Dental development and mineralization in humans starts before birth and continues till adolescence when after the all permanent molars complete their mineralization. The first sign of tooth mineralization is seen in the primary lower incisors in the beginning of the second trimester of pregnancy and is finished around three month's post-partum. The first tooth in the permanent dentition to mineralize is first permanent molar, a process that starts around birth and is completed at approximately three years of age **(Reid & Dean 2006)**[50]. Enamel and dentin are formed by secretory cells and the enamel forming cells, the ameloblasts, are highly specialized cells of ectodermal origin **(Simmer 2010; Mahoney 2011)**[53, 45]. After mineralization is complete neither enamel nor dentin is remodeled.

Tooth enamel is unique among the mineralized tissues due to its high mineral content. Enamel is made up of highly organized, tightly packed crystallites that comprises 87% of its volume and 95 of its weight. Other mineralized tissues have about 20% of organic matter whereas mature enamel has less than 1% of organic matter. Enamel crystallites contain more than 1000 times the volume of corresponding crystals in bone, dentin and cementum. Superior organization and mineralization give dental enamel its outstanding physical properties, making it hardest tissue in the vertebrate body. Despite its hardness, tooth enamel can be destroyed fairly rapidly by dental caries. Additionally enamel is also afflicted by various structural defect which could be inherited or acquired.

In 1992, a working group of the **World Dental Federation (FDI)** presented an epidemiological index of DDE (Developmental Defects of the Dental Enamel). The DDE index allows recording of a broad range of defects, with no assigning of etiology and the defects are categorized as demarcated opacities, diffuse opacities, or hypoplasia. A developmental defect was defined as a disturbance arising in hard tissue matrices and in

their mineralization during odontogenesis **(FDI Commission on Oral Health R&E 1992)**[23]. Developmental defects of enamel were commonly defined as hypoplasia, but according to the FDI Commission on Oral Health, Research and Epidemiology (1992), and these defects are precisely been classified into two distinct categories: hypomineralised enamel or enamel opacities and enamel hypoplasia. While opacity is defined as a qualitative defect of the enamel, hypoplasia is defined as a quantitative defect of the enamel **(Suckling, 1989)**[56].

The term Molar Incisor Hypomineralization (MIH) was first cited by **Weerheijm et al, 2001**[59] and this terminology was adopted by the International Dental Scientific Community as a result of a consensus after innumerous discussions in relation to developmental defects of enamel **(Weerheijm et al 2003)**[60]. MIH was defined as the clinical appearance of morphological enamel defects involving the occlusal and/or incisal third of one or more permanent molars or incisors as result as "hypomineralization of systemic origin" **(Weerheijm, 2004)**[62]. The first permanent molar enamel is commonly affected to an extent ranging from mild to severe; as well as in many cases the incisor enamel is also affected. Furthermore, this specific form of developmental defects of enamel **(Baroni & Marchionni, 2011)**[6] show opacities often asymmetrically distributed. There can be a marked variation in severity within an individual and it ranges from small demarcated white, yellow or brown opacities. **Weerheijm et al.** suggested that more severe cases are characterized with post enamel breakdown (PEB) with soft porous enamel which looks like discolored chalk or Old Dutch cheese. **Brook, 2009**[8]**, Chawla et al. 2008**[10] suggested that yellow–brown enamel defects are more severe than white–opaque ones. The stained degree of MIH enamel, may be used clinically to reflect the severity of the defect **(Farah et al., 2010)**[22]**.** In severe cases, the defective enamel is lost shortly after molar eruption, exposing

underlying dentine favoring the tooth sensitivity and the dental carious lesion **(Kilpatrick, 2009)[35].**

Weerheijm KL et al. 2001 stated a term Molar Hypomineralization (MH) as a qualitative defect of enamel with a decreased amount of mineralized, inorganic substance resulting in alterations of translucency of enamel[59]. The term MH includes only the first four permanent molars excluding the incisors. Clinically, the defects of MH/MIH appear to extend gingivally from cuspal tips, rarely involving cervical enamel , and present as demarcated opacities varying in color and usually the enamel shears off under masticatory forces, resulting in PEB.

Lately, MIH has been understood as a hypocalcified subtype of enamel defect with reduced mineral content with low residual content of amelogenins and the presence of more than 16 types of proteins in affected teeth, thirteen of these proteins are found in saliva and crevicular fluid **(Kojima et al. 2000, Denny et al. 2008)[37, 16]** and the 3 others (hemoglobin, albumin, complement C3) are major components of blood. Moreover, protein composition of MIH enamel varies with severity of enamel defect **(Mangum et al., 2010)[46].**

Majority of the studies have revealed that the etiology of Molar Incisor Hypomineralization is very complex associated with systemic and genetic factors disrupting normal amelogenesis in affected tooth. The various medical factors proposed as causing or contributing to MIH includes prenatal and postnatal illness, antibiotic consumption, low birth weight and toxins from breast feeding. Few authors also suggested the correlation of systemic conditions like bronchitis, pneumonia with the MIH / MH.

The MIH porous enamel can easily break down especially under the influence of masticatory forces, leaving unprotected dentin and favoring the development of carious lesions. Many authors suggests that there is an association between the dental caries and

these defects. Although MIH is an asymmetrical defect, when a severe injury exists in a tooth, it is common for the contra lateral tooth to be also affected.

Reports of the prevalence of MIH vary considerably throughout the world and rates range from 2.4% to 40.2% with the highest prevalence reported in children of Rio de Janeiro, Brazil[54]. While these large variations may reflect real differences between regions and countries, differences in recording methods, indices used and populations investigated may also be contributory. A majority of MIH prevalence studies have been conducted in Europe[66].

Saliva plays a vital role in oral health. The saliva has effectiveness in promoting remineralization over demineralization which is associated with calcium and phosphate content, viscosity, pH, flow rate and acid buffering capacity. An acidic environment encourages aciduric and cariogenic bacteria proliferation, resulting in the net loss of minerals in tooth. The properties of individual's saliva might also have an effect on the integrity of defective enamel[7]. Hence, lack of adequate data in Indian population and the possible impact of salivary characteristics of MIH resulted in planning this study.

AIM AND OBJECTIVES

AIM

The aim of this study was to determine the prevalence of Molar Incisor Hypomineralization (MIH) in school children aged 8 to 12 years in Tiruchengode, Tamilnadu.

OBJECTIVES

The present study was conducted with the following objectives

1. To determine the prevalence of MIH / MH in school children of Tiruchengode.
2. To assess the dental caries lesion severity in MIH / MH children using ICDAS II.
3. To assess the salivary pH, its buffering capacity and plaque pH of children with MIH / MH.
4. To compare the salivary characteristics of MIH / MH children with otherwise normal children of the same age.

REVIEW OF LITERATURE

Dummer PM, Kingdon A, Kingdon R (1986)[18] assessed the prevalence of enamel developmental defects in 579 children with mean age of 11 – 12 years. Normal dental operating light was used along with DDE Index. Their results suggests that the enamel defects in boys was significantly higher than girls. Boys tended to have a higher prevalence of yellow opacities compared to girls.

Koch G, Hallonsten AL, Ludvigsson N, Hansson BO, Holst A, Ullbro C (1987)[36] conducted an epidemiologic study to analysis the prevalence, extension and severity of hypomineralization in 2,226 Swedish children who were born in between 1966-1974. They reported that 15.4% children had "idiopathic hypomineralization" of permanent first molars.

Amerongen WE van, Kreulen CM (1995)[58] conducted a retrospective pilot study to identify the possible etiology of cheese molars. They concluded that there was a positive correlation between birth related conditions, childhood systemic conditions like bronchitis, pneumonia, infections of upper respiratory organs, high fever, gastro Intestinal disorders and the occurrence of hypocalcified molars.

Alaluusua S, Lukinmaa PL, Vartiainen T, Partanen M, Torppa J, Tuomisto J (1996)[2] evaluated the correlation between dioxin exposure from mother's milk in normal breast-fed child population with enamel hypomineralization of teeth. In the 102, 6 to 7 year old Finnish children who were breast-fed for an average of 10.5 months, hypomineralization was found in 17 children. Their results suggested that dioxin in human milk may be an important cause of hypomineralization in the developing teeth.

Seow WK (1996)[52] conducted a longitudinal cohort study to investigate the dental development and prevalence of enamel defects in a group of 55 children with very low birth weight and compared the data with 55 normal birthweight children. They concluded that

very low birth weight (VLBW) prematurely born children have delayed dental development with higher prevalence of 11% enamel defects in first permanent molars and incisors.

Alaluusua S, Lukinmaa PL, Koskimies M, Pirinen S, Holtta P, Kallio M, Holttinen T, Salmenpera L (1996)[1] evaluated the association of prolonged breast feeding with the occurrence of dental defects. They concluded that long term breast feeding may increase the risk of mineralization defects in healthy children due to possible chance of environmental contaminants that will interfere with the tooth development.

Jalevik B, Klingberg G, Barregard L, Noren JG (2001)[33] examined developmental enamel defects in 516 Swedish children with a mean age of 7-8 years. The study was conducted in a low-fluoridated areas. They concluded that 19% of children had "nonfluoride hypomineralizations" of permanent first molar and it required considerable treatment.

Weerheijm KL, Groen HJ, Beentjes VE, Poorterman JH (2001)[61] conducted a longitudinal standardized epidemiological survey to determine the prevalence of cheese Molars in 497 Dutch children, aged 11 years. The first permanent molars and central incisors were examined for hypoplasia, opacities, post eruptive enamel loss, premature extraction, and typical restorations. The results of this study showed that in 10% of these children, cheese molars were found.

Weerheijm K L, Jaleirk B, Alaluusua S (2001)[59] did a short communication study to describe briefly about a specific type of Enamel Hypomineralization. The authors concluded that a common consensus was adopted and the type of enamel hypomineralization with typical features affecting the permanent first molars and permanent Incisors was named as "Molar Incisor Hypomineralization".

Leppaniemi A, Lukinmaa PL, Alaluusua S (2001)[40] determined the prevalence of non-fluoride enamel hypomineralization in the first permanent molars in 488 children aged 7 – 13 years. The authors further examined the impact of such defects on the treatment needs by evaluating the number of carious lesions, restorations and extractions of the first permanent molars. They concluded that 19.3% children had non-fluoride hypomineralization. The severity of defects varied from mild lesions like color change to more severe ones. The authors also concluded that the non-fluoride hypomineralization have a significant impact on treatment need in the present child population with low caries activity.

Dietrich G, Sperling S, Hetzer G (2003)[17] evaluated the prevalence and the possible causes of the MIH in 2,408 children aged 10 -17 years living in Dresden (Germany). Enamel defects were recorded using the modified DDE index. They concluded that the overall prevalence of MIH was low and there was no association between peri and neonatal complications or other health problems and MIH.

Sui W, Boyd C, Wright J T (2003)[57] conducted a biochemical pH estimation study in mice to evaluate the effect of altered pH regulation on mineral content during enamel development. They hypothesized that the abnormal extracellular pH in the enamel matrix of mice with the cystic fibrosis gene knocked out (CF mice) resulted in altered enamel mineralization. The enamel matrix pH during amelogensis was studied in 10 normal and 10 CF mice. These results concluded that the CFTR gene (cystic fibrosis transmembrane conductance regulator) plays a main role in pH regulation during enamel development. They also stated that a reduced pH resulted in a lack of calcium during enamel maturation.

Lygidakis NA, Dimou G, Marinou D, Gouva G (2004)[43] assessed the potential medical etiological factors involved in the development of MIH. They examined 3,518

children aged 5.5 to 12 years and MIH was seen in 360 (10.2%) children. They concluded that the possible etiological medical factors could likely be neonatal illness, high fever, respiratory conditions, perinatal and postnatal problems.

Weerheijm K L (2004)[62] stated that childhood diseases could be a precursor for MIH and suggested that dentists should always plan a frequent monitoring of erupting first permanent molars. He also stated that the management of MIH included pain management followed by functional management with long term favorable prognosis.

Kosem R, Senk Erpic A, Kosir N, Kastelec D (2004)[38] evaluated the prevalence of enamel defects with emphasis on MIH in 2,339 children aged 12-18 years in Slovenian population. They concluded that 14% of children had at least 1 permanent first molar with "demarcated opacity".

Chawla N, Messer LB, Silva M (2004)[10] studied the distribution and etiological factors of molar-incisor hypomineralization. The authors conducted their study in Australian children who were attending a large pediatric dental specialist referral practice. They concluded that 70% of children had ≥1 affected permanent first molars.

William V, Messer B L, Burow M F (2006)[65] done a review and stated that the number of affected permanent first molars can vary from 1 – 4 depending on the severity. They also explained a 6 step approach for the management of MIH which included a)risk identification, b)early diagnosis, c)remineralization and demineralization, d)prevention of caries and post-eruptive breakdown, e)restorations and extractions and maintenance.

William V, Burrow MF, Palamara JE, Messer LB (2006)[64] investigated the adhesion property of resin composite bonded to enamel and hypomineralised enamel with an all etch single bottle adhesive or self-etching primer adhesion. They included 44 control enamel and 45 hypomineralised enamel and bonded with either 3M ESPE single bond or

clearfil SE bond. Enamel adhesive interface and etched enamel surfaces were viewed under scanning electron microscope. The results suggested that the micro shear bond strength of resin composite bonded to hypomineralised enamel was significantly lower than the control enamel.

Rodd HD, Boissonade FM, Day PF (2007)[51] determined the pulpal status of hypomineralised teeth in an in vitro trial. They included 25 healthy teeth in the control group and 19 hypomineralised permanent first molars. Pulp sections were processed using protein gene product 9.5 leukocyte common antigen, and Ulex europaeus lectin. The results showed that the innervation density was significantly greater in the pulp horn and subodontoblastic region of hypomineralised teeth compared to sound teeth. They also stated that vascularity was found to be similar for both hypomineralised and healthy teeth, but was greater in hypersensitive hypomineralised samples.

Heijs SC, Dietz W, Noren JG, Blanksma NG, Jalevik B (2007)[31] investigated the morphology and distribution of some inorganic elements in dentin with MIH in permanent first molars. Sixty four tooth sections from 32 children were examined in polarized light. 5 sections were used for Scanning Electron Microscope (SEM) and 10 were used for X-Ray Micro Analysis (XRMA). The XRMA analysis showed a difference in the concentration of elements between dentin below normal and hypomineralised enamel. The authors concluded that the odontoblasts were not affected in cases of MIH, but may be affected by hypocalcemia which was reflected by the presence of interglobular dentin.

Fagrell Tg, Lingstrom P, Olsson S, Steiniger F, Noren JG (2008)[20] did a histological study to investigate the bacterial invasion of dentinal tubules in an hypomineralised enamel in permanent first molars with MIH. Five extracted permanent first molars diagnosed with MIH were fixated, demineralized and sectioned in a bucco-lingual

direction in a microtome with a thickness of 4-5 micron. Later, the sections were stained using modified Brown and Benn staining for bacteria and analyzed under SEM. The analyzed results suggested that the presence of bacteria in the dentinal tubules and inflammatory reactions in the pulp revealed that oral bacteria like streptococci may penetrate through the hypomineralised enamel into the dentin contributing to hypersensitity of teeth with MIH.

Chawla N, Messer LB, Silva M (2008)[10] examined the perinatal and medical histories in 416 children aged 6 – 14 years for putative associations with MIH. A questionnaire regarding perinatal and medical histories was sent to the parents and reviewed. The authors concluded that in MIH children combinations of ear infections, fevers, perinatal conditions and other illness seem to be occurring in child's first 3 years

Chawla N, Messer LB, Silva M (2008)[11] examined the records of 182 children aged 6 to 14 years with MIH in order to develop and examine a Hypomineralization severity index for permanent first molars. The records of 429 first permanent molars in MIH children were examined and scored for eruption status, extent of hypomineralization, sensitivity, number of restorative treatments. The summoned scores were converted to an index for each dentition and they concluded that the index has indicated to assess the severity of hypomineralization of the molars.

Chan YL, Ngan AH, King NM (2010)[9] stated that the quality of the enamel adjacent to the defects in teeth with MIH limits the success rate of the restorations placed in these teeth. The authors compared the microstructure and mechanical properties of two MIH teeth with Nanoindentation, bend tests on micro – cantilevers and transmission electron microscope (TEM). The enamel in the transitional region have prism sheaths that were

significantly lesser mineralized than unaffected enamel. They were weaker in holding prisms together when measured using bend tests on micro-cantilever.

Fagrell T (2011)[19] in his literature review enhanced the understanding of MIH in areas of the histological, chemical and mechanical properties of the hypomineralized enamel. The author stated certain clinical implications like the lower hardness value in hypomineralized enamel authenticates the posteruptive breakdown of MIH teeth, which is one of several clinical objective symptoms of MIH. He also quoted that that an ozone treatment kills oral bacteria involved in the caries process in vitro. Therefore, ozone treatment of MIH teeth should be considered, especially with symptoms of hypersensitivity.

Garg N, Jain AK, Saha S, Singh J (2012)[26] stated in their review article that MIH children should be monitored on their eruption of their permanent first molar so that remineralization and preventive measures can be instituted as soon as the affected surfaces are accessible.

Stefano M, Gugielmo C, Laura S, Alessandro V, Cagetti MG (2012)[55] reported a case report of a 17 year old male with a moderate MIH. The patient was treated with GC tooth mousse with Casein Phosphopeptide – Amorphous Calcium Phosphate (CCP-ACP) and bleaching agent. The authors stated that the combined use of CPP-ACP and hydrogen peroxide after a follow up of 5 months was found to be effective and safe for teeth with MIH.

Ozqul BM, Saat S, Sonmez H, Oz FT (2013)[47] evaluated the hypersensitivity observed in MIH-affected teeth and the effect of desensitizing agents applied with and without ozone to incisors affected by MIH. They included 120 teeth from 42 patients with MIH and treated them with fluoride, CPP-ACP and CPP-ACP with fluoride. The results of this study revealed that girls exhibited significantly more sensitivity compared with boys.

Desensitizing agents effectively reduced the hypersensitivity of teeth with MIH. CPP-ACP paste was found to be more effective, and ozone therapy prolonged the effect of CPP-ACP paste.

Fagrell TG, Salmon P, Melin L, Norén JG (2013)[21] estimated the onset and timing of the MIH and related the hypomineralized enamel to the incremental lines. The study included 13 extracted permanent first molars, which were analyzed with light microscopy and XMCT. The hypomineralized areas were mainly located in the mesio-buccal cusps, starting at the enamel-dentin-junction and continuing towards the enamel surface. The findings indicated that the ameloblasts in the hypomineralized enamel are capable of forming an enamel of normal thickness, but with a substantial reduction of their capacity for maturation of enamel. Chronologically, it is estimated that the timing of the disturbance is at a period during the first 6-7 months of age.

Kuhnisch J, Mach D, Thiering E, Brockow I, Hoffmann U, Neumann C, Heinrich-Weltzien R, Bauer CP, Berdel D, von Berg A, Koletzko S, Garcia-Godoy F,,Hickel R, Heinrich J (2014)[39] evaluated the association of MIH with prospective potential causative factors like respiratory diseases, breastfeeding, maternal smoking and parental education. The study included a total of 692 children aged 10 years old and they were examined using the EAPD criteria. They stated that there was no correlation between breast feeding and MIH but early respiratory diseases seemed to be directly or indirectly related to MIH.

Bhaskar SA, Hegde S (2014)[7] did a cross sectional descriptional study to assess the prevalence, clinical characteristics, distribution, severity and association with caries of MIH defects in Udaipur, Rajasthan. The study included 1173 children aged 8-13 years selected by random sampling procedure and examined under EAPD and WHO criteria. On

the basis of their results, they concluded that MIH was observed in about 10% of the children and first permanent molar appeared to be more vulnerable to early caries.

Loli D, Costacurta M, Maturo P, Docimo R (2015)[41] did a retrospective case control study to evaluate the correlation between the use of aerosol therapy in early childhood and the presence of MIH. The study included children aged 6 to 13 years and the data about the aerosol therapy (used for respiratory diseases) and the presence of MIH were obtained respectively by medical history and intraoral clinical examination. They stated that aerosol therapy carried out in early childhood appears to be a risk factor for the development of MIH, particularly in male subjects.

Americano GC, Jacobsen PE, Soviero VM, Haubek D (2016)[3] did a systematic review to assess the association between MIH and caries. They included children with all ages with MIH and caries in permanent dentition. The search was performed in PubMed and was limited to the period from January 2003 to November 2015. Seventeen publications were compiled in the review. On basis of the literature the authors concluded that there was a significant association between MIH and caries.

Wuollet E, Laisi S, Salmela E, Ess A, Alaluusua S (2016)[67] assessed whether childhood illnesses or medication were associated with MIH in first permanent molars and incisors in 287 Finnish children. EAPD criteria was used for examining MIH. Health data from the first 3 years of life were collected from medical records and the associations with MIH were assessed using simple and multiple logistic regression analyses. The authors concluded that acute otitis media and the use of certain antibiotics were associated with the elevated risk of MIH.

Dantas-Neta NB, Moura LF, Cruz PF, Moura MS, Paiva SM, Martins CC, Lima MD (2016)[15] did a cross sectional study to evaluate the impact of MIH on oral

health-related quality of life (OHRQoL) in a 594 schoolchildren between 11 to 14 years of age. Their parents/caregivers were asked to answer the questionnaires being provided. It includes experience of dental caries, malocclusion, and socioeconomic status were treated as confounding variables. The results suggested that the schoolchildren with severe MIH had a greater negative impact on the oral symptom and functional limitation domains than those without MIH. According to parents/caregivers perceptions, schoolchildren with severe MIH had a greater negative impact on the functional limitation domain than those without MIH.

METHODOLOGY

The present cross sectional study was conducted from the Department of Pediatric and Preventive Dentistry, K.S.R Institute of Dental Science and Research (KSRIDSR). The study was planned and organized in association with various schools in Tiruchengode, western TamilNadu in South India, to determine the prevalence, caries lesion severity and salivary characteristics of Molar Incisor Hypomineralization (MIH) in children. The study design and protocol was analyzed and approved by the Institutional Review Board and Institutional Ethics Committee of KSRIDSR, Tiruchengode, TamilNadu. The purpose of the study was explained to the school authorities and their approval was obtained. A written consent in mother tongue (Tamil) was also obtained from the parents of the children who participated in the study.

Armaterium used

1. Diagnostic instrument set – consisting of a mouth mirror, explorer and tweezer in kidney tray.
2. Disposable mouth masks.
3. Disposable gloves.
4. Cotton holders.
5. Modelling Wax No.2 (Hindustan, Hyderabad)
6. Sterile sample container (uricups).
7. 3.0 ml disposable pipette.
8. 5.0 ml glass measuring pipette.
9. 5.0 ml disposable test tubes with caps.
10. 2-octanol solution (500ml). (Himedia, All India laboratories)
11. Hannah pH meter. (Oakton, pH tester 20, Singapore)

12. Neutral pH -4 and pH 7 tablets. (Merck, Mumbai)
13. Disposable sterile tooth picks
14. Distilled water
15. 0.005 mol/litre HCL

Sample selection

The study population included children aged 8 to 12 years belonging to 7 private and 7 government schools of Thiruchengode.

Inclusion criteria

1. Children with fully erupted index teeth (all first permanent molars and incisors).

Exclusion criteria

1. Children with special health care needs
2. Children with other dental defects like amelogensis imperfecta, turner's hypoplasia.
3. Children with medical illness at the time of examination since medications alter the salivary flow.
4. Children who did not cooperate for saliva testing.
5. Children with fixed appliances that interfere with index teeth examination.
6. Children with dental fluorosis.

Clinical examination

Clinical examination was done in school premises. The children were seated on the chair and subjected to dental examination under the natural sun light. Hypo-mineralized permanent molars and incisors were diagnosed clinically based on the **European Academy Pediatric Dentistry (EAPD) criteria recommended in 2003[60]** and revised at an **Interim Seminar and Workshop concerning MIH organized by the EAPD in 2009[44].** The index

teeth (four first permanent molars (FPMs) and eight permanent incisors) were kept wet for examination to distinguish opacities from incipient carious lesions. All clearly visible opacities were measured and recorded. All examinations were carried out by one investigator.

Detection of caries

A full-mouth caries assessment was performed using **The International Caries Detection and Assessment System (ICDAS II)** scoring criteria. A visual tactile caries assessment of four permanent and incisors was performed, supplemented by natural sun light with a mouth mirror. Cotton rolls were used to wet and dry the teeth surfaces. The Shepherd hook explorer was used to remove any remaining plaque or debris and detect surface contour, enamel, or dentine cavitation, the presence or absence of sealants, or restorations. Data were recorded for all permanent molars and incisors.

Assessment of saliva and plaque

Whole saliva was stimulated by asking the children to chew a piece of modelling wax which is made in the form of pellet for 3 min. To alleviate potential inconvenience in chewing modelling wax, a demonstration was done. This familiarized the procedure to the participants. Participants were assured that the procedure was not invasive to eliminate dental treatment-related anxiety that may affect saliva production. The seating position for the child was in an upright relaxed position under natural light following the criteria of **Lussi (1996)[42].** The stimulated salivary specimens were collected from children in a sterile container for determination of pH and buffering capacity. The saliva from 100 normal children (without caries) and 50 children with MIH and 50 children with MH of same age and sex were collected by convenience sampling method and analyzed. Plaque was collected with sterile toothpicks from those 200 samples and stored in a sterile container. The

collected plaque is diluted in 20 ml of distilled water and measured using Hannah pH meter. The collected samples were then tested, within 1 hour of time **(Henson BS and Wong DT 2010)[32].** Saliva (stimulated) and plaque samples were then subjected to pH measurements and buffer capacity to obtain their base line values.

By using a standard protocol, the pH meter was standardized with pH calibration solutions ranging from pH 4, 7 and 10. The head of the pH bulb was immersed in the calibration solution, until the pH of the solution was determined correctly in all the three ranges. The manufacturer claims that the pH meter must be stored in the storage solution when not utilized. For saliva and plaque pH and buffer measurements, the head of pH bulb was completely immersed into the sample. After a few minutes of waiting, the readings stop to fluctuate. The values displayed digitally were recorded.

For determination of salivary buffering capacity, the classical **Ericson's test (1959)** was used. They recommended to use 0.005 mol per L of HCl for stimulated saliva. The desired HCl was calculated by using Solcalc software (solution calculator Inc). To prepare 1000ml of a 0.005 mol per L of HCl, 0.14 ml of 37.2% HCl was added with distilled water. The hydrochloric acid was collected by using micropipette to obtain accurate volume for preparation.

For buffering capacity 1.0ml of the saliva is transferred to 3.0 ml of 0.005 mol per L. One drop of Octanol was added to prevent the foaming reaction and the sample was mixed for 20 minutes to remove carbondioxde. Finally the buffering capacity was evaluated electrometrically by Hannah pH meter. The inference for the pH values were decoded using the following criteria.

Final pH value	Evaluation
More than 6.50	High
5.75-6.50	Normal
4.00-5.74	Low
Less than 4.00	Very low

FLOW CHART OF METHODOLOGY

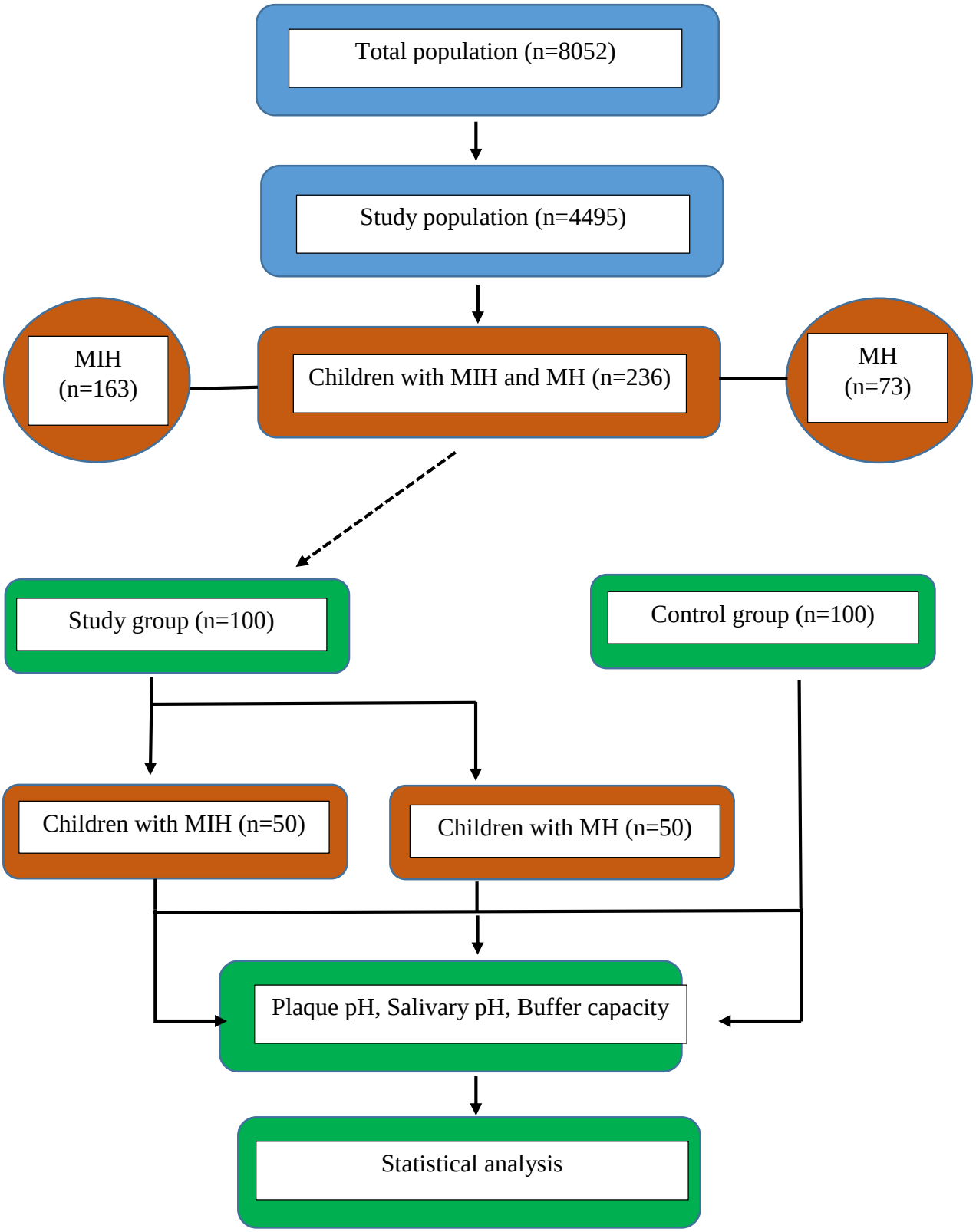


Figure 1: Armamentarium

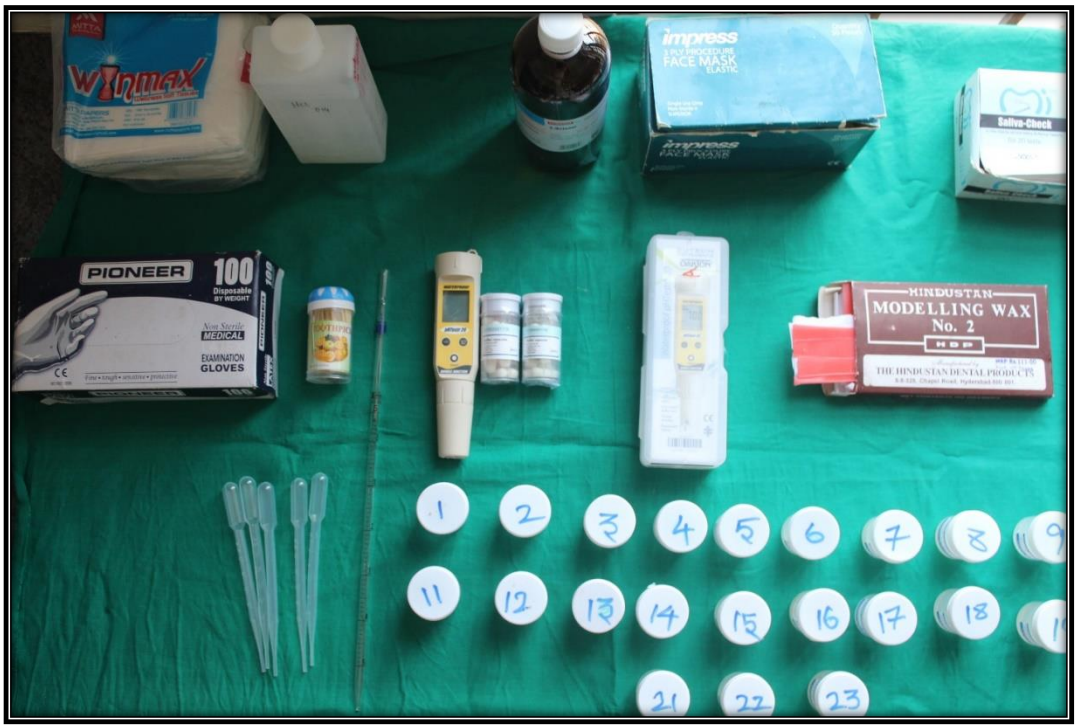


Figure 2: Hannah pH meter and buffer capsules (4 and 7)

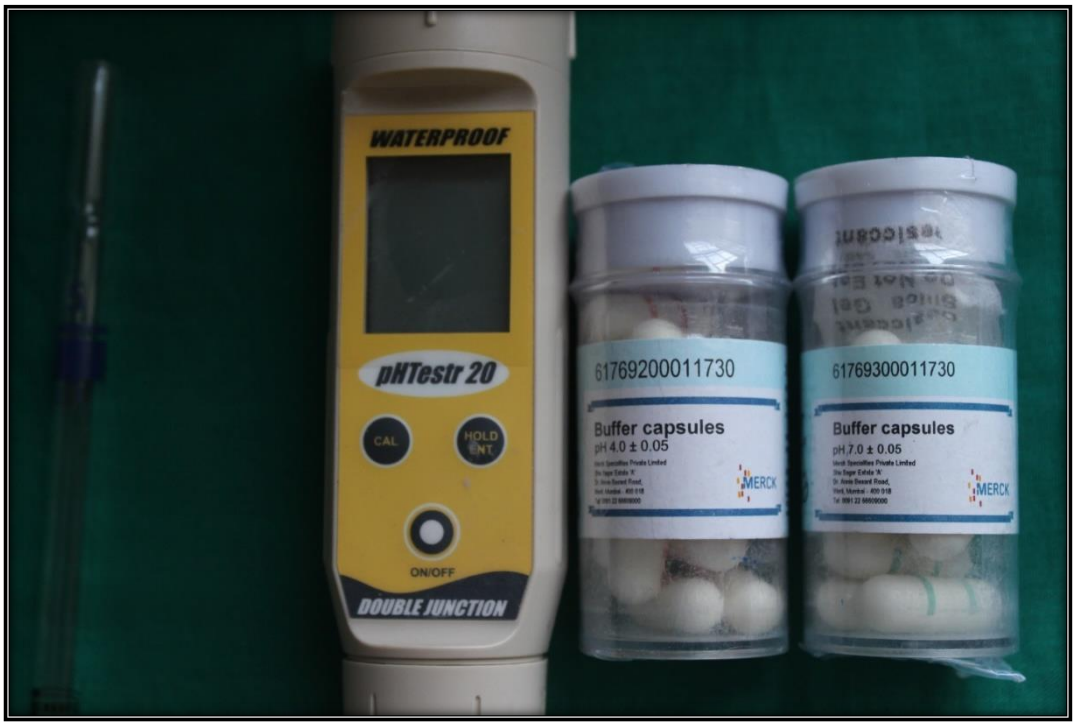


Figure 3 & 4: Sterile sample container were distributed for collection of saliva

Figure 5: Monitoring children while chewing wax

Figure 6: Saliva sample collection by spitting method

Figure 7: Collection of plaque using sterile tooth pick

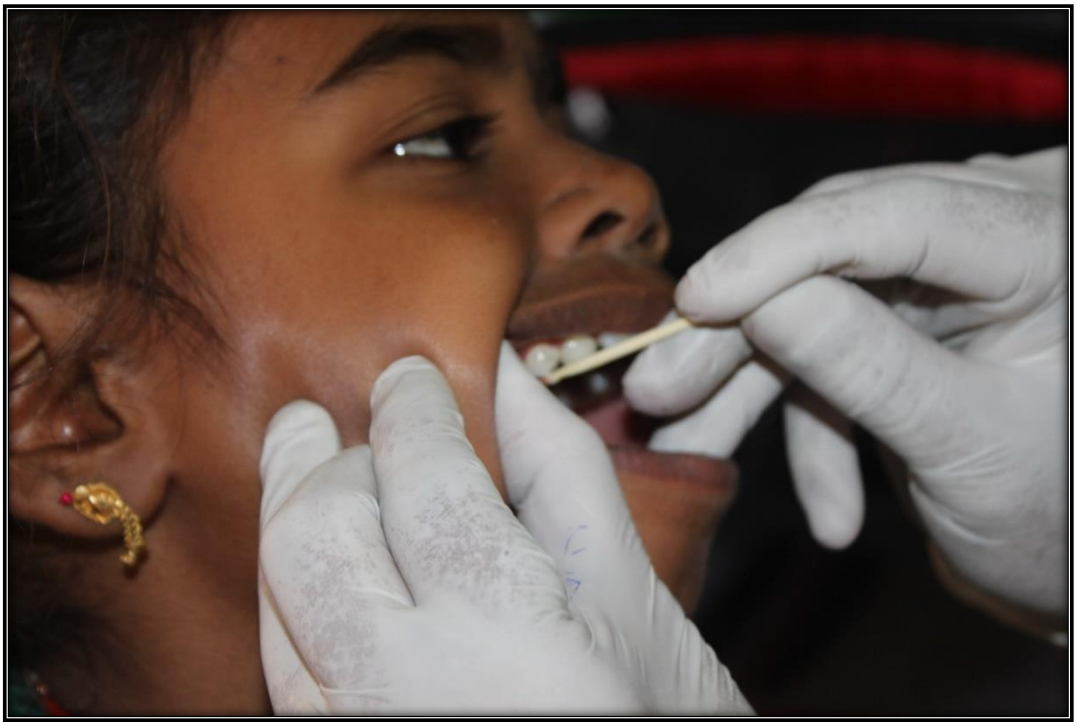


Figure 8: Hannah pH meter used for checking salivary characteristics and plaque pH

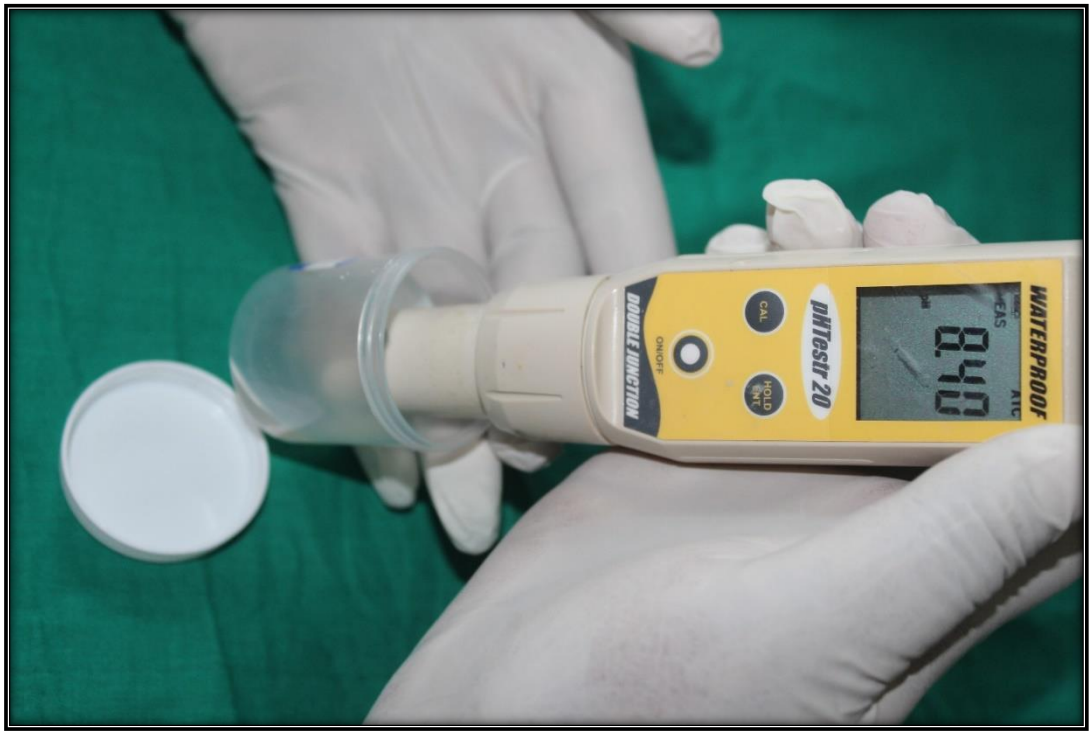
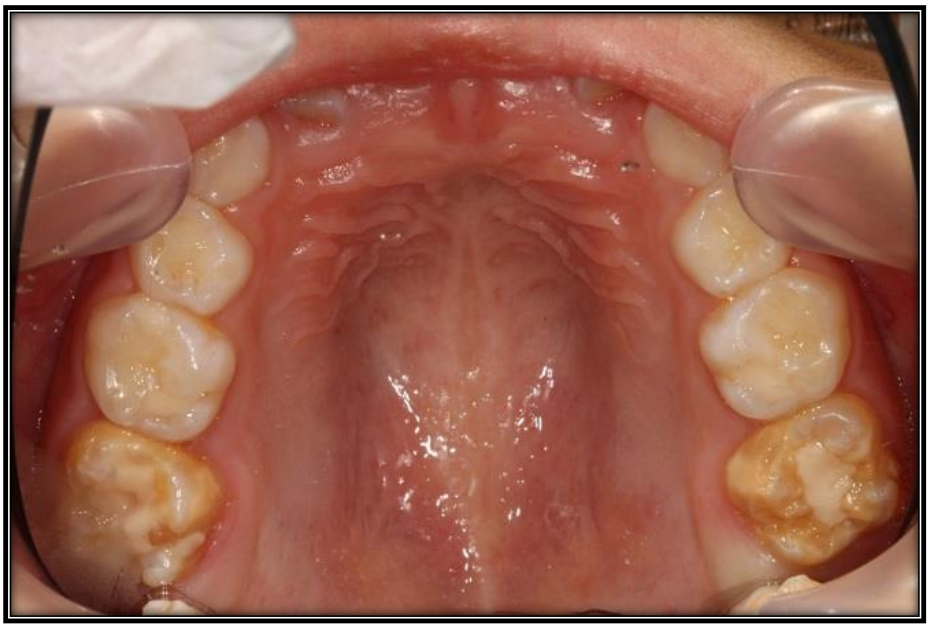
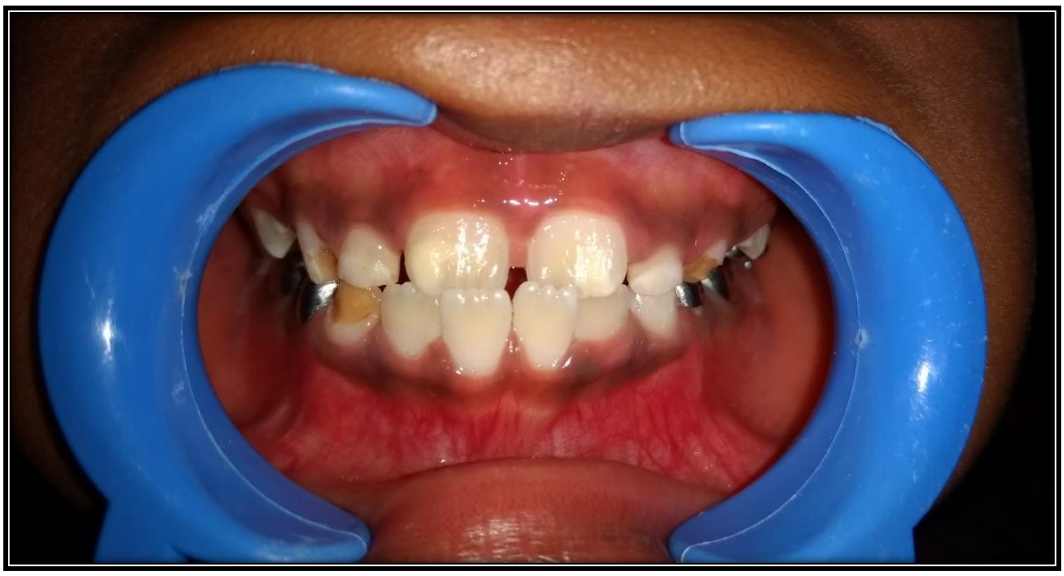


Figure 9 and 10: Molar Incisor Hypomineralization





RESULTS

Table 1. Sample distribution

Schools	Population screened	Study population	Boys	Girls
A	1800	994	520	474
B	1725	861	478	383
C	842	483	268	215
D	198	77	44	33
E	697	322	147	175
F	442	271	148	123
G	1500	826	364	386
H	146	128	60	68
I	46	38	20	18
J	30	15	7	8
K	150	130	72	58
L	134	123	58	65
M	162	145	62	83
N	180	158	85	73
Total	8052	4495	2333	2162

Table 1 shows the distribution of the school children in Tiruchengode district. Of 8052 school children screened, 55.8% (n=4495) children aged 8 to 12 years were included in the study. Among the study population, 51.9% (n=2333) were boys and 48% (n=2162) were girls.

Table 2. Descriptive statistics of Molar Hypomineralization and Molar Incisor Hypomineralization

Schools	Population screened	Study population	MH/MIH	Boys	Girls
A	1800	994	24	16	8
B	1725	861	26	18	8
C	842	483	17	12	5
D	198	77	5	3	2
E	697	322	9	7	2
F	442	271	13	4	9
G	1500	826	34	19	15
H	146	128	20	15	5
I	46	38	16	10	6
J	30	15	3	3	0
K	150	130	14	11	3
L	134	123	10	4	6
M	162	145	20	13	7
N	180	158	25	15	10
Total	8052	4495	236	150	86

Table 2 shows the descriptive data of total enamel defects including MH and MIH. 5.25% (n=236) of children had enamel defects, of 63.5% (n=150) of them were boys and 36.4% (n=86) were girls.

Table 3. Descriptive statistics of Molar Hypomineralization (MH)

Schools	Population screened	Study population	MH	Boys	Girls
A	1800	994	10	6	4
B	1725	861	10	5	5
C	842	483	8	7	1
D	198	77	0	0	0
E	697	322	2	2	0
F	442	271	1	0	1
G	1500	826	11	7	4
H	146	128	5	4	1
I	46	38	0	0	0
J	30	15	0	0	0
K	150	130	7	5	2
L	134	123	2	0	2
M	162	145	7	6	1
N	180	158	10	6	4
Total	8052	4495	73	48	25

Table 3 shows the prevalence of MH among the study population of 4495 children. 1.62% (n=73) children were found to have MH in which 1.06% (n=48) were boys and 0.55% (n=25) were girls.

Table 4. Descriptive statistics of Molar Incisor Hypomineralization (MIH)

Schools	Population screened	Study population	MIH	Boys	Girls
A	1800	994	14	10	4
B	1725	861	16	13	3
C	842	483	9	5	4
D	198	77	5	3	2
E	697	322	7	5	2
F	442	271	12	4	8
G	1500	826	23	12	11
H	146	128	15	11	4
I	46	38	16	10	6
J	30	15	3	3	0
K	150	130	7	6	1
L	134	123	8	4	4
M	162	145	13	7	6
N	180	158	15	9	6
Total	8052	4495	163	102	61

Table 4 shows the prevalence of MIH among the study population of 4495 children. 3.62% (n=163) children were found to have MIH in which 62.5% (n=102) were boys and 37.4% (n=61) were girls.

Table 5. Comparison of prevalence of MH and MIH between genders

Gender	Study population	Children with MH and MIH	p value*
Boys	2333	150 (6.4%)	**0.001**
Girls	2162	86 (3.9%)	
Total	4495	236 (5.2%)	

***Pearson Chi-Square test

Table 5 / Graph 1 shows the prevalence of MH and MIH in boys and girls. Boys [n=150; (6.4%)] had more hypomineralization (MH/MIH) when compared to girls [n=86; (3.9%)]. The scores suggested that there was a significant difference between the genders.

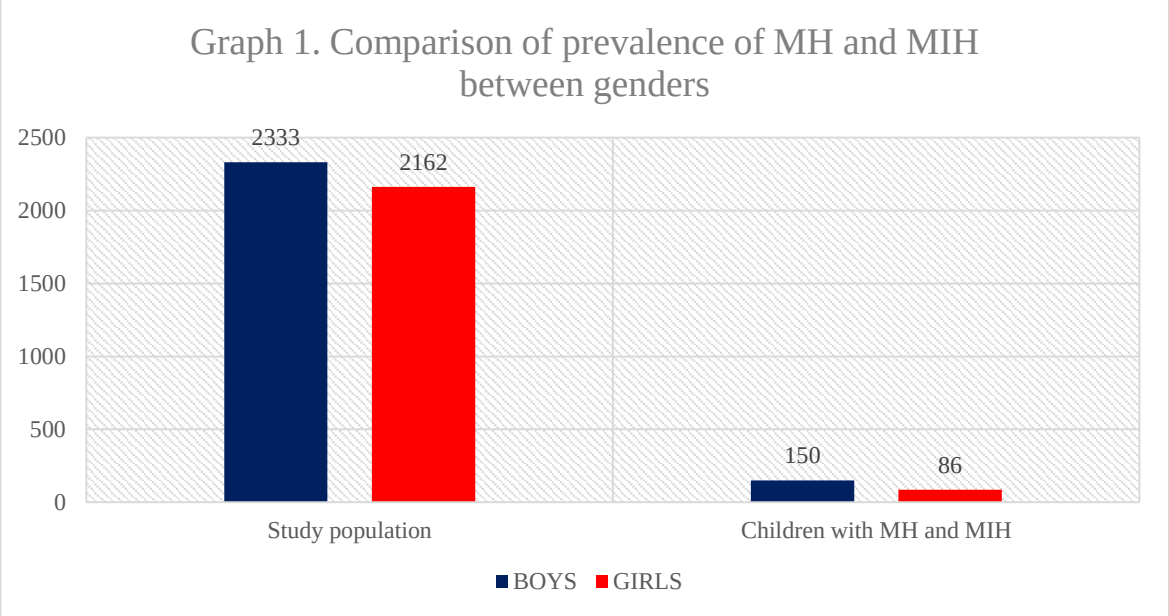


Table 6. Comparison of prevalence of Molar Hypomineralization between genders

Gender	Study population	Children with MH	p value*
Boys	2333	48 (2.0%)	**0.019**
Girls	2162	25 (1.1%)	
Total	4495	73 (1.6%)	

*Pearson Chi-Square test

Table 6 / Graph 2 shows the comparison of prevalence of MH between boys and girls. Boys [n=48; (2.0%)] had more MH when compared to girls [n=25; (1.1%)]. The prevalence shows that there was a significant difference between the genders.

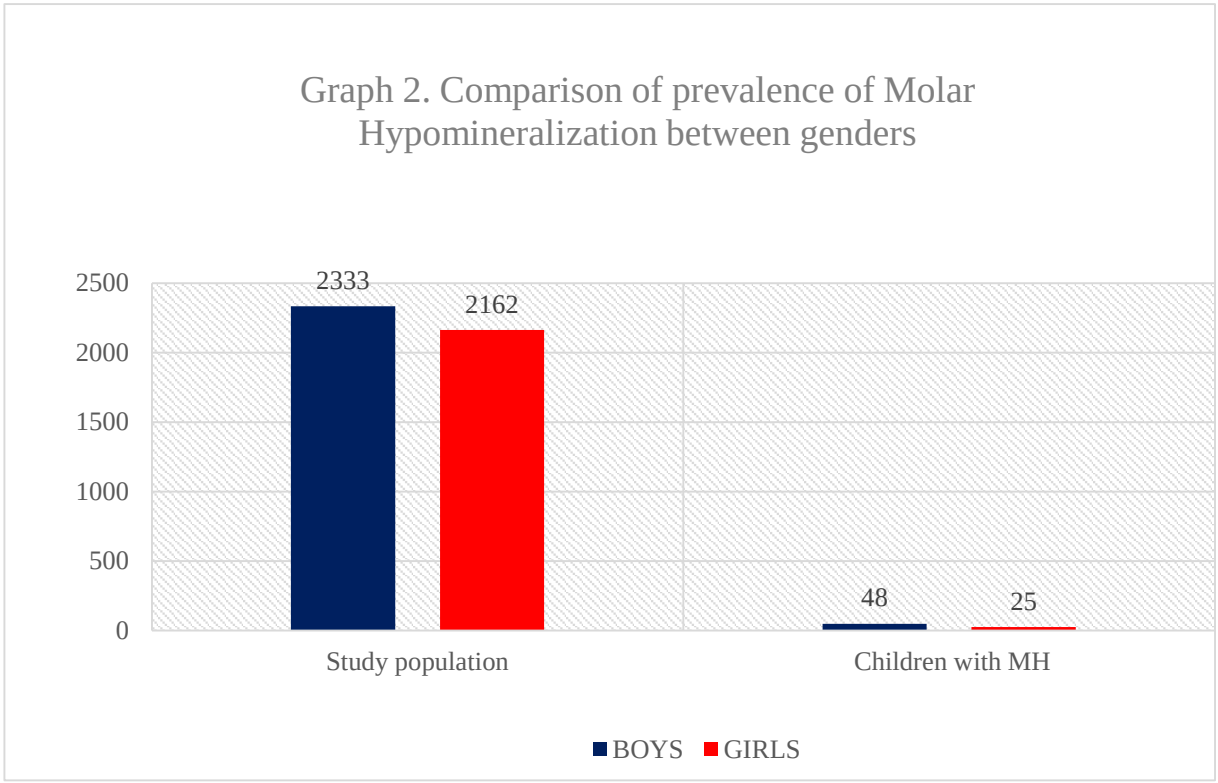


Table 7. Comparison of prevalence of Molar Incisor Hypomineralization between genders

Gender	Study population	Children with MIH	p value*
Boys	2333	102 (4.3%)	**0.007**
Girls	2162	61 (2.8%)	
Total	4495	163 (3.6%)	

*Pearson Chi-Square test

Table 7 / Graph 3 shows the comparison of prevalence of MIH between boys and girls. Boys [n=102; 4.3%)] had more MIH when compared to girls [(n=61; 2.8%)]. The prevalence shows that there was a significant difference between the genders.

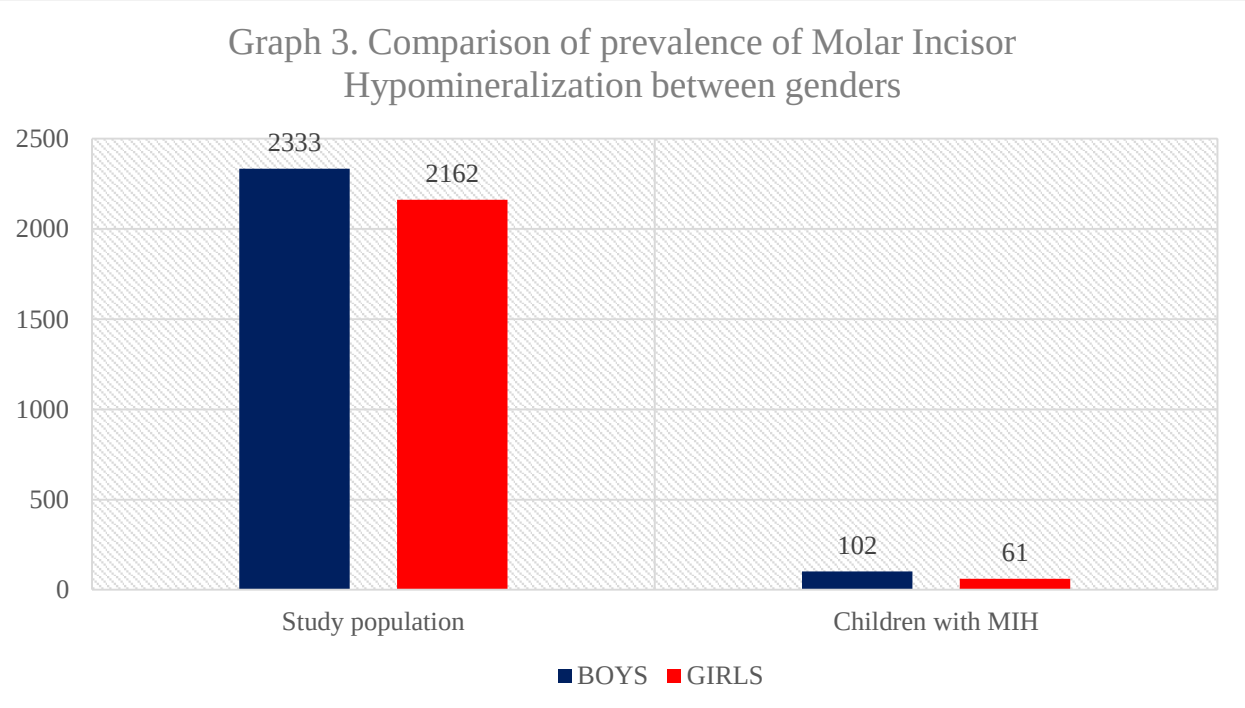


Table 8. Comparison of mean plaque pH between Molar Hypomineralization group and control group

Plaque pH (MH)	N	Mean ± SD	p value*
Cohort of children with MH	50	7.03 ± 1.29	**0.001**
Control group without MH	50	5.60 ± 1.48	

*****Student t- test

Table 8 shows the comparison of mean plaque pH between children with and without MH. A significant difference (p=0.001) was observed between the study group and control group. The mean plaque pH of children with MH (7.03 ± 1.29) was relatively higher than the control group (5.60 ± 1.48).

Table 9. Comparison of mean plaque pH between Molar Incisor Hypomineralization group and control group

Plaque pH (MIH)	N	Mean ± SD	p value*
Cohort of children with MIH	50	6.91 ± 1.46	**0.001**
Control group without MIH	50	5.25 ± 1.51	

*****Student t- test

Table 9 shows the comparison of mean plaque pH between children with and without MIH. A significant difference (p=0.001) was observed between the study group and control group. The mean plaque pH of children with MIH (6.91 ± 1.46) scored relatively higher than the control group (5.25 ± 1.51).

Table 10. Comparison of mean salivary pH between Molar Hypomineralization group and control group

Salivary pH (MH)	N	Mean ±SD	p value*
Cohort of children with MH	50	6.72± 1.59	**0.001**
Control group without MH	50	8.37 ± 0.65	

*****Student t- test

Table 10 shows the comparison of mean salivary pH between children with and without MH. A significant difference (p=0.001) was observed between the study group and control group. The mean salivary pH of children with MH (6.72 ± 1.59) scored relatively lower than the control group (8.37 ± 0.65).

Table 11. Comparison of mean salivary pH between Molar Incisor Hypomineralization group and control group

Salivary pH (MIH)	N	Mean ±SD	p value*
Cohort of children with MIH	50	5.92 ± 1.56	**0.001**
Control group without MIH	50	8.54 ± 0.73	

*****Student t- test

Table 11 shows the comparison of mean salivary pH between children with and without MIH. A significant difference (p=0.001) was observed between the study group and control group. The mean salivary pH of children with MIH (5.92 ± 1.56) scored relatively lower than the control group (8.54 ± 0.73).

Table 12. Comparison of mean salivary buffer capacity between Molar Hypomineralization group and control group

Salivary buffer capacity (MH)	N	Mean ±SD	p value*
Cohort of children with MH	50	5.79 ± 1.97	0.605
Control group without MH	50	5.58 ± 2.16	

*****Student t- test

Table 12 shows the comparison of mean salivary buffer capacity of children with and without MH. No significant difference was seen between the study group (5.79 ± 1.97) and control group (5.58 ± 2.16).

Table 13. Comparison of mean salivary buffer capacity between Molar Incisor Hypomineralization group and control group

Salivary buffer capacity (MIH)	N	Mean ± SD	p value*
Cohort of children with MIH	50	5.65 ± 1.94	0.114
Control group without MIH	50	5.03 ± 2.20	

*****Student t- test

Table 13 shows the mean comparison of mean salivary buffer capacity of children with and without MIH. No significant difference was seen between the study group (5.65 ± 1.94) and control group (5.03 ± 2.20)

Table 14. Comparison of mean plaque pH, salivary pH and salivary buffer capacity between Molar Hypomineralization and Molar Incisor Hypomineralization group

Group	MH (n=50)	MIH (n=50)	p value*
Salivary pH (Mean ± SD)	6.72± 1.59	5.92 ± 1.56	**0.001**
Buffer capacity (Mean ± SD)	5.79 ± 1.97	5.65 ± 1.94	**0.001**
Plaque pH (Mean ± SD)	7.03 ± 1.29	6.91 ± 1.46	**0.001**

***Student t- test**

Table 14 / Graph 4 shows the comparison of mean plaque pH, salivary pH and salivary buffer capacity between MH group and MIH group. A significant difference (p=0.001) was observed in all the parameters between MH and MIH group.

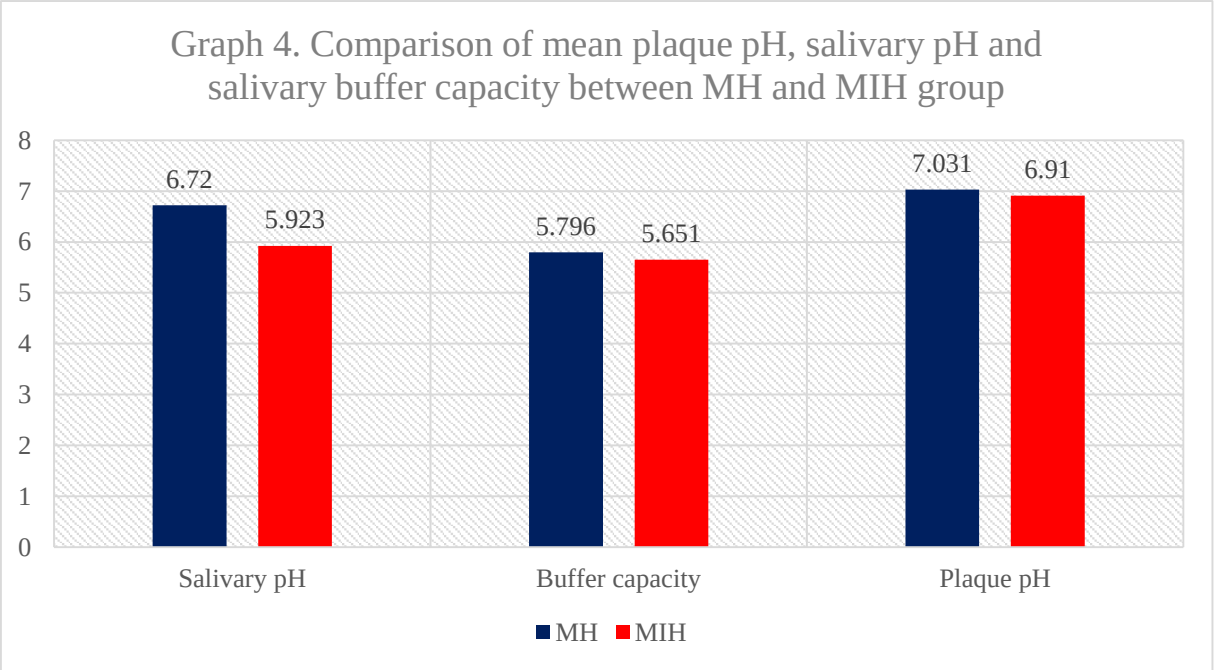


Table 15. Descriptive statistics of Caries status in children with Molar Hypomineralization and Molar Incisor Hypomineralization

Caries severity	MH	MIH	MH/MIH
With caries	36 (49.3%)	87 (53.3%)	123
Without caries	37 (50.6%)	76 (46.6%)	113
Total	73	163	236

Table 15 shows the descriptive data of caries status in children with MH and MIH. The caries prevalence in children with MH was 49.3% (n=36) whereas in the children with MIH the prevalence was 53.3% (n=87)

Table 16. Comparison of dental caries in Molar Hypomineralization and Molar Incisor Hypomineralization group in genders

Gender	Dental caries		p value*
	Number of children	Number of Carious teeth	
Boys	70 (56.9%)	93	0.263
Girls	53 (43%)	47	
Total	123	140	

***** Pearson Chi-Square test

Table 16 / Graph 5 depicts the prevalence of dental caries in MH and MIH children. Among 236 children with MH and MIH, 123 children had dental caries. In the 123 children with dental caries, 140 teeth were affected. There was no significant difference in the caries status between the genders.

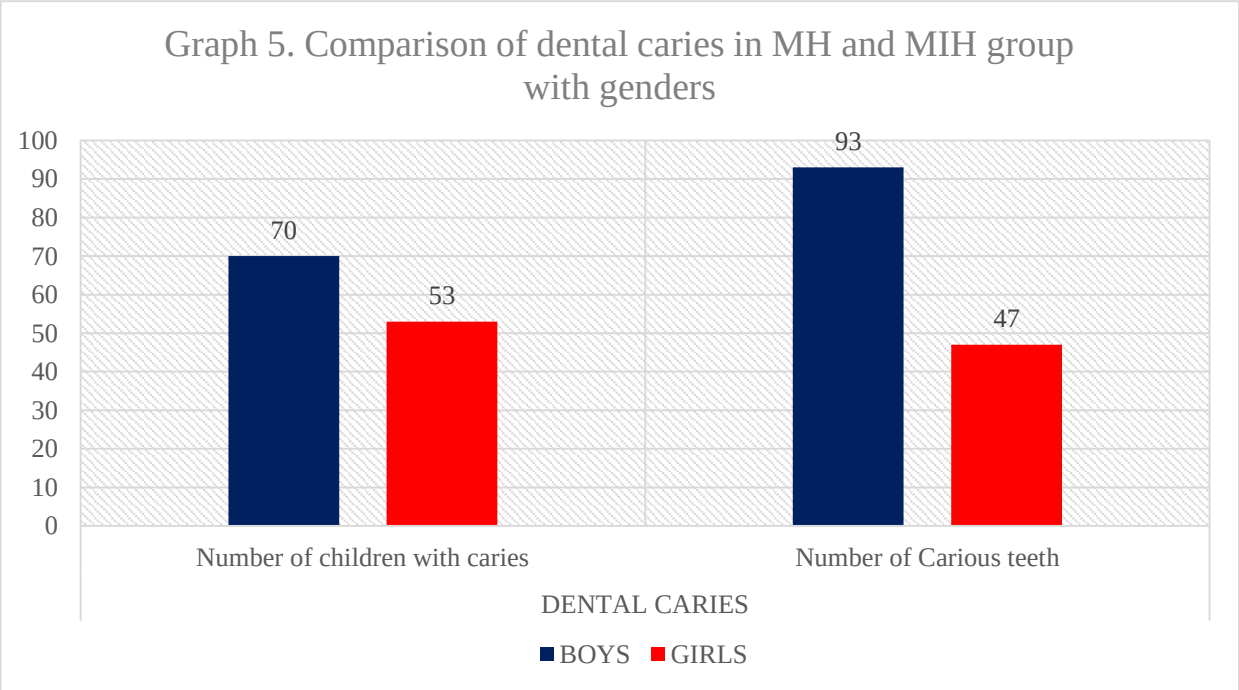


Table 17. Descriptive statistics of severity of dental caries according to ICDAS II

Caries severity (ICDAS II)	Number of teeth
CODE6	18
CODE5	24
CODE4	38
CODE3	35
CODE2	11
CODE0	14
Total	140

Table 17 shows the severity of caries according to ICDAS II in children with MH and MIH. Code 3 and 4 were the most common form of caries severity seen.

DISCUSSION

Dental caries and developmental enamel defects are very common in children. Developmental enamel defects are still an enigma both in scientific aspects as well as in clinical practice. One developmental defect of great clinical importance is Molar Incisor Hypomineralization (MIH). It was first acknowledged in Sweden in late 1970, however this term was introduced by **Weerheijm et al** in 2001[59]. MIH describes the clinical picture of hypomineralization of systemic origin of one or more permanent molars, as well as any associated incisors.

In national epidemiological surveys on caries prevalence, children are normally not screened for the presence of MIH. So little is known about its occurrence. Various studies done worldwide had shown the prevalence of MIH ranging from 2.4% to 40.2% **(Jalevik B 2010, Lygidakis NA 2010)[34, 44].** MIH is worthy of further investigation since it is widespread and majority of clinicians perceive MIH to be a clinical problem, with insufficient data.

Thus this cross sectional study provides the information on the prevalence, distribution, caries severity and salivary characteristics of MIH population in Tiruchengode district. The study sample included children from 14 schools around Tiruchengode district. Children aged 8 to 12 years was selected for this study. The age from 8 years to 12 years is considered the best time for examination for MIH because, at this age, most children have all four permanent first molars and most of the incisor teeth erupted **(Weerheijm KL, 2003, Lygidakis NA et al 2010)[63, 44].**

DIAGNOSTIC CRITERIA

Dental examination was carried out by cleaning and wetting the teeth, to detect the presence of enamel defects and to allow distinction between diffuse opacities, demarcated opacities and white spot carious lesions. Different criteria have been used to record MIH like DDE index, Modified DDE index and diagnostic criteria (**Alalausua 1996)**[1]. In the present study, the **EAPD criteria** established in 2003 and further revised in 2009 was used to record MIH[60, 44].

PREVALENCE AND DISTRIBUTION

MIH is recognized as a global dental problem and epidemiologic reports from all over the world are continuously published **(Jalevik 2010)**[34]**.** The prevalence varies considerably from a few percent in China **(Cho et al. 2008)**[12] to almost 40% percent in Denmark and Brazil **(Wogelius et al. 2008; Soviero et al. 2009)**[54]**.** The large variations in the prevalence rates of MIH may reflect real differences between regions and countries, differences in sample sizes, differences in recording methods, indices and diagnostic criteria used and the age groups studied. Often, the presence of dental caries may mask the true prevalence of MIH. When older children are studied, occlusal wear and restorations could have superimposed the developmental defects **(Crombie F 2009, Willmott N 2011, Jalevik 2010)**[13, 66 34]**.**

The prevalence of MIH and MH in the present study group was observed to be 5.25% (n=236) on a whole study population (n=4495). Among the study population, 3.33% (n=150) were boys and 1.91% (n=86) were girls.

Bhaskar SA et al (2014)[7] stated a prevalence rate of 9.46% among 8 to 13 years old children in Udaipur, India which was very similar to the study done by **Parikh DR et al (2012)**[48] with a prevalence rate of 9.2% among 8 to 12 years in Gujarat, India.

In the study population, boys (6.42%) were found to have significantly higher prevalence of MIH and MH compared to girls (3.97%). Among the 236 children with MIH and MH, 63.5% of them were boys and 36.5% of them were girls. **Preusser SE 2007[49], Soviero V 2009[54], da Costa-Silva CM 2010[14], Ghanim A 2011[28], Parikh DR 2012[48]** also found slightly higher prevalence among boys in Germany, Brazil, Iraq, Gujarat respectively. However **Chawla N 2008[10], Zawaideh FI 2011[68]** have reported a higher prevalence among girls in Australian and Jordanian children respectively.

Weerheijm KL et al. 2001[59] stated Molar Hypomineralization (MH) as a qualitative defect which includes only the first four permanent molars excluding the incisors. A majority of the children screened for MIH had only hypomineralised molars (MH) and this finding was similar to **Jasulaityte l 2007, Lygidakis NA 2008, Zawaideh FI 2011** in Lithuanian, Greece and Jordanian populations[68]. In our study prevalence of MH was found to be 1.62% which was comparatively lower than the MIH frequency of about 3.62%.

The increased prevalence of MH and MIH was seen in older children of aged 11 to 12 years. This may be due to easier recognition and diagnosis once post-eruptive discoloration and breakdown has occurred in mild defects that might otherwise go unnoticed in younger children **(Lygidakis NA 2008, Chawla N 2008)[11].**

SALIVARY pH AND PLAQUE pH

Saliva is a dilute fluid, over 99% being made up of water. Whole saliva collected from the mouth is a complex mixture. Saliva has a pH normal range of 6.2 to 7.6 with 6.7 being the average pH. Resting pH of mouth does not fall below 6.3. In the oral cavity, the pH is maintained near neutrality (6.7 to 7.3) by saliva. The saliva contributes to maintenance of the pH by two mechanisms. First, the flow of saliva eliminates carbohydrates that could be metabolized by bacteria and removes acids produced by bacteria. Second, acidity from

drinks and foods, as well as from bacterial activity, is neutralized by the buffering activity of saliva.

Baliga et al. 2014[5] stated 3 conditions of salivary pH in accordance to the neutral pH of 7. A saliva pH of 7.0 usually indicates a healthy dental and periodontal situation. At this pH, there is a low incidence of dental decay combined and little or no calculus. A saliva pH below 7.0 usually indicates acidemia (abnormal acidity of the blood). If a chronic condition exists, the mouth is more susceptible to dental decay, halitosis and periodontitis. Chronic acidemia can be a causative factor for a multitude of diseases affecting the whole body. A saliva pH above 7.0 usually indicates alkalinity. Excessive alkalinity can bring about the same anaerobic conditions as acidemia, but it is much rarer condition.

In present study, salivary pH, buffering capacity and plaque pH was compared between a) MH group and control group, b) MIH group and control group. The control group were included after age matching and sex matching with the study group. In this study, salivary characteristics were measured by a simple, quickly manipulated chair-side method. The children were given a modelling wax in the form of pellets to stimulate saliva as done in the study by **Hebbel et al. 2012**[30]**.** A significant difference in salivary pH and plaque pH was found between the study group and control group. The mean plaque pH of MH / MIH individuals (7.03 / 6.91) was found to be higher when compared with the control group (5.60 / 5.25). The mean salivary pH of MH / MIH individuals (6.72 / 5.92) was found to be lower than the control group (8.37 / 8.54). The reason behind the differences in salivary and plaque pH properties between the groups is not clear. One possible explanation given by **Ghanim et al. 2013**[29] is that children with hypomineralization may be those with higher predisposition to salivary changes because of medical conditions that have caused the hypomineralization in the first instance. On the other hand, it is well recognized that salivary composition shows considerable inter-subject variation, particularly in young individuals

whose saliva properties are known to be still immature. Moreover, unlike the compositions of other body fluids, saliva is subject to variation depending on many factors such as the psychological status of the subject and the state of hydration. Therefore, to better address the nature of saliva properties in MH/MIH affected individuals, future prospective investigations into clinical characteristics and etiological factors in combination with exploratory laboratory studies are recommended.

The buffering capacity of saliva is a significant property affecting the dental caries process with the salivary bicarbonate system being the main mechanism for buffering. The bicarbonate concentration is very low in resting saliva that has limited acid-buffering capacity. Therefore, stimulated saliva was collected in the present study for measuring the buffering capacity. The salivary-buffering capacity between the groups showed no significant difference. Only few of the children examined in our study had a low buffering capacity, possibly explaining the lack of correlation observed between hypomineralization severity and buffering capacity of saliva.

In this study we also compared the salivary characteristics between the Molar hypomineralization (MH) group and Molar Incisor Hypomineralization (MIH). There was a significant difference in salivary pH (6.72 in MH group and 5.92 in MIH), plaque pH (7.03 in MH group and 6.91 in MIH group) and buffer capacity (5.79 in MH group and 5.65 in MIH group). The reason behind the difference between these two groups are unknown. More studies should be done on this criteria to relate the difference between MH and MIH.

DENTAL CARIES AND ITS SEVERITY

MIH has long been associated with an increased incidence of dental caries, which has been attributed to the characteristics of hypomineralized enamel, such as higher porosity and lower mechanical resistance. There is also a higher relative concentration of carbon in

the hypomineralized enamel which could be the cause for increased acid solubility of the enamel. The resultant hypersensitivity may compromise oral hygiene procedures thereby increasing the caries risk. MIH is considered as a risk factor of dental caries in populations with a low prevalence of dental caries **(Garcia-Margarit M 2014)[25].** In populations with high caries activity, the hypomineralized lesions could be disguised by carious lesions **(Weerheijm L 2001, Jalevik B 2001)[59, 33].**

In the present study, among 236 children with MH and MIH, 123 children had dental caries which shows a higher prevalence rate of dental caries among MIH and MH group. In the 123 children with dental caries, 140 teeth were affected. The presence of dental caries was about 56.9% boys and 43% girls with no statistical difference between both the genders. Similarly, the caries prevalence in children with MH was 49.3% (n=36) whereas in the children with MIH, the prevalence was 53.3% (n=87). **Jalevik B 2001[33], Preusser SE 2007[49], Cho SY 2008[12], da Costa-Silva CM 2010[14], Jeremias F 2013, Garcia-Margarit M 2014** also stated a higher prevalence of dental caries in MIH affected than in unaffected individuals[25].

In the present study, ICDAS II criteria was used to deliver a more accurate picture of the defect–caries relationship. ICDAS II is the revision of ICDAS criteria (2002) which was formulated in a workshop funded ***by*** **American Dental Association (ADA)**. Only three tooth surfaces (buccal, occlusal, lingual/palatal) were considered in the analysis to achieve a better assessment of this 'lesion-site-specific association. A detailed description of the caries status through categorization of the carious lesions into stages (code 0 to code 6) allow better assessment of its pattern. The presence of hypomineralised defects such as opacities and with post-eruptive breakdown (PEB) was assessed. Impracticality of using compressed air for lesion dehydration, lead to removal of code 1 (first visual change in enamel) in this study. Only code 0, 2, 3, 4, 5, 6 were used. When a tooth had both opacity and PEB, the

most severe form was accounted for. Among those teeth with dental caries (n=140), 65.7% (n=92) of teeth were limited to opacity and 34.2% (n=48) of teeth had PEB. Code 4 (n=38) and Code 3 (n=35) were the most common type of caries severity.

In the present study, teeth with PEB were not as prevalent as teeth with demarcated opacities. The possibility of confusing PEB resulting from hypomineralization or severe caries exists. Nonetheless, a significant relationship was revealed between hypomineralization and dental caries in terms of lesion severity. This finding indicates multiple burdens on treatment need for the child not only through the need for continuous replacement of large restorations but also for possible tooth extraction and orthodontic consequences. Even in tooth surfaces with less extensive defect extension and caries severity, an increased risk of more severe lesions in the immediate future exists. Hence, continuous follow-up is required to limit deterioration and to increase the survival of these teeth.

MIH is a varied and dynamic defect highly influenced by individual characteristics of the oral environment. The findings of this study may increase the level of knowledge amongst dentists towards the appropriate management strategies for MIH-affected teeth. Salivary testing could be a part of routine diagnosis when treating patient with demarcated hypomineralization lesions. This study also found a positive association between MIH and decay and therefore warns dentists on the increased need for treatment of affected children.

LIMITATIONS

- This study compared the caries severity between MIH and MH subjects but didn't compare the severity with the control group.
- More studies with larger sample age can be considered to establish a cause and effect relationship between the risk factors and the occurrence of MIH.

SUMMARY AND CONCLUSION

The present study was conducted in Department of Pedodontics and Preventive dentistry, KSR Institute of Dental Science and Research, Tiruchengode, Tamil Nadu. The aim of this study was to determine the prevalence of Molar Incisor Hypomineralization (MIH) in school children aged 8 to 12 years in Tiruchengode, Tamilnadu. The objectives of the study included a) the prevalence of the children with MIH and MH b) to assess the dental caries lesion severity, and c) to compare the salivary characteristics of children with and without MIH and MH. In this cross sectional study, a total of 4571 children aged 8 to 12 years from various private and government schools were included. Hypo-mineralized permanent molars and incisors were diagnosed clinically based on the European Academy Pediatric Dentistry (EAPD) criteria recommended in 2003 and revised at an Interim Seminar and Workshop concerning MIH organized by the EAPD in 2009. The caries severity was assessed by using International Caries Detection Assessment System II scoring criteria. The saliva and plaque sample of 50 children with MH and 50 children with MIH was collected in a sterile container by spitting method. Similarly the saliva and plaque sample of the 100 control group children (same age and gender) was collected and compared with the study group children. Hannah pH meter was used to calculate salivary pH, plaque pH and salivary buffer capacity. Ericson's test (1959) was employed to determine the salivary buffering capacity. The data were collected and subjected to statistical analysis.

The specific findings of this study is as follows

1. The prevalence of enamel defects (MIH and MH) was 5.25% in the age group of 8 to 12 years in Tiruchengode district.
2. The prevalence of enamel defects was found to be higher in boys (6.42%) when compared with girls (3.97%).
3. The prevalence of MH (1.62%) was lower than that of MIH (3.62%).
4. The prevalence of dental caries in children with MIH/MH was 52.1% (n=123 children). There was no statistical difference between the genders [Boys 70 (56.9%); Girls 53 (43%)].
5. On basis of the caries severity according to ICDAS II criteria, the enamel opacities were 92 teeth (65.7%) were found higher than the post eruptive breakdown 48 teeth (34.2%) in the study population.
6. The mean plaque pH of MIH / MH children was significantly higher than control group ($p = 0.001$).
7. The mean salivary pH of MIH / MH children was significantly lower than the control group ($p = 0.001$).
8. There was no significant difference in the mean salivary buffer capacity between the MIH/ MH group and control group.
9. On comparing MIH children with MH children, there was a significant difference in salivary pH ($p=0.001$), salivary buffer capacity ($p=0.001$), plaque pH ($p=0.001$).

RECOMMENDATIONS

1. The prevalence of Molar Incisor Hypomineralization (MIH) has been increasing globally and it's been essential to rate the prevalence in all localities.
2. Oral health education and awareness program about MIH should be conducted and parents should be educated in order to overcome the complications.
3. An effort should be taken by all the dentist to treat the children with MIH in an effective and efficient manner
4. More research should be done to determine the etiology of MIH thus helping in early diagnosis and prompt treatment planning

CONCLUSION

MIH is a widespread problem all over the world. In the present study MIH and MH was found in 5.25% of children examined (n=4495) with boys 3.33% (n=150) and girls 1.91% (n=86). MIH threatens to become a concerning developmental enamel defect. Considering the low awareness of this condition among the dentists and general population of India, the demanding nature and the costs involved, the urgent need for further investigations into this problem becomes clearly evident. A diligent follow-up and recall program for children who are affected is essential for developing preventive and therapeutic measures, and formulating public awareness and prevention programs.

REFERENCES

1. Alaluusua, Lukinmaa PL, Koskimies M, Pirinen S, Holtta P, Kallio M, Holttinen T, Salmenpera L. Developmental dental defects associated with long breast feeding. Eur J Oral Sci 1996;104(5-6):493-7.
2. Alaluusua S, Lukinmaa PL, Vartiainen T, Partanen M, Torppa J, Tuomisto J. Polychlorinated dibenzo-p-dioxins and dibenzofurans via mother's milk may cause developmental defects in the child's teeth. Environ Toxicol Pharmacol 1996;1(3):193-7.
3. Americano GC, Jacobsen PE, Soviero VM, Haubek D. A systematic review on the association between molar incisor hypomineralization and dental caries. Int J Paediatr Dent 2017;27(1):11-21.
4. Antoine D, Hillson S, Dean MC. The developmental clock of dental enamel: a test for the periodicity of prism cross-striations in modern humans and an evaluation of the most likely sources of error in histological studies of this kind. J Anat 2009;214:45-55.
5. Baliga S, Mugilkar S, Kale R. Salivary pH: A diagnostic biomarker. J Indian Soc Periodontol 2013;17(4):461–65.
6. Baroni C, Marchionni S. MIH supplementation strategies: prospective clinical and laboratory trial. J Dent Res 2011;90(3):371-376.
7. Bhaskar SA, Hegde S. Molar-incisor hypomineralization: prevalence, severity and clinical characteristics in 8- to 13-year-old children of Udaipur, India. J Indian Soc Pedod Prev Dent 2014;32(4):322-9.
8. Brook AH. Multilevel complex interactions between genetic, epigenetic and environmental factors in the aetiology of anomalies of dental development. Arch Oral Biol 2009;54(1):3-17.

9. Chan YL, Ngan AH, King NM. Degraded prism sheaths in the transition region of hypomineralized teeth. J Dent 2010;38(3):237-44.
10. Chawla N, Messer LB Silva M. Clinical studies on molar-incisor-hypomineralisation part 1: distribution and putative associations. Eur Arch Paediatr Dent 2008;9(4):180-90.
11. Chawla N, Messer LB, Silva M. Clinical studies on molar-incisor-hypomineralisation part 2: development of a severity index. Eur Arch Paediatr Dent 2008;9(4):191-9.
12. Cho SY, Ki Y, Chu V. Molar incisor hypomineralization in Hong Kong Chinese children. Int J Paed Dent 2008;18:348-52.
13. Crombie F, Manton D, Kilpatrick N. Aetiology of molar incisor hypomineralization: A critical review. Int J Paediatr Dent 2009;19:73-83.
14. da Costa-Silva CM, Jeremias F, de Souza JF, Cordeiro Rde C, Santos-Pinto L, Zuanon AC. Molar-incisor hypomineralization: Prevalence, severity and clinical consequences in Brazilian children. Int J Paediatr Dent 2010;20:426-34.
15. Dantas-Neta NB, Moura LF, Cruz PF, Moura MS, Paiva SM, Martins CC, Lima MD. Impact of molar-incisor hypomineralization on oral health-related quality of life in schoolchildren. Braz Oral Res 2016;30(1):117.
16. Denny P, Hagen FK, Hardt M, Liao L, Yan W, Arellanno M, Bassilian S, Bedi GS. The proteomes of human parotid and submandibular/sublingual gland salivas collected as the ductal secretions. J Proteome Res 2008;7(5)1994-2006.
17. Dietrich G, Sperling S, Hetzer G. Molar incisor hypomineralisation in a group of children and adolescents living in Dresden (Germany). Eur J Paediatr Dent 2003;4(3):133-7.

18. Dummer PM, Kingdon A, Kingdon R. Prevalence of enamel developmental defects in a group of 11- and 12-year-old children in South Wales. Community Dent Oral Epidemiol 1986;14(2):119-22.
19. Fagrell T. Molar incisor hypomineralization. Morphological and chemical aspects, onset and possible etiological factors. Swed Dent J Suppl 2011;(216)5:11-83.
20. Fagrell Tg, Lingstrom P, Olsson S, Steiniger F, Noren JG. Bacterial invasion of dentinal tubules beneath apparently intact but hypomineralized enamel in molar teeth with molar incisor hypomineralization. Int J Paediatr Dent 2008;18(5):333-40.
21. Fagrell TG, Salmon P, Melin L, Norén JG. Onset of molar incisor hypomineralization (MIH). Swed Dent J 2013;37(2):61-70.
22. Farah RA, Swain MV, Drummond BK, Cook R, Atieh M. Mineral density of hypomineralised enamel. J Dent 2010;38(1):50-58.
23. FDI Commission on Oral Health Research & Epidemiology. A review of the developmental defects of enamel index (DDE Index). Commission on Oral Health, Research & Epidemiology. Report of an FDI Working Group. Int Dent J 1992;42:411-26.
24. Fincham AG, Moradian-Oldak J, Simmer JP. The structural biology of the developing dental enamel matrix. J Struct Biol 1999;126:270-99.
25. Garcia-Margarit M, Catalá-Pizarro M, Montiel-Company JM, Almerich-Silla JM. Epidemiologic study of molar-incisor hypomineralization in 8-year-old Spanish children. Int J Paediatr Dent 2014;24:14-22.
26. Garg N, Jain AK, Saha S, Singh J. Essentiality of early diagnosis of molar incisor hypomineralization in children and review of its clinical presentation, etiology and management. Int J Clin Pediatr Dent 2012;5(3):190-6.

27. Ghanim A, Marin OR, Morgan M, Bailey D, Manton D. An in vivo investigation of salivary properties, enamel hypomineralisation, and carious lesion severity in a group of Iraqi school children. Int J of Paediatr Dent 2013;23:2–12.
28. Ghanim A, Morgan M, Marino R, Bailey D, Manton D. Molar-incisor hypomineralization: Prevalence and defect characteristics in Iraqi Children. Int J Paediatr Dent 2011;21:413-21.
29. Ghanim A, Manton D, Bailey D, Marino R, Morgan M. Risk factors in the occurrence of molar-incisor hypomineralization amongst a group of Iraqi children. Int J Paediatr Dent 2013;23(3):197-206.
30. Hebbal M, Ankola A, Metgud S. Caries risk profile of 12 year old school children in an Indian city using Cariogram. Med Oral Patol Oral Cir Bucal 2012;17(6):1054-6.
31. Heijs SC, Dietz W, Noren JG, Blanksma NG, Jalevik B. Morphology and chemical composition of dentin in permanent first molars with the diagnose MIH. Swed Dent J 2007;31(4):155-64.
32. Henson BS, Wong DT. Collection, storage, and processing of saliva samples for downstream molecular applications. Methods Mol Biol 2010;666:21-30.
33. Jalevik B, Klingberg G, Barregard L, Noren JG. The prevalence of demarcated opacities in permanent first molars in a group of Swedish children. Acta Odontol Scand 2001;59(5):255-60.
34. Jalevik B. Prevalence and diagnosis of Molar-Incisor Hypomineralization (MIH): A systematic review. Eur Arch Paediatr Dent 2010;11:59-64.
35. Kilpatrick N. New developments in understanding development defects of enamel: optimizing clinical outcomes. J Orthod 2009;36(4)277-282.

36. Koch G, Hallonsten AL, Ludvigsson N, Hansson BO, Holst A,Ullbro C. Epidemiologic study of idiopathic enamel hypomineralization in permanent teeth of Swedish children. Community Dent Oral Epidemiol 1987;15(5):279-85.
37. Kojima T, Andersen E, Sanchez JC, Wilkins MR, Hochstrasser DF, Pralong WF, Cimasoni G. Human gingival crevicular fluid contains MRP8 (S100A8) and MRP14 (S100A9), two calcium-binding proteins of the S100 family. J Dent Res 2000;7(2):740-747.
38. Kosem R, Senk Erpic A, Kosir N, Kastelec D. Prevalence of Enamel Defects With Emphasis on Molar Incisor Hypomineralization in Slovenian Children and Adolescents [abstract]. Barcelona, Spain: 7th Congress of the European Academy of Paediatric Dentistry; 2004.
39. Kuhnisch J, Mach D, Thiering E, Brockow I, Hoffmann U, Neumann C. Respiratory diseases are associated with molar-incisor hypomineralizations. Swiss Dent J 2014;124(3):286-93.
40. Leppaniemi A, Lukinmaa PL, Alaluusua S. Nonfluoride hypomineralizations in the permanent first molars and their impact on the treatment need. Caries Res 2001;35(1):36-40.
41. Loli D, Costacurta M, Maturo P, Docimo R. Correlation between aerosol therapy in early childhood and Molar Incisor Hypomineralisation. Eur J Paediatr Dent 2015;16(1):73-7.
42. Lussi A. Dental erosion, clinical diagnosis and case history taking. Euro J Oral Sci 1996;104:191–198.
43. Lygidakis NA, Dimou G, Marinou D, Gouva G. Aetiology of Molar-incisor Hypomineralization. A retrospective study of 151 children with the defect [abstract].

Barcelona, Spain: 7th Congress of the European Academy of Paediatric Dentistry; 2004.

44. Lygidakis NA, Wong F, Jälevik B, Vierrou AM, Alaluusua S, Espelid I. Best clinical practice guidance for clinicians dealing with children presenting with Molar-Incisor Hypomineralization (MIH): An EAPD Policy Document. Eur Arch Paediatr Dent 2010;11:75-81.
45. Mahoney P. Two dimensional patterns of human enamel thickness on deciduous (dm1, dm2) and permanent first (M1) mandibular molars. Arch Oral Biol 2011;55:115-26.
46. Mangum, JE, Crombie FA, Kilpatrick N, Manton DJ, Hubbard MJ. Surface integrity governs the proteome of hypomineralized enamel. J Dent Res 2010;89(10):1160-1165.
47. Ozqul BM, Saat S, Sonmez H, Oz FT. Clinical evaluation of desensitizing treatment for incisor teeth affected by molar-incisor hypomineralization. J Clin Pediatr Dent 2013;38(2):101-5.
48. Parikh DR, Ganesh M, Bhaskar V. Prevalence and characteristics of Molar-Incisor Hypomineralization (MIH) in the child population residing in Gandhinagar, Gujarat, India. Eur Arch Paediatr Dent 2012;13:21-6.
49. Preusser SE, Ferring V, Wleklinski C, Wetzel WE. Prevalence and severity of molar-incisor hypomineralization in a region of Germany - a brief communication. J Public Health Dent 2007;67:148-50.
50. Reid DJ, Dean MC. Variation in modern human enamel formation times. J Hum Evol 2006;50:329-46.
51. Rodd HD, Boissonade FM, Day PF. Pulpal status of hypomineralized permanent molars. Pediatr Dent. 2007;29(6):514-20.

52. Seow WK. A study of the development of the permanent dentition in very low birthweight children. Pediatr Dent 1996;18(5):379-384.
53. Simmer JP, Papagerakis P, Smith CE, Fisher DC, Rountrey AN, Zheng L, Hu JC. Regulation of dental enamel shape and hardness. J Dent Res 2010;89:1024-38.
54. Soviero V, Haubek D, Trindade C, Matta TD, Poulsen S. Prevalence and distribution of demarcated opacities and their sequelae in permanent 1st molars and incisors in 7 to 13-yearold Brazilian children. Acta Odontol Scand 2009;67:170-175.
55. Stefano M, Gugielmo C, Laura S, Alessandro V, Cagetti MG. An Innovative Approach to Treat Incisors Hypomineralization (MIH): A Combined Use of Casein Phosphopeptide-Amorphous Calcium Phosphate and Hydrogen Peroxide—a Case Report. Case Reports in Dentistry 2012 Article ID 379593.
56. Suckling GW, Nelson DG, Patel MJ. Macroscopic and scanning electron microscopic appearance and hardness values of developmental defects in human permanent tooth enamel. Adv Dent Res 1989;3:219-33.
57. Sui W, Boyd C, Wright JT. Altered pH regulation during enamel development in the cystic fibrosis mouse incisor. J Dent Res 2003;82(5):388-92.
58. Van Amerongen WE, Kreulen CM. Cheese molars: a pilot study of the etiology of hypocalcifications in first permanent molars. ASDC J Dent Child 1995;62(4):266-9.
59. Weerheijm KL, Jaleirk B, Alaluusua S. Molar-incisor hypomineralization. Caries Res 2001;35(5):390-1.
60. Weerheijm KL, Duggal M, Mejàre I, Papagiannoulis L, Koch G, Martens LC. Judgement criteria for molar-incisor hypomineralization (MIH) in epidemiologic studies: A summary of the European meeting on MIH held in Athens. Eur J Paediatr Dent 2003;4:110-3.

61. Weerheijm KL, Groen HJ, Beentjes VE, Poorterman JH. Prevalence of cheese molars in eleven-year-old Dutch children. ASDC J Dent Child 2001;68(4):259-62.
62. Weerheijm KL. Molar incisor hypomineralization (MIH): clinical presentation, aetiology and management. Dent Update 2004;31(1):9-12.
63. Weerheijm KL. Molar-incisor-hypomineralisation (MIH). Eur J Paediatr Dent 2003;4:115-120.
64. William V, Burrow MF, Palamara JE, Messer LB. Microshear bond strength of resin composite to teeth affected by molar hypomineralization using 2 adhesive systems. Pediatr Dent 2006;28(3):233-41
65. William V, Messer BL, Burow MF. Molar incisor hypomineralization: review and recommendations for clinical management. Pediatr Dent 2006;28(3):224-32
66. Willmott N. Molar incisor hypomineralization. Dent Nurs 2011;7:132-7.
67. Wuollet E, Laisi S, Salmela E, Ess A, Alaluusua S. Molar-incisor hypomineralization and the association with childhood illnesses and antibiotics in a group of Finnish children. Acta Odontol Scand 2016;74(5):416-22
68. Zawaideh FI, Al-Jundi SH, Al-Jaljoli MH. Molar-Incisor Hypomineralization: Prevalence in Jordanian children and clinical characteristics. Eur Arch Paediatr Dent 2011;12:31-6.

APPENDIX

APPENDIX - I

INSTITUTIONAL ETHICAL COMMITTEE

KSR INSTITUTE OF DENTAL SCIENCE & RESEARCH

KSR Kalvi Nagar, Tiruchengode-637 215, Tamilnadu.
Phone : 04288-274981, Fax : 04288-274761,
email : ksrdentalcollege@yahoo.com

Chairman
Dr. P. PONMURUGAN, Ph.D.,
Prof. & Head Dept. of Biotechnology
KSR College of Technology,
KSR Kalvi Nagar, Tiruchengode.

Member Secretary
Dr. G.S. KUMAR, MDS.,
Principal,
KSR Institute of Dental Science & Research
KSR Kalvi Nagar, Tiruchengode.

Members

Dr.G.Ayppadasan,Ph.D.,
Biotechnologist

Mr.A.Thirumoorthi,M.A.B.L.,
Human Activist

Dr.R.Renuka,M.D.S.,(Perio),M.Sc.,
Family Counsellor

Dr.K.Sivakumar,MDS.,(Cons.Dent.)

Dr.Suman, M.D.S.,(OMDR)

Dr.Sharath Ashokan,MDS.,(Pedo)

Dr.G.Rajeswari,Ph.D.,(Biochemistry)

Dr.K.Karthick,MDS.,(Cons.Dent.)

Mr.V.Mohan,M.Sc.,M.Phil.,(Physicist)

Mr.A.P.S.Raja,B.A.,

Ref.: 081 /KSRIDSR/EC/2014 **Date : 26.11.2014**

To

Dr.J.Allwyn Samuel,
Postgraduate Student,
Dept. of Paedodontics,
KSR Institute of Dental Science & Research,

*****

Your dissertational study titled "PREVALENCE, DISTRIBUTION, CARIES LESION SEVERITY AND SALIVARY CHARACTERISTICS OF MOLAR INCISOR HYPO MINERALIZATION" presented before the ethical committee on 24th Nov.2014 has been discussed by the committee members and has been approved.

You are requested to adhere to the ICMR guidelines on Biomedical Research and follow good clinical practice. You are requested to inform the progress of work from time to time and submit a final report on the completion of study.

Signature of Member Secretary
(Dr.G.S.Kumar)

APPENDIX – II

K.S.R Institute of Dental Science and Research
Tiruchengode -637215

CONSENT FORM

I ....., as legally responsible parent/guardian give my consent for the participation of my child ....., standard..... In the study title "Prevalence, Distribution, Caries severity and salivary characteristics of Molar Incisor Hypomineralization - A Cross Sectional Study". Dr. Allwyn Samuel. J discussed with me to my satisfaction, the procedures, possible discomforts, as well as possible benefits of the study. I have read this consent and have clearly understood the procedures to be performed on my child.

Legally responsible parent/guardian: ..... Date: .....

Address: .....

Contact number:

I certify that I explained the above information to the parent/guardian, before requesting his or her signature.

Signature of the dentist: ..... Date: .....

APPENDIX – III

K.S.R பல் மருத்துவக் கல்லூரி மற்றும் ஆராய்ச்சி மையம்

திருச்செங்கோடு -637215

தகவலறிந்த ஒப்புதல் படிவம்

பெயர் _____ வயது _____ ஆண்/பெண் ஆகிய நான் என் மகன்/மகள் _____ ,வயது __ அவர்களை மருத்துவர் அவர்களின் ஆராய்ச்சிக்கு உட்படுத்த அனுமதி கோரப்பட்டுள்ளது. இவ்வாராய்ச்சிப்பற்றி விளக்கங்களும், முறைகளும் நான் படித்துப் பார்த்தேன்/படித்துக் காட்டப்பட்டது. எனது சந்தேகங்களுக்கு தெளிவாக விளக்கம் அளிக்கப்பட்டது.எனவே நான் எனது மகனை/மகளை இவ்வாராய்ச்சியில் பங்கெடுக்க அனுமதி அளிக்கிறேன்.

இடம்:

தேதி : (பெற்றோர் கையொப்பம்)

APPENDIX – IV

LIST OF SCHOOL NAMES

- A – Avvai KSR Matriculation School
- B – KSR Matriculation Higher secondary School
- C – KSR Akshara Academy
- D – Vidyut Pulic School
- E – V School
- F – ASS Matriculation. Higher secondary School
- G – SPB Matriculation School
- H – Kootapalli Government School
- I – Anandha Malar Trust
- J – Award Trust
- K – Thokkavadi Government School
- L – Anangur Government School
- M – Devanalanguruchi Government School
- N – SPB Government School

APPENDIX – V

ABBREVATIONS

- MIH – Molar Incisor Hypomineralization
- MH – Molar Hypomineralization
- pH - Potential of Hydrogen
- FDI – World Dental Federation
- EAPD – European Academy Pediatric Dentistry
- ADA – American Dental Association
- DDE – Developmental Defects of the Dental Enamel
- ICDAS – International Caries Detection and Assessment System
- FPMs – First Permanent Molars
- PEB – Post Eruptive Breakdown
- VLBW - Very Low Birth Weight
- HCl – Hydrochloric acid
- CCP – Casein Phosphopeptide
- ACP – Amorphous Calcium Phosphate
- CFTR – Cystic Fibrosis Transmembrane Conductance Regulator
- SEM – Scanning Electron Microscope
- TEM – Transmission Electron Microscope
- XRMA – X-Ray Micro Analysis

APPENDIX – VI

DIAGNOSTIC CRITERIA AND CLINICAL APPEARANCE OF MIH EAPD (2003, 2009)

FPMs and incisors	One to all four permanent first molars shows hypomineralization of the enamel. Simultaneously, the permanent incisors can be affected. To diagnose MIH, at least one FPM has to be affected. The defects can also be seen in second primary molars, incisors and the tips of canines. More the molars and incisors affected, the more severe is the defect
Demarcated opacities	The affected teeth show clearly demarcated opacities at the occlusal and buccal parts of the crown. The defects vary in color and size. The color can be white, creamy or yellow to brownish. The defect can be negligible or comprise the major part of the crown. It is recommended that defects less than 1 mm not be reported
Enamel disintegration (PEB)	The degree of porosity of the hypomineralized opaque areas varies. Severely affected enamel subjected to masticatory forces soon breaks down, leading to unprotected dentin and rapid caries development
Atypical restorations	FPMs and incisors with restorations revealing similar extensions as MIH are recommended to be judged as affected
Tooth sensitivity	The affected teeth may be reported frequently as sensitive, ranging from a mild response to external stimuli to spontaneous hypersensitivity; these teeth are usually difficult to anaesthetize
Extracted teeth	Extracted teeth can be defined as having MIH only in cases where there are notes in the records or demarcated opacities on the other FPM. Otherwise it is not possible to diagnose MIH
Recording the severity of defects	Severity should be recorded as mild or severe in order to help the clinician. In mild cases, there are demarcated enamel opacities without enamel breakdown, occasional sensitivity to external stimuli e.g. air/water but not brushing and only mild aesthetic concerns on discoloration of the incisors

APPENDIX – VII

ICDAS II INDEX

Code	Criteria
0	Sound teeth
1	First visual change in enamel (seen only after prolonged air drying or restricted to within the confines of a pit or fissure)
2	Distinct visual change in enamel
3	Localized enamel breakdown (without clinical visual signs of dentinal involvement)
4	Underlying dark shadow from dentin
5	Distinct cavity with visible dentin
6	Extensive distinct cavity with visible dentin