

ROLE OF GENETICS IN MALOCCLUSION

Dr.Shrada.B.Kumar,
CRI, Saveetha Dental College.
Dr.Dhanraj, Saveetha Dental College.
Department of Prosthodontics.

ABSTRACT:

Class III malocclusion is thought to be a result of interaction of genes and environment, studies on family pedigree chart have pointed on its probability of its monogenic dominant inheritance. Vascular endothelial growth factor, insulin like growth factor 1 and HOX -3 are few of those genes .The influence of genetic factors can be understood only when studied in quantitative terms. This review is about the role of genetics and how it influences facial morphology.

INTRODUCTION:

Class III malocclusion has been the subject of study of interest because of the challenge in treating class III malocclusion. Class III malocclusion is defined in cases in which the mandibular 1st molar is placed mesially to the maxillary 1st molar.[1] It can be skeletal or dento-alveolar. Its etiology is generally believed to be genetic and familial occurrence.

A wide range of environmental factors contribute to class III malocclusion .They are enlarged tonsil, difficulty in nasal breathing[2]congenital anatomic defects, disease of pituitary gland[2]hormonal disturbances and premature loss of primary molars[3] and loss of deciduous incisors [4]

A wide range of hormones such as IHH (Indian hedgehog homolog) [5], PTHLH (Parathyroid like hormone), VEGF (vascular endothelial growth factor), etc., are seen to play a major role in development of class III occlusion. Recent studies have shown that erythrocyte membrane protein band (EPB4) also has influence in bringing about mandibular prognathism.

MODE OF INHERITANCE IN CLASS III MALOCCLUSION:

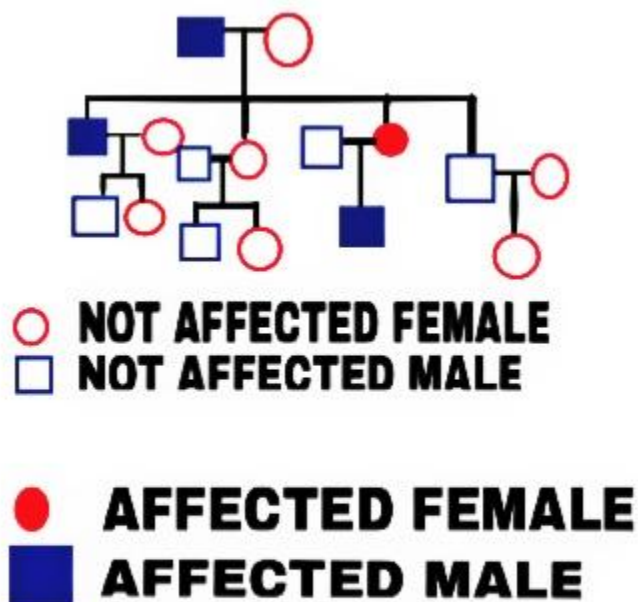
The inheritance of phenotypic features in mandibular prognathism was first reported by Strohmayer [6] and then by Wolff et al[7]their analysis of pedigree chart of Hapsburg family. Suzuki [8] reported a frequency of 31 percent was affected if the father was affected, 18 percent if the mother was affected and 40 percent if both parents were affected on studying 243 Japanese families. Nikolova [9] Studied 251 Bulgarian families and showed great parental influence for head height and nose height.

Heritability of craniofacial morphology Has also been investigated in siblings, from parents to twins or from parents to off springs in longitudinal studies. Horowitz et al [10] demonstrated a significant hereditary component for anterior cranial base, mandibular body length, lower facial height and total facial height. Hunter et al [11] reported a strong genetic correlation between fathers and children especially in mandibular dimension. Fernex et al [12] found that the sizes of skeletal facial structures were transmitted with more frequency from mothers to sons than to mothers to daughters.

ROLE OF GENES IN EXPRESSION OF CLASS III MALOCCLUSION :

Although mandibular prognathism has been said to be a polygenic [13] or multi factorial trait, in majority of the cases, there are families in which the trait appears to have autosomal dominant inheritance.

PEDIGREE CHART -AUTOSOMAL DOMINANT INHERITANCE -CLASS III MALOCCLUSION



The Animal studies have shown that IGF-1 significantly increased when mandible was repositioned with a propulsive appliance. Rabie [14, 15] et al indicated that forward positioning of the mandible triggered the expression of *Ihh* and *Pthlh*, which promote mesenchymal cell differentiation and proliferation, respectively, and that these proteins acted as mediators of mechano- transduction to promote increased growth of the cartilage. Also an

increase in transcription factors like sex-determining region Y and Runx2 was noted during mechanical loading of mandible. These factors induce differentiation of chondrocytes .

A number of genes have documented the influence of various genes that are involved in the regulation of mandibular morphogenesis. Growth factors and cytokines are local mediators and can be secreted in response to mechanical strain. These mediators regulate cell proliferation and the expression of differentiation products by activating signal transduction pathways in the target cells [19]. In an experimental model of enhanced condylar growth, Rabie et al. [14,15] indicated that forward positioning of the mandible triggered the expression of Ihh and Pthlh, which promote mesenchymal cell differentiation and proliferation, respectively, and that these proteins acted as mediators of mechanotransduction to promote increased growth of the cartilage. In another rat model, the expression of IGF-1 increased significantly when the mandible was repositioned by means of a propulsive appliance [15]. In addition, growth factors such as Vegf and transcription factors such as the sex-determining region Y [SRY]-box 9 [Sox9] and runt-related transcription factor 2 [Runx2] play important roles in the differentiation of chondrocytes in the growth plate under conditions of mechanical loading or exposure to other stimuli.

The genes that have been implicated in condylar growth by studies in the mouse might serve as potential candidates to increase our understanding of Class III malocclusion in humans. The discovery of candidate genes provides the possibility to identify genes that confer susceptibility to this phenotype. In the search for susceptibility genes that are involved in Class III malocclusion, polymorphisms in the aforementioned genes and the genes for the molecules that they regulate will be prime targets.

DISCUSSION:

Various treatment modalities prescribed by the orthodontist are expected to lead to improved orofacial function. However some patient show relapses. This is due to lack of cooperation of the patient. Other factors like Growth, inherent lack of muscle adaptability has to be considered. A complicating factor for diagnosis and treatment of class III malocclusion is the etiologic diversity. The etiology is usually multifactorial.

Even though various studies have contributed to our understanding of the inheritance of the class III phenotype, there are still significant gaps in the knowledge of the specific genetic contribution.

Typically class III malocclusion is inherited in a polygenic manner. Sometimes it is monogenic too.

Progression in molecular biology has made it possible to recognize various genes that are involved in mandibular growth.

Recent genetic research using linkage analysis and association studies has identified the genes that confer susceptibility to class III malocclusion.

CONCLUSION:

Class III malocclusion is the result of multiple factors that interact during the morphologic period of the mandible and it might be possible to regulate some of these factors during infancy, hence it is been suggested that better understanding of genetic variables that contribute to class III phenotype is necessary to develop new preventive strategies .

REFERENCE:

- 1]Profit WR fields HW contemporary orthodontics ,4th edition , St.Louis ,the CV Mosby co;2000
- 2]Angle EH .Treatment of malocclusion of teeth ,7th edition .Philadelphia SS ; white manufacturing company ;1907.p 52-4,58.
- 3]Gold JK A new approach to the treatment of mandibular prognathism Am J Orthod 1949;35:893-895
- 4]Pascoe JJ Haywardr JR, Costich ER ,mandibular prognathism Its etiology and classification. J Oral Surgery 1960,18;21-4
- 5]Trainor PA,Krumalauf R. Patterning the cranial neural crest ; hindbrain segmentation and HOX gene plasticity Nat Rev Neurosci 2000 ;Nov; 1 [2];116-24
- 6]Strohmayer W.Die Vereburg des Hapsburger Familientypus,Nova Acta Leopoldina 1937;5 219- 296.
- 7]Wolff G Wienker TF, Sander H on the genetics of mandibular prognathism ; analysis of large European noble families . J Med Genet ,1993;30:112-116
- 8]Suzuki S studies on the so- called reverse occlusion J.Nihon Univ Sch Dent 1936;3;51-58.
- 9]Nikolova M.Similarities in anthropometrical traits of children and their parents in a Bulgarian population . Ann Hum Genet 1996;60,517-525.
- 10]Horowitz S.Osborne R.De George F. A cephalometric study of craniofacial variation in adult twins .Angle Orthod 1960;30:1-5
- 11]Hunter W. Ballouch D. Lamphier D.The heritability of attained growth in the human face. Am J of Orthod.1970;58:128-134
- 12]Fernex E hauenstein P.Roche M. Heredity and craniofacial morphology.Transactions of the European orthodontic society.1967:239-257
- 13]Litton SF, Ackermann LV, Issacson RJ ,et al.A genetic study of class 3 malocclusion. Am J Orthos 1970;58[6]:565-577.
- 14] Tang GH Rabie AB, Hagg U. Indian hedgehog: a mechanotransduction mediator in condylar cartilage. J Dent Res 2004;83:434–8
- 15] Rabie AB, Tang GH, Xiong H, Hagg U. PTHrP regulates chondrocyte maturation in condylar cartilage. J Dent Res 2003;82: 627–31.