

Genetics and Orthodontics .

Nezar Wattad¹, Ali Wattad², Rand Ghoul³, Iosif Amparat⁴, Muhamad Abu Hussein⁵

¹Department of Orthodontics, Faculty of Dentistry, Arab America University, Jenin 919000, Palestine

²Department of Cranio-Maxillo-Facial Surgery, University Hospital Augsburg, Germany

³Department of Orthodontics, Faculty of Dentistry, Arab America University, Rammalla, Palestine

⁴General private practice

⁵ Department of Orthodontic and Pediatric Dentistry, University of Athens, Greece

Cite this paper as: Nezar Wattad, Ali Wattad, Rand Ghoul, Iosif Amparat, Muhamad Abu Hussein (2026) Genetics and Orthodontics ...Journal of Neonatal Surgery, 15, (1s) 250-256

ABSTRACT

Genetics explains a great deal of variation seen in the population when facial deformities and malocclusions are considered. However, genetics is not synonymous of a deterministic concept in which a single gene, segregating in families, determines malocclusion. These monogenic models explain very few cases of malocclusion and the other human diseases, as well as traits such as height, weight, amount of sugar in the circulating blood, blood pressure, intelligence, behavior, and sexual orientation. All these traits, as well as the majority of human diseases and congenital defects, have complex or multifactorial modes of inheritance, which can be influenced by the environment, and determine the presence of the majority of traits and diseases...

Keywords: History, dental anomalies, genetics, orthodontics.

INTRODUCTION

Genetics plays a great role in the growth and development of human body and any disturbance in the growth of craniofacial complex leads to malocclusion.[1]

Types of studies: Main research studies which have provided the information on role of heredity on malocclusion are of two types: (1) Family studies and (2) twin studies. Twin studies have been done on the monozygotic twins and the dizygotic twins. Studies have been done to compare different races and ethnic groups also to determine the differences in the dentofacial patterns of different groups. [1-3]

Twin studies: The classical twin approach for separating the effects of nature and nurture involves comparing identical (monozygous: MZ) and nonidentical (dizygous) twin pairs

different. In MZ, twins reflect environmental factors whereas differences between dizygous (DZ) pairs are due to both genetic and environmental factors. [1-5]

Etiology of the malocclusion: Genetic and environmental factors play important role in etiology of malocclusion. It has been shown by many family and twin studies. However, the characteristics are not controlled by a single gene, but they show a polygenic inheritance pattern. [1-6]

Polygenic inheritance: The "normal" variation in dentition is a result of multiple rather than single genes. Normal variation means that the genetic defects or syndromes associated with the dentition are excluded, which may be caused by other associated factors like mutations also. Thus, the size or shape of the teeth, etc. is determined by many genes interacting with each other and the environment. Tooth size, tooth eruption, congenitally missing teeth, tooth morphology, etc. are affected by polygenic inheritance, as has been shown by various family and twin studies. [1-7]

Genetic Studies on Growth

Growth and development of craniofacial complex is the result of interplay of genetic and environmental influences. Various twin studies confirm that the size and shape of body parts, the rate/progress of growth (fast maturing/slow maturing tendencies), fat deposition, growth patterns and the onset of growth events viz. menarche, dental calcification, dental eruption, ossification of bones and start of adolescent growth spurt, are controlled by genetic factors. The fast maturing and slow maturing tendencies run in the families and also in some racial and

..

ethnic groups. Growth differences in males and females are also controlled by genetic factors, as Y chromosome in males is responsible to delay the growth and maturation in males to allow them to grow for a longer time. Individuals having XXY chromosomes (Klinefelter's syndrome) follow a growth pattern similar to males despite presence of two X chromosomes, whereas individuals having one X chromosome (Turner's syndrome) follow that similar to females in absence of Y chromosomes. Individuals having XYY chromosomes follow a growth pattern similar to males but result in increased height due to the presence of two Y chromosomes to allow them to grow for a longer period of time. [6-12].

Genetic Studies on Malocclusion in Different Populations

The prevalence and phenotypic expression of malocclusion have long been associated with genetic, ethnic, and population-related factors. Anthropological and orthodontic investigations suggest that malocclusion was less prevalent in primitive and genetically isolated human populations than in modern heterogeneous populations. One possible explanation is that relative genetic homogeneity and restricted interpopulation mixing may have contributed to greater craniofacial uniformity and more stable occlusal relationships. In contrast, modern populations are characterized by increased genetic admixture and environmental influences, both of which may contribute to greater variability in craniofacial morphology and occlusal relationships. [1-12]

Numerous craniofacial and dentofacial characteristics have been shown to exhibit familial and racial aggregation, indicating a substantial hereditary component in the development of skeletal and dental malocclusions. Specific craniofacial patterns are known to occur more frequently in certain ethnic groups and populations. For example, Polynesian populations have historically demonstrated a characteristic mandibular morphology with a broad and smoothly curved gonial region, commonly referred to as the "Rocker Jaw." This mandibular configuration has been considered a population-specific morphological trait with a strong hereditary background.

Similarly, characteristic vertical facial patterns differ among populations. A reduced lower facial height associated with a deep bite tendency has been reported more frequently in White populations, whereas increased vertical facial proportions and anterior open bite tendencies are more commonly observed in Black populations. These differences in facial morphology are believed to be partially related to inherited craniofacial growth patterns, particularly the orientation of the cranial base and the vertical growth direction of the maxillomandibular complex. Increased inclination of the cranial base, especially an increased saddle angle, has been associated with greater anterior facial height and vertical skeletal growth tendencies. [1-12]

Genetic influences on malocclusion are not limited to isolated skeletal traits but involve complex interactions between multiple genes regulating craniofacial growth, mandibular rotation, dentoalveolar development, muscular function, and soft tissue balance. Consequently, malocclusion should be regarded as a multifactorial condition resulting from the interaction of hereditary predisposition and environmental influences.

In modern civilization, increased population mobility and interethnic admixture have contributed to greater genetic diversity. Several investigators have suggested that outbreeding may increase the variability of craniofacial characteristics and occlusal relationships, thereby potentially increasing the prevalence and complexity of malocclusion. A classical investigation conducted by Chung et al. in the Hawaiian population demonstrated an increased prevalence of malocclusion associated with interethnic admixture among European, Chinese, Japanese, and other ethnic groups. These findings support the hypothesis that genetic heterogeneity may influence the expression of dentofacial discrepancies and craniofacial disharmony. [7-9]

Current genetic and molecular studies further support the concept that malocclusion is influenced by polygenic inheritance involving numerous genes related to craniofacial growth and development. Modern advances in genomics, epigenetics, artificial intelligence, and craniofacial phenotyping are expected to improve the understanding of population-specific growth patterns and may contribute to more individualized orthodontic diagnosis and treatment planning in the future.

Genetic Studies on Malocclusion in Family Members

Genetic factors play a fundamental role in the etiology, growth pattern, and phenotypic expression of malocclusion. It has been estimated that approximately 40% of dental and craniofacial variations associated with malocclusion may be attributed to hereditary influences. Both skeletal and dentoalveolar components are affected by genetic regulation, although the degree of genetic influence differs among craniofacial structures and functional systems. Local occlusal variables, dental arch form, tooth size, eruption sequence, and craniofacial growth direction have all been shown to exhibit varying levels of heritability. [1-8]

Lundström (1984) emphasized that genetic influence is considerably stronger on skeletal morphology than on purely dental characteristics. Skeletal growth patterns, mandibular rotation, facial proportions, and cranial base morphology appear to be more genetically determined, whereas dental alignment and occlusal relationships are influenced by a greater interaction between genetic predisposition and environmental factors. Furthermore, van der Linden described that not only skeletal structures, but also soft tissue morphology and functional behavior are influenced by hereditary factors. Since soft tissues

play a major role in shaping dentoalveolar structures, muscular balance, lip posture, tongue position, and functional activity may indirectly contribute to the development and maintenance of malocclusion. [1–8]

One of the most important methods for evaluating hereditary influences in craniofacial growth and malocclusion is the study of twins. Monozygotic twins (MZ twins), who share nearly identical genetic material, provide the most reliable model for investigating genetic contributions. Comparisons between monozygotic and dizygotic twins (DZ twins) allow differentiation between hereditary and environmental influences. Studies have demonstrated that differences observed between monozygotic twins are primarily attributable to environmental factors, whereas differences between dizygotic twins result from both genetic and environmental variability. Numerous twin studies have shown a remarkable similarity in craniofacial morphology, growth direction, biological maturation, and skeletal development among monozygotic twins, supporting the dominant role of genetics in craniofacial growth and dentofacial development. [11–20]

Heritability of Skeletal Variables

Among the skeletal patterns associated with malocclusion, mandibular prognathism represents one of the most extensively studied hereditary craniofacial deformities. A classical example is the prognathic mandible observed throughout generations of the European royal Habsburg family, commonly referred to as the “Habsburg Jaw.” Pedigree analyses have suggested that mandibular prognathism may be transmitted as an autosomal dominant (AD) trait in certain families.

Skeletal Class III malocclusion, particularly mandibular prognathism, has consistently demonstrated a strong hereditary component. In addition, vertical skeletal discrepancies, especially long-face morphology associated with anterior open bite, also appear to exhibit substantial genetic determination. Cranial base morphology plays an important role in the development of skeletal Class III relationships. Features such as:

- a more acute cranial base angle,
- a shortened posterior cranial base,
- and a more anteriorly positioned glenoid fossa

may contribute to mandibular prognathism and are themselves influenced by multifactorial genetic inheritance. Ellis and McNamara (1984), as well as Singh et al. (1997), emphasized the multifactorial genetic regulation of these craniofacial structures. [16–22]

Twin studies involving monozygotic and dizygotic twins have further demonstrated that specific mandibular regions, including:

- the lateral surface of the ramus,
- the lingual symphysis,
- and the frontal curvature of the mandible,

are predominantly under genetic control. [16–22]

Importantly, skeletal characteristics in all three spatial dimensions:

- anteroposterior,
- transverse,
- and vertical

have been shown to exhibit considerable heritability. Hughes and Moore (1941) concluded that the maxilla and mandible are under partially independent genetic control. Furthermore, different anatomical regions of the same bone, such as the ramus, body, and symphysis of the mandible, may be influenced by distinct genetic and environmental mechanisms.

Class II Division 1 malocclusion has also been associated with hereditary skeletal patterns. Harris demonstrated that patients with Class II Division 1 malocclusion frequently exhibit:

- mandibular retrusion,
- reduced mandibular body length,
- and decreased total mandibular length

compared with skeletal Class I subjects, supporting the concept of polygenic inheritance in Class II malocclusion. [16–27]

Syndromes and Craniofacial Asymmetry

Genetic influences are particularly evident in congenital craniofacial anomalies and syndromic malformations. One of the most common congenital craniofacial disorders is cleft lip and palate, which involves complex interactions between genetic predisposition and environmental influences.

Craniofacial asymmetry may result from hereditary, developmental, functional, or pathological factors. Certain syndromes, including:

- neurofibromatosis,

hemifacial microsomia,
and other craniofacial developmental disorders,
demonstrate strong genetic associations and may affect either the right or left side of the face.

Interestingly, unilateral cleft lip has been reported more frequently on the left side than on the right side, with an approximate ratio of 2:1. In the absence of obvious environmental explanations, this asymmetrical distribution has been hypothesized to reflect genetically determined developmental differences between the two halves of the craniofacial complex. [15–28]

Current evidence strongly supports the concept that malocclusion is a multifactorial condition in which hereditary influences play a major role in craniofacial growth, skeletal morphology, dentoalveolar development, and functional adaptation. Genetic regulation affects not only skeletal structures but also muscular function, soft tissue behavior, cranial base morphology, and biological maturation patterns. Twin studies, family analyses, pedigree investigations, and modern molecular genetic research continue to demonstrate that many forms of malocclusion, particularly skeletal discrepancies, exhibit substantial heritability.

At the same time, environmental and functional factors interact continuously with genetic predisposition, resulting in the wide phenotypic variability observed in orthodontic patients. Modern advances in molecular genetics, genomics, epigenetics, artificial intelligence, and craniofacial phenotyping are expected to further improve the understanding of hereditary mechanisms underlying malocclusion and may contribute to more individualized orthodontic diagnosis and treatment strategies in the future.

Heritability of Dental Variables

Lundstrom 1948 in his study pointed out that dental arch width and length, overbite and crowding and spacing of teeth are influenced by heredity. Dental arch asymmetry also may be genetic or non-genetic in origin. Cranial type and facial type are genetically determined and are correlated with arch width, i.e. brachycephalic cranial type is associated with euryprosopic facial type and wide dental arch and dolichocephalic cranial type is associated with leptoprosopic facial type and narrow dental arch, so the arch width is found to be primarily under genetic control. [11,12]

Arch interrelationship assessed by maxillary and mandibular permanent first molar relationship likely to be reflection of anteroposterior skeletal relationship is also found to be under genetic influence. [11-14]

Number and the size of the teeth: Osborne et al 1958 showed that tooth crown size is strongly determined by heredity. Variation in number of teeth ie hypodontia, supernumerary teeth, etc. and the size of teeth also shows a familial tendency, and polygenic inheritance. Abnormal shapes of teeth, e.g. maxillary lateral incisors and Carabelli cusps trait are strongly under genetic influence showing familial trends, showing a polygenic inheritance. Variation in eruption pattern of teeth eg impaction, ectopic maxillary canine; transposition of teeth and submerged primary molars all show familial tendency and strong genetic component having incomplete penetrance and variable expressivity. [11-16]

Butler's Field Theory

There are certain specific teeth which show more variation than others. According to Butler's field theory 1939, the mammalian dentition can be divided into several developmental fields. There is a "key" tooth within each field, one that is more stable developmentally, and on either side of this key tooth, the remaining teeth within the field become progressively less stable. The three fields are those for molars/premolars, incisors and canines. [17-20]

The molars/premolars field consists of the first molar as the key tooth, the second the third molars on the distal end of the field, and the first and second premolars on the mesial end. The theory predicts that the third molar and first premolar would be most variable in size and shape. It can be questioned that the third molar is agreed as the variable tooth but not the first premolar. Actually the earliest mammals had four premolars and some of the higher primates, including man, have lost the first two premolars. So the premolars that we refer to first and second should

really be labeled third and fourth. The point is that as Butler's theory predicted, the premolars farthest from the first molar were the first to be lost in an evolutionary sense and therefore can be considered the least stable.

However, a modern modification of this concept considers the dental arch in four segments: (1) incisors, (2) canines, (3) premolars and (4) molars. It depicts that the distal tooth of each segment is most often missing congenitally. [17-24]

In maxillary incisor region, the lateral incisor is found to be missing more often than the central incisor. However, an exception is noted in lower incisors region, where central incisor is found missing more often than the lateral incisor. Canines being the only teeth in their respective field, they are found missing least percentage of time. In premolar region, it is the second premolar which is found more often congenitally missing, and in molar region, the third molar is missing congenitally. [15-28]

Numerous differences exist in dentofacial characteristics of the individuals, even among family members. A multifactorial etiology, with genetics and environmental factors are main contributors to such variation. Skeletal dimension for parent-child pair have higher correlation coefficient than dental characteristics indicating skeletal dimension to be much under genetic influence than dental characteristics. It has been a known fact that craniofacial abnormalities are not monogenic but are multifactorial, i.e. gene – environmental interaction. Heritability for skeletal variables viz position of lower jaw, anterior and posterior face heights, and cranial base dimensions is stronger, while that of dental variables was low. [17-24]

Class II division 1 malocclusion: Cephalometric studies have shown that

mandible in class II is significantly more retruded than class I patients, with the body

of mandible smaller and overall mandibular length reduced. These studies also show a higher correlation between the patients and his immediate family, thus supporting the concept of polygenic inheritance for class II division 1 malocclusion. [21-29]

Class II division 2 malocclusion: Its main features are deep overbite, retroclined

incisors, class II skeletal discrepancy, high lip line with strap like activity of the lower lip

and active mentalis muscle. There is a tendency to a forwardly rotating mandibular

movement, which contributes to deep bite, chin prominence, and reduced lower face height. Family and twin studies have shown the familial occurrence of class II division 2 malocclusion.

Class III malocclusion: Skeletal class III condition esp due to mandibular prognathism

observes familial inheritance. It was seen as Hapsburg jaw in a German royal family. Various familial studies of mandibular prognathism suggest heredity as the etiology of this condition. [16-24]

Cleft lip and palate: It is a very common congenital condition, and has a strong genetic link. The chances of CL/P increases in the children of parent/s having CL/P. [22-38]

Contemporary View on Inheritance

Primitive population with low prevalence of malocclusion has drawn the attention of researchers. It was thought that the coarse diet put increased functional demands on the jaws which led to normal growth of jaws in primitive population. Changes in dietary habits to soft and processed food reduced the functional demands on the jaws, thereby causing lesser growth and less attrition of teeth, leading to malocclusion in the existing population. [1-9]

Another aspect to explain the low prevalence of malocclusion in primitive population and high prevalence of malocclusion in the existing population came into view. The low prevalence of malocclusion in primitive population was explained on the basis of genetic isolation and uniformity since, all the individuals who were involved in mating, carried same genetic information. In modern times, genetic melting due to outbreeding led to increase in malocclusion. [11-20]

The skeletal characteristics are primarily under the control of hereditary factors showing less vulnerability of change under environmental influences, whereas dental characteristics showed low heritability and more vulnerability to change under environmental influences with advancing age. [11-38]

CONCLUSION

Role of heredity in growth and development of dentoskeletal complex cannot be underemphasised. Genes from both the parent guide the formation of jaws, teeth, soft tissues and their inter-relationship. Malocclusion is not inherited in a simple Mendelian fashion,

but shows a polygenic inheritance, accompanied by several environmental influences. Skeletal characteristics show much higher heritability than dental characteristics.

Tooth size and shape, congenitally missing teeth, supernumerary teeth and median

diastema are also under genetic control. Dental arch form and permanent first molar relationship, which are usually harmonious with certain skeletal pattern, are also under

genetic influences. As during treatment, the genetically determined factors cannot be

controlled, the main emphasis remains to control the environmental factors to treat or

improve the malrelation. Knowledge of genetic pattern of an individual helps in determining his expected growth and facial pattern and hence the interceptive methods (e.g. functional appliance, dentofacial orthopedic Rx) can be preplanned and instituted timely to avoid development of a severity of the condition. Also, advanced consultation and advice can be provided to the future parents to plan their family in consultation with genetic experts to avoid any congenital conditions in their children.

REFERENCES

1. Mencken HL. A Mencken chrestomathy. New York: A. A. Knopf, 1949.
2. Moss ML. The regulation of skeletal growth Regulation of Organ and Tissue Growth. Acad Press NY, 1972, 127-142.
3. King RA, Rotter JI, Motulsky AG. Approach to genetic basis of common diseases. Oxford Oxford University Press, 2002.
4. Gangane S. Human Genetics. 2nd ed New Delhi: Churchill Livingstone, 2000.
5. Bishara SE. Textbook of orthodontics, WB Saunders, 2001.
6. Abdulgani Azzaldeen. Genetics and Dental Disorders – A Clinical Concept. Part; 1. IOSR Journal of Dental and Medical Sciences (IOSR-JDMS). 2017; 16(11):35-42.
7. Proffit WR. Contemporary orthodontics. 4th ed. St Louis: Mosby, 200
8. Ellis E and McNamara JA Jr. Components of adult class III malocclusion. J Oral and Maxillofac Surg. 1984;42: 295–305.
9. Graber TM, Vanarsdall RL, Vig KWL (eds). Orthodontics: Current principles and techniques, 4th edition, St. Louis, Mosby, 2005.
10. Harris JE. Genetic factors in the growth of the head: Inheritance of the craniofacial complex and malocclusion. Dent. Clin. North Am 1975;19:151–60.
11. Lundstrom A. Tooth size and occlusion in twins. 2nd ed. S Karger AG, Basle, 1948.
12. Lundstrom A. Some asymmetries of the dental arches, jaws and skull, and their etiological significance. Am J Orthod 1961;47:81-106.
13. Moyers RE. Handbook of orthodontics. 4th edition. Year book medical publishers, Inc. 1988.
14. Abu-Hussein M, Watted N, Yehia M, Proff P, Iraqi F. (Clinical Genetic Basis of Tooth Agenesis, Journal of Dental and Medical Sciences. 2015; 14(12):68-77. Doi: 10.9790/0853-141236877
15. Abu-Hussein M, Watted N, Yehia M, Proff P, Iraqi F. Clinical Genetic Basis of Tooth Agenesis, Journal of Dental and Medical Sciences. 2015; 14(12):68-77. Doi: 10.9790/0853-141236877
16. Proffit WR, Phillips C, Dann C IV. Who seeks surgical-orthodontic treatment? The characteristics of patients evaluated in the UNC Dentofacial Clinic. Int J Adult Orthod Orthognath Surg 1990;5:153-60.
17. Abu-Hussein Muhamad and Nezar Watted et al; (2019) “Genetics and Orthodontics”, International Journal of Applied Dental Sciences; 5(3): pp384-390
18. Abu-Hussein M, Watted N, et al; (2015) “Clinical Genetic Basis of Tooth Agenesis” Journal of Dental and Medical Sciences; 14(12): pp68-77.
19. Abdulgani Azzaldeen, et al; (2017) “Tooth Agenesis; Aetiological Factors”. Journal of Dental and Medical Sciences; 16(1): pp75-85
20. Abu-Hussein Muhamad and Nezar Watted, et al; (2019) “Genetics in pediatric dentistry: A review”, International Journal of Applied Dental Sciences; 5(3): pp401-408
21. Kolomvos N, Azzaldeen A, et al; (2024) “Congenitally missing lateral incisors: Case management with a multidisciplinary approach”. J Adv Med Dent Scie Res;12(8): pp34-47.
22. Rand Ghoul, et al; (2025). “Multidisciplinary Approach To Treatment Of Midline Diastema”. Journal of Neonatal Surgery, 14 (22s) pp240-284
23. Hanali Abu Shilbayih, Nezar Watted, Muhamad Abu-Hussein (2016). “Multidisciplinary Aesthetic Dental Treatment; Peg lateral with Congenitally Maxillary lateral Incisors” -JDMS Volume 15, Issue 10 Ver. I PP 83-91
24. Qawasmeh Nour, Abdulgani Azzaldeen, et al; (2024). “Digital Technologies in Dentistry”. J Oral Dental Care;1(1): pp1-2.
25. Abu-Hussein Muhamad, Watted Nezar, et al; (2015) “The Curve of Dental Arch in Normal Occlusion”. Open Science Journal of Clinical Medicine. 3, 2, pp47-54

26. M. Abu-Hussein, Aspasia Sarafianou (2012). "Mathematical Analysis Of Dental Arch Of Children In Normal Occlusion: A Literature Review". International Journal of Medical Dentistry, 16,1, pp33-40
27. Abu-Hussein M.; Cleft Lip and Palate – Etiological Factors. Dent. Med. Probl. 2012, 49, 2, 149–156
28. Abu-Hussein M.; Cleft lips and palate ;the roles of specialists, Minerva Pediatr. 2011 .63(3):227-32.
29. Muhamad AH, Azzaldeen A (2012) Genetic of Non-syndromic Cleft Lip and Palate. 1:510.doi: 10.4172/scientificreports.510
30. Muhamad Abu-Hussein, Nezar Watted, Viktória Hegedűs, Péter Borbély, Abdulgani Azzaldeen (2015); Human Genetic Factors In Non-Syndromic Cleft Lip And Palate: An Update International Journal Of Maxillofacial Research 1,3,7-23
31. Abu-Hussein M.; Cleft Lip and Palate – Etiological Factors. Dent. Med. Probl. 2012, 49, 2, 149– 156
32. Abu-Hussein M.; Cleft lips and palate the roles of specialists, Minerva Pediatr. 2011 .63(3):227-32
33. Abu-Hussein Muhamad, Abdulgani Azzaldeen and Nizar Watted; CLEFT LIP AND PALATE; A COMPREHENSIVE REVIEW International Journal of Basic and Applied Medical Sciences 2014 Vol. 4 (1), 338-355
34. Abu-Hussein Muhamad, Abdulgani Azzaldeen, Watted Nezar, Kassem Firas. The Multifactorial Factors Influencing Cleft Lip- Literature Review. International Journal of Clinical Medicine Research. Vol. 1, No. 3, 2014, pp. 90-96
35. Cobourne MT, DiBiase AT. Handbook of orthodontics (1st edn), Elsevier, Edinburgh, London, New York, Oxford, Philadelphia, St Louis, Sydney, Toronto, 2009
36. M. Abu-Hussein, N. Watted, E. Hussien, A. Watted; Feeding Appliance for A Newborn Baby with Cleft Palate. International Journal Dental and Medical Sciences Research Vol 1, 6 (Nov- 2017), 05-09
37. Muhamad AH, Azzaldeen A. Genetic of Non-syndromic Cleft Lip and Palate, 2012.1:510.doi:10.4172/scientificreports.510
38. Strachan T., Read A.P. Human Molecular Genetics, 2nd ed.; New York: Wiley-Liss, 1999; Chapter 22.