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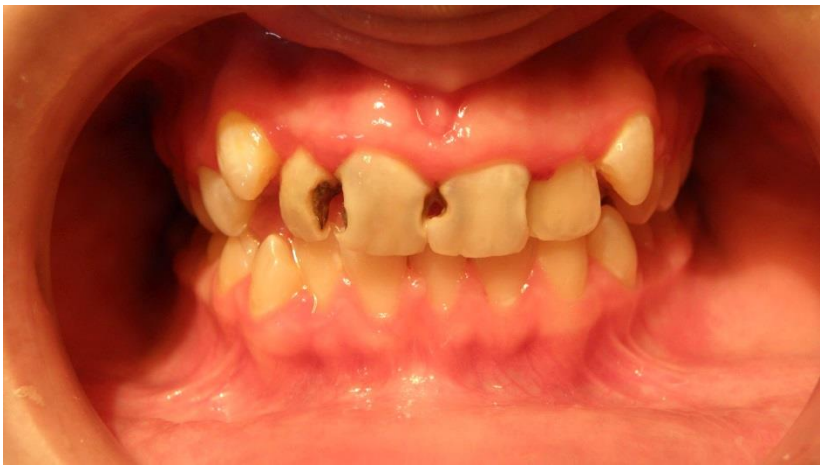
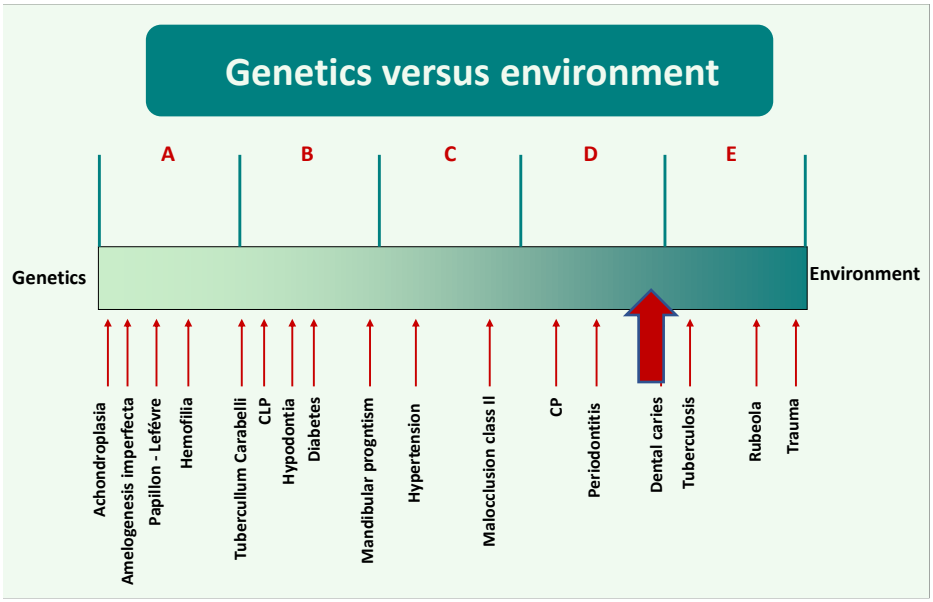
Heredity of dental caries



Every dentist has experienced families whose members attribute their susceptibility to dental caries to bad teeth inherited from parents. The dentist must differentiate if the problem is really inherited or if there are environmental factors included, such as diet or hygiene. Despite the infectious character of dental caries, anatomical factors, chemical composition of the tooth, composition and consistency of saliva, dietary habits, inborn taste preferences or immunological condition could modify its susceptibility.

Dental caries is a complex infectious disease and the susceptibility to it is multifactorial. In Stewart and Prescott the figure is situated on the environment side.



The caries disease model has been rapidly changing during last years. A lot of new publications have appeared.

Despite of its complex character, in some families presence of an underlying condition could imitate monogenic transmission of dental caries. It is called single gene effect. In the next picture is a family with fructose intolerance.

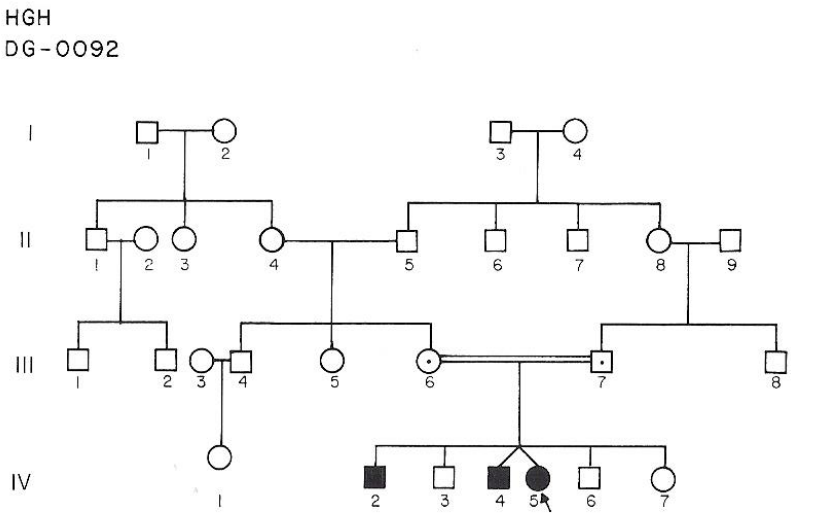
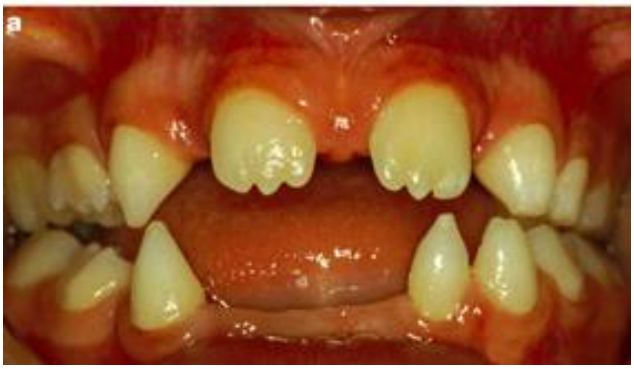


Fig. 3-7. Pedigree of family showing autosomal recessive inheritance of hereditary fructose intolerance, a metabolic disorder leading to intolerance of fructose from infancy and resulting in greatly reduced incidence of dental caries in affected individuals. Note horizontal transmission, both sexes affected, clinically normal parents, and consanguineous marriage of first cousins.

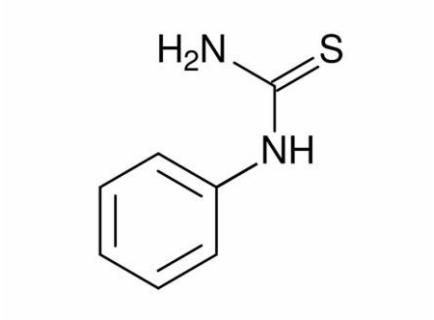
Following example of single gene effect is ectodermal dysplasia. Affected individuals have specific teeth, conic, shape reduced with absence of pits and fissures. And, as a result of the anatomic condition, very low caries risk.



Heredity has been linked with dental caries incidence in scientific literature for many years. Experimental animal studies has been proceeded from 40s. Laboratory rats were selectively breeded and after 28 generations genetically resistant and genetically susceptible strains were obtained. Placing caries–resistant or caries-susceptible newborns with opposite strain foster mother until weaning resulted in a caries index typical of the true prenatal strain (Hunt et all 1944).

In 60s special substance – phenylthiocarbamide was tested. Some people perceive the bitter taste of this substance and some not. E.g. 60% of all United States whites are tasters, 40 % are non-tasters

Tasters had 25% lower caries experience than non-tasters (Chung, Witkop et al, 1964)



Interesting population study was made in the north of Brasil -Amazonia. Colony of Santo Antonio do Prata is a former leprosarium with extremely isolated, poor and homogenous population. Complex segregation analysis was performed and existence of dominant major gene responsible for caries resistence has been proved (Werneck et al, 2011).

Twin studies still play role in contemporary genetics of dental caries.

Finn and Caldwel (1963*)* proved, that monozygotic twins showed fewer intrapair differences than did dizygotic and significantly fewer than did paired unrelated children.

Part of the Minnesota study of twins reared apart was also caries research. Conry et al (1993) examined teeth present, teeth restored, surfaces restored or carious in 46 MZ twin pairs and 22 DZ pairs. Despite being treated in different environment, different diet and different dental professional care, MZ pairs showed greater within-pair similarity than DZ.

Bretz et al (2005) evaluated the group of twins from poor area with no access to optimally fluoridated water or professional dental care – model for natural caries. The heritability estimation was 30 %.

Genomic studies drive heredity of dental caries research in last decade. The first genome-wide association scan for childhood caries was done in 2011 by Shaffer et al. This study reinforces the complexity of dental caries, suggesting that numerous loci, mostly having small effects, are involved in cariogenesis.

Numerous candidate genes connected with dental caries has been detected.

Firstly, it was genes involved enamel forming. It means genes for proteins ameloblastin, amelogenin, enamelin and tuftelin.(Deeley et al: 2008.)

The amelogenin (*AMELX*) gene resides on the p arm of the X chromosome It forms a scaffold for enamel crystallites and controls their growth. *AMELX* may be responsible for higher caries frequency in women.

ENAM gene has been repeatedly reported as a possible candidate for caries susceptibility.

The ameloblastin (*AMBN*) gene located on chromosome 4 is a key adhesion molecule

Genes for tuftelin and kallikrein 4 are also involved.

Attention is paid to genes connected with sweet and bitter perception *TASR1, TASR2, TASR38*

Variability of immunity complex and saliva composition may play a role too.

In the last figure from Stewart and Prescott you can see all factors influencing caries resistance and susceptibility together.

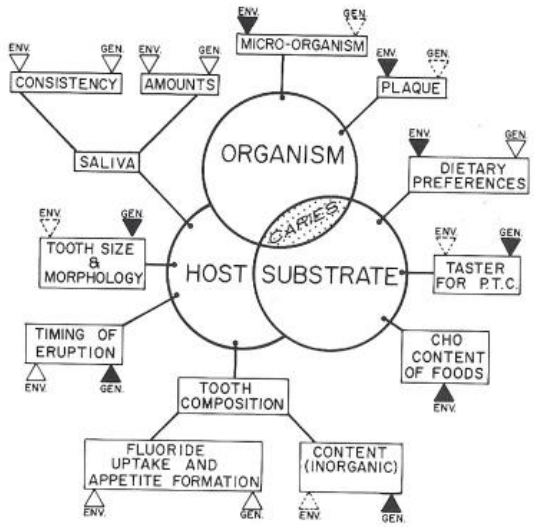


Fig. 4-3. Multifactorial etiology of dental caries, illustrating relationship between genetic and environmental factors affecting saliva, oral flora, enamel formation and mineralization, tooth shape and structure, and dietary habits. *Broken-line arrows,* minor contribution; *solid-line arrows,* moderate contribution; *filled arrows,* extensive contribution.

Currently prepared commercial dental panels offer possibility of testing resistance or susceptibility to common dental disorders including dental caries.