Taurodontism in Modern Populations

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ABSTRACT Taurodontism is a variation in root formation, resulting in an enlarged pulp chamber (Mena, 1971). This character has been used as a marker for differences between populations. Sex-linked disorders, autosomal chromosome disorders, and environmental factors have been reported to cause taurodontism (Reichart and Quast, 1975; Aldred and Crawford, 1988; Varrela and Alvesalo, 1989). When examining the mode of inheritance of taurodontism, it appears to be a polygenic trait that is controlled by only a few genes. At least one of these genes appears to be located on the X-chromosome. In addition, taurodontism appears to be linked to congenitally missing teeth, however these traits are not interdependent. Further study into the mode of inheritance of taurodontism and its relation to hypodontia is necessary to uncover the significance of taurodontism and its possible application to population studies. However, due to procedural differences in the assessment of this trait, the results between studies are difficult to compare. Therefore, a standard set of measurements is needed to make meaningful comparisons between studies of taurodontism.

INTRODUCTION

Part of understanding humanity is sought in the study of human dental variation. When Sir Arthur Keith first used the term taurodont in 1913, he was attempting to describe a variant in the pulp chamber of molar teeth to distinguish a monothetic difference between *Homo sapiens* and Neanderthals (Mena, 1971). However, cases of taurodontism in modern human populations have been reported (Goldstein and Gottlieb, 1973; Barker, 1976; Shifman and Buchner, 1976). Many researchers now focus upon determining what etiological factors cause this condition (Reichart and Quast, 1975; Varrela and Alvesalo, 1988, 1989). This study attempts to unravel the inheritance pattern of taurodontism.

Taurodontism is defined as an apical displacement of the furcation of the roots, resulting in an enlarged pulp chamber (Mena, 1971). Shaw (1928) created a typology for taurodont teeth by dividing them into three categories: hypotaurodont, mesotaurodont, and hypertaurodont (Fig. 1). Researchers identify taurodontism by comparing molar teeth to drawings similar to Figure 1 (Lysell 1962, 1965). While these categories are still utilized to describe the relative expression of this trait, clear breaks between them do not exist (Laatikainen and Ranta, 1996). In order to correctly recognize taurodontism and compare results with other researchers, a standard method that is more objective than those available is necessary. Many researchers have devised different methods by measuring various criteria, yet no particular scheme is used consistently (Blumberg et. al, 1971; Shifman and Chanannel, 1978; Seow and Lai, 1989). Due to the inconsistencies in the methods of measurement, these studies produce results that are not directly comparable.

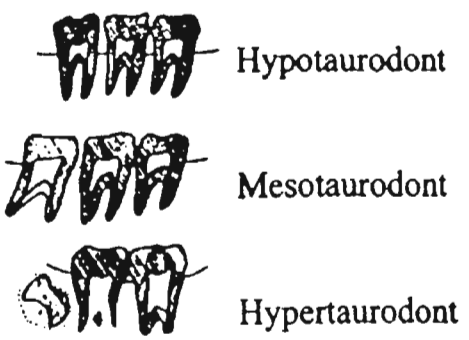

Fig 1. Types of taurodont teeth. Adapted from Lysell (1962).

Lysell (1962) mentions that the degree of taurodont expression decreases from the first molars to the third molars. Following the Butler-Dahlberg polar field concept, the first molar is the most stable and least effected while the third molars are the least stable and most effected (Scott and Turner, 1997). Lysell suggested the use of second molars as the standard for measurement because they are the most likely to express the trait consistently. In addition, the three roots of the maxillary molars tend to obscure identification of taurodontism, resulting in many researchers scoring mandibular molars (Shifman and

Chananmel, 1978; Brinkmann and Scheil, 1993). Yet, scoring mandibular molars is not conventional. Some researchers score both maxillary and mandibular dentition, while others score only maxillary dentition (Blumberg et al., 1971; Darwazeh et al., 1998).

THE USE OF TAURODONTISM

Dental traits, such as taurodontism, can be key in understanding human population variation. When traits are known to be independent, selectively neutral, and easily observable, they can be used to generate information that is characteristic of populations (Scott and Turner, 1997). Crown traits have been the most intensively studied thus far (ibid.). However, with the use of radiographs and orthopantomograms, tooth roots are as easy to observe as crown traits (Tulensalo et al., 1989). In addition, with time and intensive chewing, crown traits can wear off (Scott and Turner, 1997). Taurodontism, a root trait, will persist longer when under duress. Yet, as the crown wears into the dentinal area, secondary dentine is laid down in the pulp chamber and can obscure taurodontism. Taking these factors into account, taurodontism must also be established as an independent or selectively neutral trait.

INHERITANCE PATTERNS OF TAURODONTISM

Genealogical studies on the incidence of taurodontism provide insight into the inheritance pattern of this trait. Laatikainen and Ranta (1996) studied taurodontism in association with cleft lip and/or cleft palate in twins. They determined that taurodontism was present in 31% of monozygotic twins and 46% of dizygotic twins in 39 pairs. Thirty seven percent of these twins were discordant for clefting, though relatively more extreme clefting was highly associated with a higher expression of taurodontism. Also, a pedigree analysis of taurodontism associated with X-linked hypohidrotic ectodermal dysplasia (XHED) showed an increased expression of taurodontism in males than in related females (Crawford et al., 1991). These studies suggest that the expression of taurodontism has a strong genetic component. In addition, both studies linked the expression of taurodontism with hypodontia.

TAURODONTISM ASSOCIATED WITH SEX-LINKED DISORDERS

Studies focusing upon X-linked disorders indicate a high correlation between taurodontism and the presence of extra X-chromosomes. Varrela and Alvesalo (1988, 1989) have examined the effects of extra X-chromosomes in males and females with respect to the occurrence of taurodontism. Focusing upon the mandibular dentition, they determined that 30% of males (n=66) with an extra X-chromosome displayed taurodont molars and 67% of women (n=6) with at least one extra X-chromosome displayed taurodont molars. Their control groups, consisting of normal males and females, had frequencies of taurodontism at 2.5% (n=157) and 2.6% (n=157), respectively. In addition, one relative of a study female displayed taurodont molars. Studies of Turner syndrome, characterized by X-chromosome deficiency (XO), have revealed no patients exhibiting taurodont molars (Midtbo and Halse, 1994; Farge et al., 1985). These studies suggest that genes located on the X-chromosome regulate the expression of taurodont molars.

At a cellular level, the epithelium produces enamel, while the mesoderm produces dentin (Scott and Turner, 1997). Analyzing the ontogeny of teeth, Alvesalo and co-workers (1987, 1991, 1997) and Varrela et al. (1988) have shown that the X-chromosome is involved in enamel production, while the Y-chromosome regulates enamel and dentin production. Hamner and co-workers (1964) noted that the production of dentin is normal in taurodont teeth. They deduced that taurodontism must result from a malfunction in the formation of Hertwig's epithelial root sheath, because dentin is laid down after root sheath production. This means enamel production is in part regulated by sex chromosomes, while the epithelium creates enamel and Hertwig's root sheath. The connection between these factors requires additional study to determine the involvement of sex chromosomes in the production of taurodont teeth.

TAURODONTISM ASSOCIATED WITH AUTOSOMAL CHROMOSOME DISORDERS

Taurodontism is also often reported in association with disorders or syndromes that are inherited on autosomal chromosomes. In a study of 22 children with Down's syndrome, or trisomy 21, Alpoz and Eronat (1997) found that 66% of the children had taurodont molars, while none of the 20 control children exhibited taurodontism. Similarly, Bell and co-workers (1989) examined 33 individuals with trisomy 21 and found that 34.8% of them had taurodont molars. In addition, both studies describe hypodontia and delayed eruption as effects of Down's syndrome. Studies focusing upon the connection between hypodontia and/or oligodontia and taurodontism indicate an association between these two dental traits.

Hypodontia is defined as the congenital absence of at least one tooth, while oligodontia is defined as the absence of six or more teeth within an individual (Shalk-Van DerWeide, et al., 1993). Brook (1984) has determined that hypodontia is more frequently expressed in females, while hyperdontia is more frequent in males. This implies that the inheritance of these traits is sex-linked.

Seow and Lai (1989; Lai and Seow, 1989) studied the relationship between hypodontia and taurodontism. They determined that out of 66 and 67 patients, 34.8% and 34.3% of them displayed the co-occurrence of hypodontia and taurodontism respectively. Their control samples of 66 and 67 individuals with full permanent dentition had rates of 7.5% and 7.1 % of taurodontism respectively. They suggest that either taurodontism and hypodontia are genetically linked or that they occur due to the same unnamed environmental influence. Using the same method of analysis, Shalk-Van Der Weide and researchers (1993) examined the occurrence of taurodontism and oligodontia. Out of 91 patients and 90 control subjects, they found that taurodontism and oligodontia occur together 28.9% of the time, while taurodontism occurred at a rate of 9.9% in the normal population. Also, a study on short root anomalies mentions the occurrence of taurodontism and hypodontia in families (Apajalahti et al., 1999). These findings strongly suggest a correlation between taurodontism and congenitally missing teeth. However, these traits do occur in the absence of one another, so they are not fully interdependent.

Autosomal disorders occurring in the absence of hypodontia while exhibiting taurodontism also have been reported. Case reports of these conditions associated with taurodontism include affiliation with supernumerary teeth, dentinal dysplasia type I, short roots, Ellis-van Creveld syndrome, hypophosphaetaemic vitamin D resistant rickets, and amelogenesis imperfecta (Gardner and Girgis, 1977; Aldred and Crawford, 1988; Crawford and Aldred, 1998; Crawford et al., 1988; Goodman et al., 1998; Hattab et al., 1998; Genc et al., 1999; Kosinski et al., 1999). Unfortunately, most of these conditions have not been systematically studied with the intent of determining their relationship to taurodontism.

Taurodontism has been investigated with respect to one type of amelogenesis imperfects (AI H-H T) (Aldred and Crawford, 1988; Crawford et al., 1988; Winter, 1996). Amelogeneis imperfecta (AI) is a discoloration of anterior tooth enamel, resulting from an autosomal dominant, autosomal recessive or X-linked abnormality (Winter, 1996). In a sample of 32 children with AI, 87.1 % of them were found to have taurodont molars (Winter, 1996). Genealogical analysis suggests that AI associated with taurodontism is inherited through an autosomal dominant mechanism (Congleton and Burkes, 1979; Crawford et al.,1988). Studies of the relationship between AI and taurodontism have only recently begun. Further examination into their association is required before any conclusions can be drawn.

TAURODONTISM ASSOCIATED WITH ENVIRONMENTAL FACTORS

Environmental factors have also been shown to influence the expression of taurodontism. Reichart and Quast (1975) examined an individual who exhibited a single taurodontic lower third molar. Between the ages of four and twenty, this individual had a mandibular osteomyelitis infection. Thus, taurodontism in this case appears to be the direct result of a childhood infection. This brief case report indicates that taurodontism can be caused by factors that are not genetic. While previous evidence suggests that taurodontism is often inherited or displayed due to genetic influence, environmental factors may also be responsible for some cases of taurodontism.

Table 1. Population Studies of the Incidence of Taurodontism

Population	%	#	Age	Method	Researcher
Jordan	8.0	875	>18 years	Subjective	Darwazeh, et al (1998)
Iarael	5.6	1,200	20-30 years	Shifman and Channanel measurements	Shifman and Channanel (1978)
Saudi Arabia	11.3	1,281	unreported	Shifman and Channanel measurements	Ruprecht et al. (1987)
American Whites	0.5	2,800	adult	Unreported	Witkop (1976)
American Blacks	4.4	1,074	children<18	Jorgenson et al. subjective measurements	Jorgenson et al. (1982)
Chicago	2.5	11,905	unreported	Reported in Ogden (1988)	Blumberg et al. (1972)
Greece	1.1	730	adult	Zografos et al. measurements	Zografos et al. (1991)
Britian	6.3	1,115	children	Reported in Ogden (1988)	Holt and Brook (1979)
China	46.4	196	15-19 years	Shifman & Channanel modified	MacDonald-Jankowski and Li (1993)

% is the percent of individuals. # is the number of individuals.

RATES OF TAURODONTISM IN POPULATIONS

Population studies, focused on the general public, have examined the incidence of taurodontism. When analyzing taurodontism occurring in the absence of genetic anomalies, it appears to be expressed in different frequencies in different populations (Table 1). These results suggest that taurodontism can be used in conjunction with other traits to identify populations. Yet, many problems arise when comparing these studies. First, taurodontism was identified using different standards between studies. Second, molars used to identify taurodontism vary from examining only first upper molars, only recording the trait on second lower molars, or using any molar that exhibits taurodontism as evidence (Blumberg et al., 1971; Shifman and Channanel, 1978; Darwazeh et al., 1998). In addition, some studies examine adults, some examine children, while others include both in analyses. Though taurodontism occurs in both deciduous and permanent dentitions (Dayan et al., 1984), dental traits can show differential degrees of expression in both cases, again confounding comparison (Scott and Turner, 1997). Third, all studies were performed on living populations that, with a few exceptions, experience greater gene flow than in the past. Therefore, admixture will factor into the reported gene frequencies.

Limitations to the study of taurodontism must be considered. Brinkmann and Scheil (1993) note that dental caries and secondary dentine obscure the identification of taurodontism. They also state that because taurodontism is a continuous trait, identifying the less expressive end can be difficult, especially when subjectively identifying the trait. In addition, most studies focused on taurodontism analyze clinical patients, therefore the samples are not random. Although these conditions exist, carefully constructed research can avoid possible misdiagnosis of the trait and heavily biased samples.

CONCLUSION

The mode of inheritance of taurodontism is still unclear. While Mendelian inheritance patterns do not appear to fit the data, its wide occurrence with many genetic abnormalities suggests that it is very susceptible to change. Researchers generally agree that taurodontism is a polygenic trait (Blumberg et al., 1971). Most polygenic crown traits are resistant to change because many genes are needed to express them (Scott and Turner, 1997). Taurodontism, while polygenic, appears to be controlled by only a few genes. In addition, the clinical research presented here suggests that at least one gene controlling the expression of taurodontism occurs on the X-chromosome.

Further analysis into the relationship between taurodontism and hypodontia is needed. Hypodontia is thought to be under selective pressure favoring dental reduction (Scott and Turner, 1997). If taurodontism is a variation of the adaptation towards dental reduction, it will be necessary to understand the selective

pressure effecting the appearance of this trait. In addition, if taurodontism is not an independent trait, it will not necessarily reflect the same genetic processes in all people, thus its use as a population marker would have to be qualified. Comparisons between studies of taurodontism, one set of measurements taken on a specific molar or set of molars is needed.

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