

Morphogenetic fields within the dentition

Grant C. Townsend, B.D.S., B.Sc.Dent. (Hons.), Ph.D. and
Tasman Brown, D.D.Sc., F.R.A.C.D.S., University of Adelaide, South Australia

Abstract

The concept of genetic control of tooth size and shape by specific morphogenetic fields that control tooth formation originated from studies of the mammalian dentition and later, from the observation that within the human dentition certain key teeth within a class were more stable in size and morphology. More recently, analyses of tooth size from different populations have supported the general ideas inherent in this field theory, however multivariate analyses of tooth size relationships suggest that this genetic control may be more complex than previously envisaged. The precise nature of cellular and tissue interactions during odontogenesis is not fully understood but experimental studies point to the important role of the dental papilla and perhaps specific RNA sequences, proteoglycans and collagens are important in these epithelial-mesenchymal interactions. Such experimental studies will help to clarify not only the nature of the odontogenic process but also the extent of the involvement of the genetic field and clone theories for tooth formation. This paper summarizes some of the evidence for morphogenetic control over tooth formation that has accrued from morphological and experimental studies. It also suggests directions along which future studies are likely to proceed.

Key Words: Tooth morphology, dental asymmetry, tooth size, genetic control.

INTRODUCTION

Morphogenetic fields are considered to pertain to specific regions under direct genetic control which determines the differentiation, growth and final morphology of structures developing within a specific field. The theory of morphogenetic fields is also used to explain the morphological gradients and patterns, such as shape and size, which are observed in a series of structures with similar basic forms, for example, phalanges and teeth. Despite widespread support for the concept of such fields, knowledge of the specific genetic and biochemical mechanisms involved in tissue differentiation is limited.

With respect to dental development, Butler (1939) analyzed the morpho-differentiation of teeth in fossil mammals and postulated that there were three distinct fields which corresponded to incisor, canine and molar tooth groups existing in the dentition. He suggested that each tooth group was under genetic control, but the extent of this control varied in intensity within each group. Dahlberg (1945) adapted Butler's theory to the human dentition and described not three but four morphogenetic fields: incisor, canine, premolar and molar. The most stable tooth within each field, termed the polar or key tooth, was thought to be nearest the centre of the overall morphogenetic influence. In the human dentition the key teeth were the maxillary II, C, PI, and MI, and the mandibular I2, C, PI and MI. Most evidence for the existence of morphogenetic fields has been derived from metrical and non-metrical studies of the dentition, but recent experimental investigations

have also thrown light on the biological mechanisms of differentiation in dental tissues.

MATERIALS AND METHODS

Odontometrical data derived from 392 dental casts, representing 184 females and 208 males, collected during a longitudinal growth study of the dentofacial characteristics of Aborigines living in the Northern Territory of Australia, have led to analyses of mesiodistal and buccolingual crown diameters of deciduous (Margetts & Brown, 1978) and permanent dentitions (Townsend & Brown, 1979a); dental asymmetry (Townsend & Brown, 1980); tooth size relationships within and between dentitions (Brown et al., 1980a, b); and inheritance of tooth size (Townsend & Brown, 1978a, b; Townsend, 1980).

A semi-automatic system was used to measure and record tooth diameters with an accuracy of 0.1 mm. Modified Helios dial calipers fitted with needle points and wired into an analog-digital converter unit, enabled metric data to be punched directly on to computer cards.

Measurement errors were analyzed by recording double determination measurements for 50 sets of casts selected at random. Errors were very small, the weighted average standard deviation of single determination being 0.11 mm for both mesiodistal and buccolingual dimensions.

RESULTS AND DISCUSSION

Morphological Evidence for Fields

Some of the findings from the Aboriginal

studies as they relate to the dental field theory are summarized:

i. Tooth Size Variability

Table 1 presents tooth size statistical data for Aboriginal males and females. As indicated by the coefficient of variation, the variability in mesiodistal and buccolingual diameters as well as that for the derived crown parameters, module, index and area, generally follows the pattern proposed by Dahlberg (1945). With few exceptions the key tooth of each class shows least variability. This pattern is particularly clear in all cases for the molars and the maxillary incisors, where the trend to increased variability towards the distal of the series is marked. In the mandibular incisors the I2 is the key tooth and, according to expectations, the least variable incisor for all comparisons, except for the mesiodistal diameter in males. In most instances the first and second premolars were similar in variability and the expected trend was not so obvious as in other tooth classes.

ii. Dental Asymmetry

Table 2 presents the means and variances for right-left differences in size of the permanent teeth in Aboriginals. The tendency for asymmetry to increase within tooth classes further away from the key tooth is striking and, with only one exception in females and two in males, this pattern was observed in all comparisons. Third molars showed greatest asymmetry and, in general, the lower incisors least. Whilst most size differences between corresponding right and left teeth were about 0.2–0.3 mm, occasional differences of 0.8–0.9 mm were noted. The significance of these higher differences, which could not be accounted for by measurement error, needs further clarification.

Although analyses of tooth size measurements have failed to indicate any systematic differences between right and left sides; small non-directional differences are common (Townsend & Brown, 1980). These random discrepancies may indicate an inability of the individual to buffer against developmental disturbances (Van Valen, 1962). The degree of this type of asymmetry, which is often termed fluctuating asymmetry, is thought to reflect the magnitude of local developmental disturbances, or as Waddington (1957) described it, developmental "noise". Some individuals, depending on their genotypes, are better buffered against "noise" than others and display greater developmental homeostasis. It appears that polygenic systems may buffer the developmental processes against environmental insults, whilst deleterious genes may reduce the buffering below a threshold level (Shapiro, 1970, 1975). Support for this

hypothesis is provided by observations that dental asymmetry is increased in individuals with genetic disorders, for example, cleft lip (Adams & Niswander, 1967; Sofaer, 1979), and Down's Syndrome (Garn et al., 1970).

In the same way that comparisons of asymmetry between individuals provide insight into genetic mechanisms, comparisons of asymmetry between teeth within individuals throw some light on the concept of morphogenetic fields. Asymmetry tends to increase from the key tooth to the more distal members of a tooth class (Lundstrom, 1960; Garn et al., 1966, 1967; Bailit et al., 1970).

iii. Heritability of Tooth Size

The heritability of a character is defined as the amount of variation in a population due to genetic differences between individuals.

Table 3 presents heritability estimates for permanent tooth size derived from correlations between Aboriginal full-siblings. Heritability estimates for earlier developing teeth are higher in some instances but there are several exceptions to the expected trend. Therefore, although heritability estimates for tooth size in Australian Aboriginals suggest a decrease in genetic control for distal members of tooth classes, the results are by no means conclusive.

Alvesalo & Tigerstedt (1974) and Sofaer et al. (1971a) have suggested that earlier developing teeth in a tooth class display higher heritabilities than later developing members of the same class, for example first and third molars. Furthermore, Sofaer et al. (1972) reported that genetic factors contributed less to the variability in size of later developing members within a tooth class. They also suggested that the resulting increased morphological variability in later developing teeth arose from a relatively larger environmental contribution to their variance in size and shape.

iv. Tooth Size Correlations

Table 4 presents correlations between tooth dimensions in the same arch for Australian Aboriginals. The pattern of correlations was similar for males and females, with very few significant differences and therefore average correlations for both sexes combined are presented. All coefficients are significant at $p < 0.05$. Generally, correlations between adjacent teeth (e.g. I1 and I2), are higher than between more distant teeth (e.g. I1 and M2). Furthermore, comparisons between maxillary and mandibular arches indicate a fairly high level of coordination between corresponding teeth (Table 5). It is also interesting to note that correlations between the combined diameters of groups of teeth (e.g. maxillary and mandibular incisors) tend to be higher than those between

Table 1. Coefficients of variation for tooth size variables measured on Australian Aborigines.¹

TOOTH	Mesiodistal Diameter		Buccolingual Diameter		Crown Module ²		Crown Index ³		Crown Area ⁴	
	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
Maxilla										
I1	6.1	6.0	7.0	6.1	5.6	5.2	6.9	5.9	11.3	10.5
I2	8.2	9.1	8.0	7.7	6.8	7.2	8.9	8.6	13.7	14.3
C	6.3	5.5	6.6	6.0	5.5	5.1	6.5	5.4	11.0	10.2
P1	5.9	5.4	5.3	5.5	5.1	5.0	4.6	4.5	10.4	9.9
P2	6.0	5.7	5.3	5.5	5.1	5.0	4.6	4.9	10.3	10.1
M1	5.0	4.6	4.5	4.4	4.3	4.0	4.0	4.1	8.7	8.0
M2	5.9	5.7	5.4	5.0	5.1	4.7	5.1	4.8	10.3	9.6
M3	6.5	7.0	6.5	6.3	5.9	6.0	6.3	5.9	11.9	11.9
Mandible										
I1	6.2	7.4	6.8	6.2	5.3	5.6	7.7	7.9	10.7	11.4
I2	6.4	6.1	6.3	5.9	5.2	5.0	7.4	6.9	10.5	9.9
C	5.9	5.1	6.1	5.4	5.2	4.5	6.3	5.3	10.4	8.9
P1	6.5	5.8	6.5	6.2	5.6	5.4	6.5	5.5	11.4	10.8
P2	6.7	5.8	5.9	6.1	5.6	5.5	5.4	4.9	11.4	11.0
M1	5.0	4.4	4.9	4.6	4.4	4.1	4.2	3.8	8.8	8.2
M2	6.0	5.6	5.1	5.0	5.1	4.8	4.3	4.0	10.2	9.7
M3	8.8	6.9	6.1	6.0	6.8	6.1	7.4	4.4	13.7	12.3

¹Data from Townsend & Brown (1979) which provides sample sizes, means and variances for all variables.

Standard errors of the coefficient of variation ranged from 0.20 to 1.36, being dependent on the sample size.

²Crown module = (Mesiodistal diam. + Buccolingual diam.)/2.³Crown Index = (Buccolingual diam. × 100)/Mesiodistal diam.⁴Crown Area (robustness) = Mesiodistal diam. × Buccolingual diam.**Table 2.** Dental asymmetry in crown diameters of the permanent teeth in Australian Aborigines.¹

TOOTH	MESIODISTAL				BUCCOLINGUAL			
	Males		Females		Males		Females	
	$\bar{x}$ diff*	S ²	$\bar{x}$ diff	S ²	$\bar{x}$ diff	S ²	$\bar{x}$ diff	S ²
Maxilla								
I1	.03	.051	.01	.044	.02	.052	-.02	.032
I2	.05	.061	-.01	.073	.04	.102	.02	.074
C	.09	.085	.06	.056	.05	.057	.02	.039
P1	-.01	.065	.01	.043	.00	.055	-.01	.045
P2	.04	.086	.04	.058	-.03	.059	-.03	.058
M1	.01	.089	.01	.065	.04	.065	.02	.056
M2	.08	.129	.10	.120	.00	.100	.05	.079
M3	.20	.324	.12	.183	-.07	.276	.11	.241
Mandible								
I1	-.03	.049	-.01	.038	-.03	.039	.01	.038
I2	-.08	.044	-.07	.039	-.01	.058	.02	.035
C	-.07	.053	-.05	.050	-.03	.058	.01	.033
P1	.08	.083	.03	.057	.05	.076	.05	.054
P2	.02	.114	-.02	.076	.00	.070	-.01	.058
M1	.09	.097	.09	.078	-.07	.070	-.12	.054
M2	-.04	.140	.01	.152	-.04	.098	-.05	.082
M3	-.01	.293	.01	.202	-.01	.337	-.01	.139

¹Data from Townsend & Brown (1980) which provides sample sizes ranging from 36 for the mesiodistal dimension of mandibular third molars in males to 207 for the buccolingual dimension of maxillary first molars in males.* $\bar{x}$ diff = $\frac{1}{N} \sum (R-L)$.

Table 3. Heritability estimates, h^2 , and standard errors, SE, for permanent tooth size in Australian Aborigines derived from full-sibling correlations.¹

TOOTH	MESIODISTAL		BUCCOLINGUAL	
	h^2	SE	h^2	SE
Maxilla				
I1	0.80	0.08	0.91	0.08
I2	0.41	0.09	0.60	0.09
C	0.65	0.08	0.58	0.09
P1	0.87	0.08	0.85	0.08
P2	0.88	0.08	0.88	0.08
M1	0.87	0.08	0.85	0.08
M2	0.80	0.09	0.93	0.08
Mandible				
I1	0.77	0.08	0.77	0.08
I2	0.69	0.08	0.61	0.09
C	0.76	0.08	0.82	0.08
P1	0.67	0.08	1.01	0.07
P2	0.67	0.09	0.78	0.08
M1	0.57	0.08	0.91	0.08
M2	0.63	0.09	0.87	0.08
Weighted mean	0.72 $\pm$ 0.08		0.81 $\pm$ 0.08	

¹ h^2 calculated as twice the full-sibling intraclass correlations, that is $h^2 = 2r_{FS}$. The analysis included 261 offspring derived from 67 fathers and 115 mothers.

Table 4. Correlations between pairs of permanent teeth in Australian Aborigines. Mesiodistal diameters (upper right half of each matrix) and buccolingual diameters (lower left). Data for males and females combined.

MAXILLA							
	I1	I2	C	P1	P2	M1	M2
I1	—	.60	.52	.55	.46	.56	.37
I2	.69	—	.48	.39	.33	.43	.22
C	.57	.54	—	.62	.48	.48	.40
P1	.52	.47	.67	—	.73	.45	.53
P2	.46	.42	.58	.81	—	.56	.53
M1	.59	.47	.59	.66	.71	—	.59
M2	.52	.41	.59	.63	.71	.80	—
MANDIBLE							
	I1	I2	C	P1	P2	M1	M2
I1	—	.67	.51	.39	.38	.46	.44
I2	.82	—	.63	.46	.35	.46	.46
C	.71	.74	—	.56	.46	.44	.49
P1	.58	.60	.65	—	.68	.47	.53
P2	.57	.54	.59	.75	—	.46	.58
M1	.54	.52	.51	.64	.70	—	.66
M2	.63	.57	.59	.66	.72	.77	—

All coefficients differ from zero at $p < .05$

individual teeth, both in the permanent and deciduous dentitions (Brown et al., 1980b). The various correlation studies of the Australian Aboriginal dentition have indicated that a reasonably strong control effects overall coordination of tooth size within arches, between maxillary and mandibular arches and between deciduous and permanent dentitions. Most likely a form of morphogenetic influence exerts this coordinating effect to produce an overall harmony of size which optimizes occlusal relationships.

Whilst correlation analyses provide some evidence for morphogenetic control, it is often difficult to interpret the extensive data that may accrue from the calculation of size relationships between a large number of teeth. Interpretative problems of this nature may be reduced by the use of multivariate procedures, such as factor analysis, which enable a number of interdependent variables to be analyzed collectively rather than individually or by pairs. Multivariate analysis not only provides a useful means of summarizing data, but it also facilitates the interpretation of complex relationships.

Factor analyses of mesiodistal and buccolingual tooth dimensions in different populations have generally disclosed factors corresponding to distinct tooth groups, indicating that fields of growth affect incisors, canines, premolars and molars separately (Potter et al., 1968; Lombardi, 1975; Hanihara, 1976). However, as Lombardi (1975) pointed out, a clearer insight into the nature of morphogenetic fields is obtained by considering both sets of dimensions together, thereby taking the inter-correlations between the groups into account. Intuitively this seems a better approach as the inclusion of both dimensions provides a more complete representation of tooth size.

Factor analysis of mesiodistal and buccolingual tooth dimensions in Australian Aborigines (Townsend & Brown, 1979b) indicated that four distinct factors may be operating on the

dentition. These affect the mesiodistal dimension of anterior teeth, the buccolingual dimensions of anterior teeth, the overall premolar size and the overall molar size.

With respect to the interpretation of morphogenetic influences, these statistical findings for factor analysis are similar to those for other populations (Potter et al., 1968; Lombardi, 1975; and Hanihara, 1976) thus adding some validity to the overall concepts inherent in morphogenetic field theory. However, it would appear that whilst the results of univariate statistical procedures generally support the notion of dental fields as proposed by Dahlberg (1945), the application of multivariate analysis, which enables simultaneous statistical examination of the role of a number of variables, points to a more complex model involving other mechanisms of tooth size coordination not only within groups but also within maxillary and mandibular arches.

Field Model of Developing Tooth Morphology

The field model to explain final tooth shape proposed that a specialized region termed a "field generator" differentiates early in the development of a series of related structures. This field generator produces a "field substance" which diffuses through the growing region (Osborn, 1978).

Field substance is thought to be destroyed slowly as it diffuses through the tissues so that the further a region is from the field generator the lower will be the concentration of field substance. Primordia are thought to form initially as a result of a totally separate control mechanism. Although theoretically all primordia in a tooth class are equivalent initially, the varying concentrations of field substance result in a morphological gradient of structure developing within the field. Furthermore, the field model postulates that all tooth primordia have the same set of target sites for the field substance and that different targets are sensitive

Table 5. Correlation coefficients for corresponding crown diameters in the maxilla and mandible in Australian Aborigines. Data for males and females combined.

TOOTH	MESIODISTAL		BUCCOLINGUAL	
	N	r	N	r
I1	172	0.67	170	0.72
I2	166	0.58	152	0.58
C	159	0.72	150	0.75
P1	161	0.76	160	0.70
P2	155	0.66	154	0.78
M1	171	0.66	172	0.79
M2	132	0.73	124	0.78

All coefficients differ from zero at $p < .01$

to specific concentrations of field substance, or possibly to different field substances. The final tooth shape depends on the particular targets which are selectively activated for each developing tooth or group of teeth.

Clone Model of Developing Tooth Morphology

An alternative model, termed the clone model, was recently proposed by Osborn (1978), to account for observed gradients in tooth form. According to this model, teeth within each class develop from a clone of cells which grows posteriorly along the forming dental lamina. Tissues become competent to initiate a new primordium only after a certain number of cell divisions have occurred and new primordia are initiated as space becomes available. Osborn (1978) has proposed that the gradients in shape and size of developing structures result from the cell ancestry of primordial tissue, for example, later forming tooth primordia are derived from cells that have undergone more divisions than those in earlier primordia. (See Figure 1)

Osborn (1978) also proposed that the final shape of a tooth results from the relative growth rates of the tissues in the primordia, that is in the internal enamel epithelium and the dental papilla. He suggested that a force analogous to viscous drag restrains the internal enamel epithelium from sliding over the developing dental papilla during its growth. The final shape of the tooth could then depend on the initial growth rates of the two adjacent tissues, and the gradients in tooth shape within a series may reflect a gradient in initial growth rates of successive primordia resulting from a decrease in the growth rate of a clone of cells as it grows distally. (See Figure 2)

Role of Neural Crest Cells

The formation of dental tissues in vertebrate embryos can be considered to commence with the appearance of a neural plate on the dorsal aspect of the notochord. As the neural plate is converted into a tube some ectodermal cells, derived from the margins of the plate, are not incorporated in the neural tube but persist as lateral projections. These projections, termed the neural crest, consist of cells which are thought to migrate throughout the embryo. Although the factors influencing migration of neural crest cells are not clear it appears that some are conditioned to differentiate into dental papilla cells or so-called ecto-mesenchyme (Slavkin, 1974).

The interaction between ecto-mesenchyme, derived from neural crest cells, and the adjacent oral epithelium is believed to determine the final tooth form (Slavkin, 1974). Neither the oral

epithelium nor the adjacent mesenchyme can differentiate into a developing bud in isolation from each other. It appears that the oral epithelium serves more of a maintenance than an instructive function, whereas the connective tissue of the dental papilla may be the main determinant of tooth form (Kollar, 1972).

Interdependence of Dental Epithelium and Papilla

Experimental interchanges of enamel organs and dental papillae from different regions have confirmed the important role of the papilla (Kollar, 1975). For example, if an incisor enamel organ is recombined with a molar papilla, a molar tooth bud results. In contrast, when an isolated enamel organ is recombined with connective tissue from an edentulous area, the dental epithelium loses its potential to form a tooth and instead it becomes a keratinizing layer of stratified cells (Kollar, 1975).

The response of the dental epithelium to foreign connective tissue is particularly interesting. Kollar (1972) has shown that if an isolated enamel organ is placed on non-dental connective tissue, for example the plantar surface of the embryonic foot, the enamel epithelium invades the underlying connective tissue and shows cellular alterations similar to those present in ameloblastoma. Alternatively, if dental papilla connective tissue is recombined with plantar epithelium, tooth development occurs once again.

There is good evidence that some form of molecular inter-communication occurs as epithelial and connective tissue elements interact. The work of Koch (1967), in which enamel organ and dental papilla were separated and then recombined across a filter of known pore size and thickness producing subsequent tooth development, provides a strong argument for some molecular inter-communication. Furthermore, isolated tissues when recombined with an extra-cellular organic matrix free of cells, also result in the formation of tooth-like tissue.

Role of RNA in Cellular Inter-communication

A number of substances or constituents within the extracellular matrix have been implicated in providing the necessary communication between epithelium and mesenchyme. On the basis of biochemical and electron microscopic evidence, Slavkin and co-workers have suggested that discrete membrane packets of RNA (ribonucleic acid) are transmitted from the dental papilla to the epithelium to serve as the cue for initiation of odontogenesis (Slavkin, 1974). It was proposed that the RNA packets transferred in this manner could result in some direct instruction message

to cells, similar to that occurring with the RNA message in viral transformation. Both in vitro and in vivo studies have shown that the predentine extracellular matrix contains RNA (Slavkin, 1974) and, as there are numerous proteases and nucleases present in the matrix, it seems likely that the RNA is bound in vesicle form. Slavkin proposed that the matrix vesicles of RNA, varying in size from 500–1000 Å may be secreted in a manner similar to that occurring at nerve terminals.

Role of Collagen in Odontogenesis

Collagen, one of the components of the connective tissue stroma, has been shown to play an important role in odontogenesis. When collagen synthesis and maturation is blocked or restricted by the use of collagenase or lathyrogens, tooth morphogenesis is inhibited, but if collagen synthesis is allowed to resume, dental development recommences (Kollar, 1972, 1973). The invasion of epithelium into underlying connective tissue, observed when dental epithelium is combined with non-dental connective tissue, also emphasizes that the collagenous framework has a possible role in determining tooth form. Kollar (1978) recently attempted to test the specificity of collagen

molecules. Initial experiments indicated that the addition of pro-collagen from dermis to inhibited tooth germs, resulted in limited tooth formation.

It is important to note that isolated enamel organ epithelium has the potential to produce collagen also, but the collagen so formed differs from that found in the dental papilla (Trelstadt & Slavkin, 1974). Several different species of collagen, acting together, may be involved in epithelial/mesenchymal interactions for odontogenesis.

Considerable evidence is now available to implicate collagen in these tissue interactions, but proteoglycans may also play an important role by interacting with collagen to modify morphogenesis (Toole & Linsenmayer, 1975). Furthermore, it appears that local alterations in the amount and type of glycosaminoglycans influence both migration and phenotypic expression of neural crest cells (Derby, 1978; Pintar, 1978).

CLINICAL APPLICATIONS AND FUTURE RESEARCH

The nature and distribution of morphogenetic fields within the dentition have clinical relevance as well as being important in aiding the



Figure 1 Coronal section through the jaws of a mouse embryo at an early stage of tooth development.
A—palate B—tongue C—mandible (tooth germ)

explanations for odontogenesis. The concept of morphogenetic fields corresponding to the various tooth classes within which the more distal teeth show less stability, whilst simple, provides a useful clinical rule of thumb. The incidence of agenesis or anomalous crown morphology is high in maxillary lateral incisors, second premolars and third molars. In the mandible the central incisor, second premolar and third molar appear most variable. However, this relatively simple pattern provides only a partial guide to the true nature of underlying morphogenetic fields. In the past, most odontometrical studies have concentrated on traditional measures of tooth size, such as the maximum mesiodistal and buccolingual dimensions. Multivariate methods that analyze the combinations of these dimensions provide additional insight into the morphological expression of dental fields. Furthermore, the use of experimental procedures to examine the occlusal areas or intercuspal distances on individual teeth, probably have greater

functional significance within the morphogenetic field hypothesis than the conventional tooth measurements now in use, and these methods would provide an interesting alternative approach to examining the morphogenetic field hypothesis.

Studies of dental asymmetry should also be expanded. The work of Sofaer et al. (1971b) suggesting that developing tooth germs have the ability to interact, depending on available space, has both evolutionary and clinical significance. Sofaer found that when a lateral incisor was missing on one side the central adjacent to the missing tooth was larger than its antimer. If a peg-shaped lateral was present, both central incisors were smaller than normal. In the former instance, Sofaer proposed that the compensatory increase in size of the adjacent central incisor possibly occurred initially in odontogenesis, but the presence of a peg lateral may have indicated that poor environmental conditions existed and this decreased the potential for the compensatory interaction with the central incisor

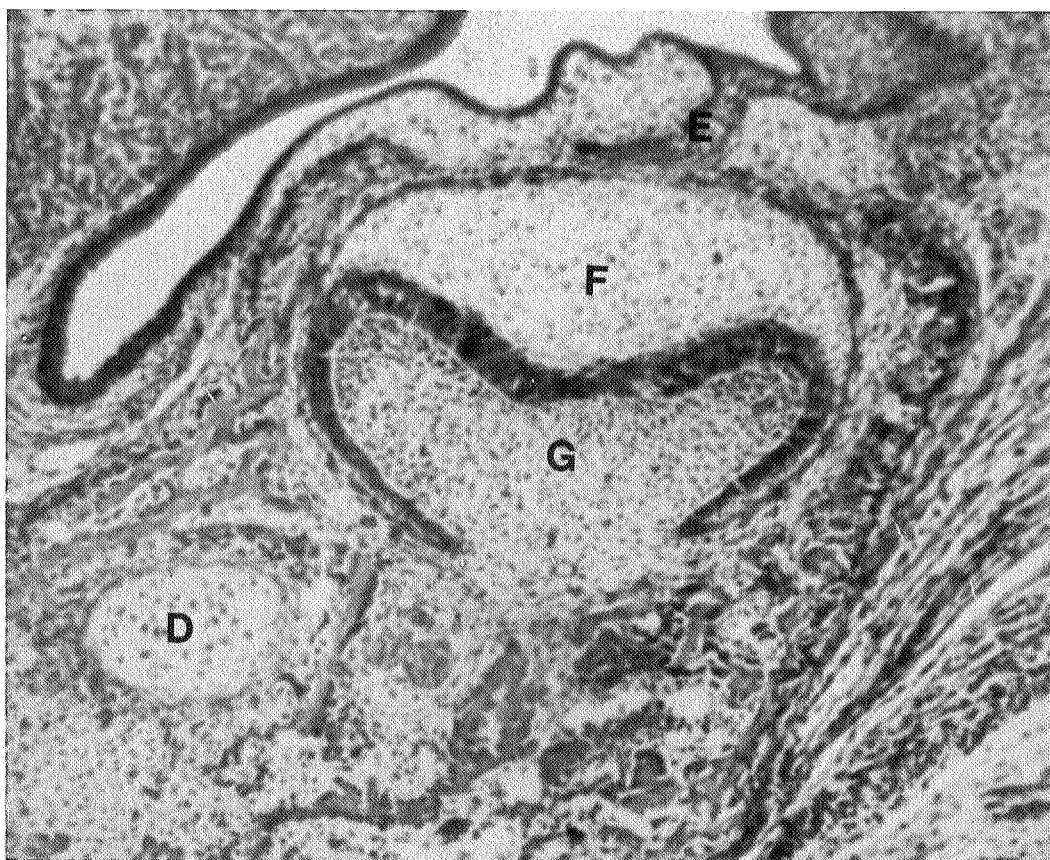


Figure 2 Enlarged photograph of developing mandibular tooth from the same specimen. D—Meckel's cartilage E—dental lamina F—epithelial enamel organ G—dental papilla

bud. The nature of the interaction between epithelium and connective tissue during tooth development is an intriguing problem. However, as Kollar (1975) pointed out, the factors involved in organizing the dentition into a functioning occlusal pattern are also important. In the early stages of odontogenesis, available evidence suggests that those migrating neural crest cells specifically destined to become the ecto-mesenchyme of the dental papilla, may play a central role. The specific roles of the blood supply and particularly the sensory innervation of the early mandibular branchial arch in determining the path of migration of these neural crest cells needs closer experimental scrutiny. There is no reason to believe that the theories of vesicle-bound RNA, of collagen and of proteoglycan involvement in odontogenesis are necessarily mutually exclusive. It is quite possible that the RNA excreted by cells of the dental papilla may direct subsequent collagen and proteoglycan synthesis. To account for the development of various tooth classes one must accept that there are differences either in the genetic makeup of the primordial cells or in the local environments of the various regions in which the teeth develop. An area of promising research would be the characterization of the extracellular matrix in different regions of the oral cavity to elucidate, for example, the nature of the proteoglycans. It may be that these molecules play a role in the so-called viscous drag concept of Osborn (1978).

A number of inherited diseases are associated with defects in the dentition. For example, ectodermal dysplasia results in missing teeth as well as small cone-shaped teeth. Although it is often thought that the condition may be due to an ectodermal defect, it is possible that connective tissue deficiencies in affected individuals may initiate the subsequent ectodermal defects.

Without doubt, the dentition provides an extremely valuable model system for studying the nature of morphogenetic fields. Clinical observations and detailed morphological studies, such as those summarized for the Australian Aboriginal dentition, are clearly suggestive of controlling mechanisms which determine tooth size, tooth morphology and the relationships between teeth in a class. However, purely descriptive studies are limited and they provide no indication of the relative importance of field or clone models in explaining odontogenesis. Experimental studies which explore the nature of cellular interactions in developing tissues are more likely to provide answers to the many aspects of tooth formation which await clarification.

REFERENCES

- ADAMS, M.S. & NISWANDER, J.D. (1967)
Developmental "noise" and a congenital malformation.
Genet. Res. Cambridge 10:313-317.
- ALVESALO, L. & TIGERSTEDT, P.M.A. (1974)
Heritabilities of human tooth dimensions.
Hereditas 77:311-318.
- BAILIT, H.L., WORKMAN, P.L., NISWANDER, J.D. & MACLEAN, C.J. (1970)
Dental asymmetry as an indicator of genetic and environmental conditions on human populations.
Hum. Biol. 42:626-638.
- BROWN, T., MARGETTS, B. & TOWNSEND, G.C. (1980a)
Comparison of mesiodistal crown diameters of the deciduous and permanent teeth in Australian Aborigines.
Aust. Dent. J. 25:28-33.
- BROWN, T., MARGETTS, B. & TOWNSEND, G.C. (1980b)
Correlations between crown diameters of the deciduous and permanent teeth of Australian Aborigines.
Aust. Dent. J. 25:219-223.
- BUTLER, P.M. (1939)
Studies of the mammalian dentition. Differentiation of the post-canine dentition.
Proc. Zool. Soc. Lond. 109, B:1-36.
- DAHLBERG, A.A. (1945)
The changing dentition of man.
J. Am. Dent. Ass. 32:676-690.
- DERBY, M.A. (1978)
Analysis of glycosaminoglycans within the extracellular environments encountered by migrating neural crest cells.
Dev. Biol. 66:321-336.
- GARN, S.M., LEWIS, A.B. & KERESKY, R.S. (1966)
The meaning of bilateral asymmetry in the permanent dentition.
Angle Orthodont. 36:55-62.
- GARN, S.M., LEWIS, A.B. & KERESKY, R.S. (1967)
Buccolingual size asymmetry and its developmental meaning.
Angle Orthodont. 37:186-193.
- GARN, S.M., COHEN, M.M. & GECIAUSKAS, M.A. (1970)
Increased crown-size asymmetry in Trisomy G.
J. Dent. Res. 49:465.
- HANIHARA, K. (1976)
Statistical and Comparative studies of the Australian Aboriginal Dentition.
The University Museum, The University of Tokyo, Bulletin No. 11.
- KOCH, W. (1967)
In vitro differentiation of tooth rudiments of embryonic mice.
1. Transfilter interaction of embryonic incisor tissues.
J. Exp. Zool. 165:155-170.
- KOLLAR, E.J. (1972)
Histogenetic aspects of dermal-epidermal interactions.
IN *Developmental Aspects of Oral Biology*.
Eds. Slavkin, H.C. & Bavetta, L.A.
Academic Press, New York and London.
- KOLLAR, E.J. (1973)
Effect of α, α' -dipyridyl on organ cultures of embryonic mouse tooth germs.
J. Dent. Res. 52:145 (Abstract).
- KOLLAR, E.J. (1975)
Gene-environment interactions during tooth development.
Dent. Clin. Nth. Am. 19:141-150.
- KOLLAR, E.J. (1978)
The role of collagen during tooth morphogenesis: some genetic implications. IN *Development, Function and Evolution of Teeth*.
Eds. Butler, P.M. & Joysey, K.A.
Academic Press London.
- LOMBARDI, A.V. (1975)
A factor analysis of morphogenetic fields in the human dentition.
Am. J. Phys. Anthropol. 42:99-104.
- LUNDSTRÖM, A. (1960)
Asymmetries in the number and size of the teeth and their aetiological significance.
Trans. Europ. Orthodont. Soc. 167-185.
- MARGETTS, B. & BROWN, T. (1978)
Crown diameters of the deciduous teeth in Australian Aborigines.
Am. J. Phys. Anthropol. 48:493-502.
- OSBORN, J.W. (1978)
Morphogenetic gradients: fields versus clones.
IN *Development, Function and Evolution of Teeth*.
Eds. Butler, P.M. & Joysey, K.A.
Academic Press, London.

- PINTAR, J.E. (1978)
Distribution and synthesis of glycosaminoglycans during quail neural crest morphogenesis.
Dev. Biol. 67:444-464.
- POTTER, R.H., YU, P.L., DAHLBERG, A.A., MERRITT, A.D. & CONNEALLY, P.M. (1968)
Genetic studies of tooth size factors in Pima Indian families.
Am. J. Hum. Genet. 20:89-100.
- SHAPIRO, B.L. (1970)
Prenatal dental anomalies in mongolism: comments on the basis and implications of variability.
Ann. N.Y. Acad. Sciences 171:562-577.
- SHAPIRO, B.L. (1975)
Amplified developmental instability in Down's syndrome.
Ann. Hum. Genet. 38:429-437.
- SLAVKIN, H.C. (1974)
Embryonic tooth formation. A tool for developmental biology.
Oral Sci. Rev. 4:1-136.
- Eds. Melcher, A.H. & Zarb, G.A.
Munksgaard, Copenhagen.
- SOFAER, J.A. (1979)
Human tooth-size asymmetry in cleft lip with or without cleft palate.
Arch. Oral Biol. 24:141-146.
- SOFAER, J.A., BAILIT, H.L. & MACLEAN, C.J. (1971a)
A developmental basis for differential tooth reduction during hominid evolution.
Evolution 25:509-517.
- SOFAER, J.A., CHUNG, C.S., NISWANDER, J.D. & RUNCK, D.W. (1971b)
Developmental interaction, size and agenesis among permanent maxillary incisors.
Hum. Biol. 43:36-45.
- SOFAER, J.A., MACLEAN, C.J. & BAILIT, H.L. (1972)
Heredity and morphological variation in early and late developing human teeth of the same morphological class.
Arch. Oral Biol. 17:811-816.
- TOOLE, B.P. & LINSSEN, T.F. (1975)
Proteoglycan-collagen interaction: possible developmental significance.
IN Extracellular Matrix Influences on Gene Expression.
Eds. Slavkin, H.C. & Greulich, R.C.
Academic Press, New York.
- TOWNSEND, G.C. (1980)
Heritability of deciduous tooth size in Australian Aborigines.
Am. J. Phys. Anthropol. 53:297-300.
- TOWNSEND, G.C. & BROWN, T. (1978a)
Inheritance of tooth size in Australian Aborigines.
Am. J. Phys. Anthropol. 48:305-314.
- TOWNSEND, G.C. & BROWN, T. (1978b)
Heritability of permanent tooth size.
Am. J. Phys. Anthropol. 49:497-504.
- TOWNSEND, G.C. & BROWN, T. (1979a)
Tooth size characteristics of Australian Aborigines.
Occasional papers in Human Biology. 1:17-38.
- TOWNSEND, G.C. & BROWN, T. (1979b)
Family studies of tooth size factors in the permanent dentition.
Am. J. Phys. Anthropol. 50:183-190.
- TOWNSEND, G.C. & BROWN, T. (1980)
Dental asymmetry in Australian Aborigines.
Hum. Biol. 52:661-673.
- TRELSTAD, R. & SLAVKIN, H.C. (1974)
Collagen synthesis by the epithelial enamel organ of the embryonic rabbit tooth.
Biochem biophys Res. Commun. 59:443-449.
- VAN VALEN, L. (1962)
A study of fluctuating asymmetry.
Evolution 16:125-142.
- WADDINGTON, C.H. (1957)
The Strategy of Genes.
George Allen & Unwin Ltd, London.

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Dr. G. C. Townsend,
University of Adelaide,
Department of Oral Biology,
G.P.O. Box, 498,
Adelaide, 5001, Australia

ORTHODONTIST

THE NORTHERN TERRITORY DENTAL HEALTH SERVICE requires an Orthodontist for a Specialist position based at the Alice Springs Dental Clinic.

The Alice Springs Dental Clinic has the additional staff of Dental Superintendent, three staff Dentists, two Dental Technicians and full laboratory facilities plus all ancillary personnel.

Duties include the diagnosis of case and/or treatment of patients referred and provision of specialist opinion on cases. Applicants must be eligible for registration as a Dentist in the Northern Territory and possess higher qualifications in Orthodontics including familiarity with the Begg Technique.

Alice Springs, a town of more than 15,000 people is geographically situated in the centre of Australia. It has its own unique charm and attraction and offers its residents ample educational, shopping, sporting and cultural facilities.

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