Effects of Dietary Consistency on Craniofacial and Occlusal Development in the Rat

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Moderate differences in the hardness of diet are related to significant differences in maxillary width and other measures of facial size. Probably even more important is a relationship to the coordination of growth of different parts of the dentofacial complex. Muscular stimulation mediated through occlusal function seems to play a significant role in the coordinated development of facial structures.

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MODERN DIET AND MALOCCLUSION

The prevalence of malocclusions in an urbanized society such as the United States is so high (50%[1]) that one is led to suspect the involvement of environmental factors in the etiology. Non-western, hunter-gatherer peoples are characterized by much lower frequencies of malocclusion.[2] However, the transition of peoples from rural or aboriginal to urban or industrial living has been rapidly followed by an increase in the incidence of malocclusion. This increase is found whether comparing medieval Europeans to recent ones[3,4] or non-western peoples to their immediate descendents living in a westernized environment.[2,5-15]

There is a strong genetic factor in the development of occlusion. Occlusal patterns within families have been demonstrated to be heritable to some degree,[16,17] and ethnic groups have been characterized by the frequencies of particular occlusal patterns.[18-21]

The prevalence of an occlusion that varies from the ideal is not the norm among mammals. Schultz[22] documents that dental crowding is quite rare in *primates,* and neither any of the specimens he presents nor any of the 400 wild primate dentitions that one of us (RSC) has examined would rate a Treatment Priority Index (TPI) score higher than 2.

The rapid increase in the incidence of malocclusion (often within one generation) gives cause to look beyond genetic explanations such as relaxed selection pressures, racial outcrossing, inbreeding, or accumulation of genetic mutations. Based on the available anthropological and experimental evidence, we believe that environmental factors, especially the physical consistency of the diet, affect the developing masticatory apparatus and create variations in the occlusal phenotype.

Experiments in effects of function

Apart from radical surgery, the experimental attempts to induce developmental change in the mammalian masticatory apparatus have used four methods: (1) forcing mouth breathing in animals; (2) placing devices in the mouth to effect alterations in mastication; (3) restricting animals to calorie-deprivation diets; and (4) varying the consistency of the diet from hard to soft.

Mouth breathing

Mouth breathing has been implicated experimentally (using macaques) in the development of open bite and of an abnormally narrow maxillary dental arch, attributed to altered muscle tension as the mandible is held slightly depressed during respiration.[23] Such effects are common in humans. Similar differences were also found in soft-diet rats, probably due to a reduction in muscle use rather than increased, abnormally-directed muscle tension. A common habit in childhood, mouth breathing is probably a factor in the development of human malocclusions.[24]

Disrupted occlusion

Several workers have applied stress to the masticatory apparatus or have altered occlusal relationships using appliances fastened to the teeth of rats or rhesus monkeys. These are usually designed to force protrusion of the mandible 1.5-2.0 mm in order to achieve molar occlusion.[25-27] A crown on the maxillary buccal teeth of rhesus monkeys was used by McNamara[27] to force the mandibular molars to occlude slightly lower than the normal occlusal surface of the uppers. Mandibular protrusion in young animals resulted in changes in the temporomandibular joint, specifically: (1) some posterior bone deposition; (2) bone resorption of the anterior edges of the glenoid fossa; (3) lengthened condylar neck; and (4) an increase in the angle formed by a line running from the condyle down the ramus with the line representing the occlusal plane.

The maxillae of these animals showed reduced vertical and prognathic growth. While the younger, growing animals responded to this stress by skeletal adaptations, older animals responded with tooth movements to adjust to the new occlusal alignments.

Using mature animals and a splint on the anterior teeth to force mandibular protrusion, Hiniker and Ramsfjord[25] also found dentitional changes as teeth were forced to move to maintain occlusal relationships. This movement resulted in short-term periodontal traumas which abated when

the teeth adapted and once again occluded adequately.

These studies demonstrate that forced realignments of occlusal relationships and mandibular movements lead to responses by the most flexible elements of the masticatory apparatus. In young animals, the growing skeletal system adjusts; in older animals, the dentition migrates to accommodate. While these experiments are important in assessing the response of the masticatory system to unusual mechanical stress, they do not create conditions likely to be encountered by the developing human masticatory system.

Nutrition

Experiments concerning the contribution of nutrition to normal occlusal development have taken the form of calorie deprivation of pigs. Tonge and McCance;[28,29] McCance, Owens and Tonge[30] used weanling animals, maintaining them for one year on a severe calorie-deficient diet, before attempting to "rehabilitate" some with proper foods. The authors found that their regime resulted in: (1) delayed dental development and eruption; and (2) "greater delay in the development and growth of the jaws."[29] The consequences were tooth crowding and abnormal tooth-tooth contacts. Rehabilitation was only partially successful. While calorie deprivation is undoubtedly a factor to consider in severely undernourished people, there is no evidence to correlate this deficiency with the occlusal problems found in so many urbanized peoples of the western world.

Dietary consistency

Although many workers have commented on the probable influence of the hardness or softness of the diet on the developing masticatory system, four studies have examined this idea experimentally. Watt and Williams,[31] Barber, Green, and Cox,[32] Moore,[33] and Henrikson, Sagne and Thilander[4] used weanling rats divided into populations eating either pelleted rat chow as a hard diet or crushed or water-softened chow as a soft diet. In all studies, the animals were maintained for approximately four months. When compared to the hard-diet rats, the soft-diet animals: (1) were slightly smaller in body mass; (2) exhibited no molar wear; (3) had mandibles that were smaller, with condyles smaller and radiographically less dense; (4) had less width of the maxillary dental arch; (5) had smaller masseter and temporalis muscles; and (6) had skulls consistently smaller in mass and in linear dimensions, although with no significant differences in shape.

The smaller masticatory muscles in soft-diet animals were almost certainly a result of lower work requirement in mastication. The smaller and less-active muscles applied less stress to the bones, which responded with less deposition and growth, especially in the mandible.

All mandibular structures related to chewing were affected by diet hardness. Wear of the molar, where tooth-food-tooth contact takes place, was less. Attachment areas for the jaw-closing muscles, where muscle force is transmitted to the mandible, were smaller. The condyles, which resist bite (especially incisal) reaction force, were smaller. Even buccolingual thickness of the mandible, which is essential to resist transverse bending, was less in the soft-diet animals.

More recently, Beecher and Corruccini[35] examined a small population of rhesus macaques which had spent a short period during adolescence on contrasting hard/soft diets. They

found significant narrowing of the maxillary arch with no lessening in length in the soft-diet monkeys. This correlated with the histological findings by Bouvier and Hylander[36] that significantly fewer secondary Haversian systems were present in the mandibular corpus of soft-diet monkeys than in the hard-diet monkeys of the same population.

While a number of measurements have been taken from animals in dietary consistency experiments, there has been no attempt to integrate these data in such a way that interactions between different parts of the growing masticatory systems could be measured. Further, with only two groups, hard and soft diet, it could not be determined whether the masticatory system responded in direct proportion to differences in dietary consistency, or whether no differences should be expected until a threshold in dietary consistency difference was reached, resulting in a quantum change in the morphology of the masticatory system. It was to fill these gaps in the experimental record that the following study was carried out.

Materials and Methods

The experimental animals were Sprague-Dawley rats, acquired at 21 days of age, evenly divided between males and females. Ninety animals were divided into three groups: Group I was fed pelletted rat chow (Purina Formulab); Group II was fed a gruel-like porridge consisting of ground chow moistened with water; Group III was fed the soft diet six days, with dry pellets provided every seventh day only.

We found the lab chow to be crumbly, easily breaking down into small granules. This indicates that the "hard" diet was minimally harder than the gruel, engendering only subtle differences in bite force. We have no way of knowing the consistency of rat diets used in earlier experiments of this type.

The three populations were maintained on their respective diets for four months, the same length of time used in previous experiments of this type. After sacrifice, the heads were randomly numbered so that the measurer would not be aware of their group membership. Measurements (Table 1) were taken as follows:

1. Body mass.
2. Fresh mass of the entire masseter.
3. Maxillary arch length, incisor to distal edge of last molar.
4. Maxillary arch breadth across buccal points of M1.
5. Mandibular length, incisor to M1.
6. Anteroposterior length of condylar articular surface.

Animals were also examined for visible differences in tooth alignments and attritional differences between the three groups.

A principal components analysis was performed on the data in order to discover the correlations among the several variables. This technique, related to factor analysis, finds the major axes of the between-variable correlation matrix. Least-squares theory is used to rotate orthogonal axes to a position where the major axis subsumes a maximized amount of the total variance. The second axis is the axis perpendicular to the first that subsumes the largest possible amount of residual variance, and so on.

The eigenvectors (also known as directional cosines or latent vectors) summarize the multiple interplay among variables. Projection of indi-

vidual cases onto the principal component axes allows intetrpretation of multivariate variability within samples, and functional interpretation of the morphological integration among variables. For instance, greater variation away from a major principal components axis describing general growth could be interpreted as meaning that there is poorer coordination of regional growth rates in those individuals.

Results

Maxillary breadth affected most

The basic descriptive statistics in Table 1 show the hard-diet population (Group I) to be larger in all dimensions and disproportionately so in some. In particular, the maxillary breadth is markedly increased in the hard-diet reared animals (Group I); this measurement alone shows a differentiation that is too large (relative to within-sample variation) to be reasonably ascribed to chance. The population reared on an intermittently hard diet (Group III) is generally intermediate in the size of structures, although more similar to the soft group.

Group I is more variable than Group II in every measurement, indicating a greater range of sizes. Again, Group III is intermediate in standard deviation, but usually closer to Group I. Group III is slightly more variable than Group I in the maxillary arch measurements, but not significantly so. The gross differences in the growth of most structures are not very marked, a result attributable to the small differences in dietary consistency.

Breakdown in growth coordination

A much more obvious feature of difference is manifested in the correlation structure between measurements within samples. Table 2 indicates the pairwise correlations in the hard and soft diet groups (I and II). All correlations are higher in the animals that had more masticatory resistance, and the differences are often significant. The strongest difference is in the growth relation of the masseter muscle, which is much more integrated with growth in the condyle and upper arch breadth in hard-diet than in soft-diet animals.

The comparison of correlation structure can be more easily accomplished through examination of the principal components of the correlation matrix. Table 3 lists key parameters of this analysis. The major (coordinated growth) axis is largest in the hard-diet sample, signifying that all structures are more tightly integrated in their growth and covariation. Inversely related to this, the second axis is largest among the soft-diet animals. Thus the residual from

TABLE 1
Mean, Dispersion, and Significance of Difference for Measured Traits

Variable	*Hard* *Mean (s.d.)*	*Soft* *Mean (s.d.)*	*Intermittent* *Mean (s.d.)*	*F*
Maxillary Length (mm) ..	24.00 (1.17)	23.79 (0.88)	23.71 (1.20)	0.57
Maxillary Breadth (mm) .	9.64 (0.38)	9.39 (0.26)	9.45 (0.40)	4.21*
Mandibular Length (mm)	8.92 (0.65)	8.89 (0.50)	8.91 (0.58)	0.01
Condyle Length (mm) ..	3.43 (0.41)	3.26 (0.36)	3.31 (0.39)	1.38
Body weight (gm)	43.72 (12.46)	38.03 (9.32)	39.43 (11.01)	2.18
Masseter weight (gm)	1.56 (0.48)	1.44 (0.28)	1.40 (0.40)	1.40

* 3-sample AoV with 2 and 87 d.f. significant at $p < .05$.

the major growth axis (which could be interpreted as error in growth or variation in growth rate) is easily largest in the soft diet sample; the F-ratio of variation on axis two (dispersion from axis one) between soft and hard samples is 2.19, which is significantly greater than expected by random error at $p = 2.5\%$.

This increased multivariate variability in soft-diet animals is all the more striking in view of the fact that they were less variable than the hard-food animals in every univariate trait. In all respects, the intermittent-hard-diet group (III) is intermediate when compared with Groups I and II but more similar to the hard-diet sample.

The correlations of variables with the axes show that the breakdown in growth coordination in soft-diet animals is especially marked in maxillary breadth, condyle length, and masseter weight. Masseter weight tends to be inversely correlated with maxillary breadth and condyle length on the second axis in soft animals. Condyle length alone shows a notable residual on the second axis in Group I and III.

Thus, at the stage of growth-differentiation represented by this difference in dietary consistency, a decrease in correlation of masticatory structures is more noteworthy than decrease in size of the structures that seems to follow with a stronger dietary differentiation. Perhaps a decrease in buffering among the growing components is the first step in their decrease in size. This phenomenon has also been noted in characters that are diminishing in size through natural selection.[37,38]

Attrition not affected

We examined the occlusal surfaces of the rat molars as a further test of the comparability of our hard chow and that used thirty years ago by Watt and Williams.[31] While they noted easily observable differences in attrition between soft and hard diet groups, we could find none. No caries were observed by us in any animals. We noticed no displaced or rotated teeth in any of the animals.

Discussion

Present-day insults to occlusion

Oppenheimer[39] and Lavell[40] document the better occlusion that existed in human populations prior to the onset of the industrial revolution. Only in the 19th and 20th centuries (it appears) has food become so processed that for practical purposes, most chewing stress has been removed and little bite force is now called into play in the jaws of the growing child.

TABLE 2
Product-moment Correlations Among Variables Within the Soft (lower triangular half of matrix) and Hard (upper triangular half) Dietary Groups

	Max. L	*Max. B*	*Mand. L*	*Cond.*	*Body*	*Masset.*
Maxillary Length		**0.83***	**0.89**	**0.75***	**0.93***	**0.91***
Maxillary Breadth	0.71		**0.73**	**0.66**	**0.83***	**0.79***
Mandibular Length	0.83	0.60		**0.58**	**0.85**	**0.78**
Condyle Length	0.54	0.55	0.52		**0.61**	**0.79***
Body weight	0.74	0.60	0.78	0.55		**0.90***
Masseter weight	0.72	0.39	0.71	0.33	0.80	

Hard diet

Soft diet

* Significantly larger r in hard diet sample at $p < .05$.

A number of other environmental factors have been suggested as influences on developing occlusion, including mouth breathing, premature deciduous tooth loss, hormonal intervention, endocrine disturbance, trauma, oral habits, posture, disturbance of synchronous development in separately growing parts, prenatal insult, and independence of tooth formation.[41,42] Some of these may be of minimal or no importance; experimental evidence which might weight such factors is absent. Because of its widespread occurrence in western society, the reduction of chewing resistance and related growth stimulation provided by "civilized" man's refined diet may be the strongest causal factor in developmental malocclusions.

Though the chewing explanation is most predictive of observed occlusal differences in the rats, it clearly is not favored by many orthodontists as an explanation of the rising rate of occlusal variation in humans.[42-45]

The growth independence of mandible and maxilla in humans has been frequently noted. In particular, the mandible may depend on muscular function to grow to average size, as shown by the tendency of some individuals with anodontia to have average-size mandibles.[46] Maxillary growth may be under closer genetic programming, since cases of anodontia show restricted growth while cases with muscular paralysis may develop normally.[46] Garn[42] lists breakdown of synchronous growth in lower and upper jaws as a possible cause of malocclusion, but does not give the source of the growth-correlation breakdown. Potter *et al.*[47] demonstrate that upper and lower dental arches are under very different sorts of genetic control, with the maxillary teeth being more conservative and controlled by a fewer number of genes.

The aging process also yields different results in upper and lower arches. Tooth spacing and crowding, especially of anterior teeth, follows different rates and peaks at different ages in the maxilla and mandible.[2,51] Lower correlation between upper and lower teeth in maloccluded individuals (as compared with normal) has been noted by Lavelle.[48] Moorrees and Reed[49] also stress the importance of high correlation between tooth and arch size to the development of good occlusion. Thus, considerable evidence ties upper-lower correlation diminution with development of mal-

TABLE 3
EigenValues (latent roots) and Eigenvectors (latent vectors) of the Correlation Matrices in Three Dietary Groups

	Principal Component One			*Principal Component Two*		
	(coordinated growth)			*(variation in growth)*		
	Hard	*Soft*	*Intermittent*	*Hard*	*Soft*	*Intermittent*
EigenValue	4.96	4.17	4.67	0.50	0.79	0.60
(% variance)	(83)	(69)	(78)	(8)	(13)	(10)
Eigenvector correlations:						
Maxillary Length	0.97	0.92	0.96	−0.05	−0.04	−0.08
Maxillary Breadth	0.89	0.77	0.82	−0.06	0.41	−0.06
Mandibular Length	0.89	0.90	0.91	−0.29	−0.12	−0.14
Condyle Length	0.80	0.68	0.68	0.59	0.57	0.73
Body weight	0.94	0.90	0.93	−0.23	−0.16	−0.18
Masseter weight	0.95	0.80	0.95	0.10	−0.51	−0.08

occlusion in humans. The prevailing explanation for such lowering of growth correspondence has been the effect of racial admixture between populations.[14,48] Thus, the inheritance of different-sized parts that must occlude is attributed to the independent segregation of disharmonious genes from disparate ancestors. Chung *et al.*[50] and Lombardi and Bailit[2] present convincing genetic arguments against this explanation.

Conclusion

We believe our results suggest future directions for fruitful research based on the following observations. The amount of maxillary arch narrowing and collapse can be predicted by the amount of time the animals were chewing on hard food. Mediolateral maxillary growth seems dependent upon the muscular stimulation provided by rough elements in the diet. In this animal population the disassociation in size of occlusal features during their growth, previously noted in certain groups of malocclueded humans and attributed to racial admixture, has apparently resulted from lower chewing stress required by a soft dietary consistency.

Replication of these experiments with a laboratory animal more closely related to man in terms of both biology and masticatory system is seen as a vital future need. The unfused mandibular symphysis in the rat alters force distribution, and continual incisor function in rodents maintains a base level of function regardless of diet that has no parallel in man.

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