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Human Dental Reduction: Natural Selection or the Probable Mutation Effect

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ABSTRACT Dental reduction has been sufficiently widespread among human populations to render the phenomenon of reduced tooth size worthy of scientific explanation. One of the most controversial models invoked to explain structural reduction in organisms is referred to as the "probable mutation effect" (PME). According to this model, structures no longer functional owing to ecological or cultural changes will experience a relaxation of selection pressure, permitting an accumulation of mutations in the population that inevitably will result in the reduction in size or the loss of the concerned structure. Although the PME continues to be offered as a viable explanation of human dental reduction, it is based upon several premises that modern dental clinical experience fails to support. Known enzyme defects resulting from mutations, factors predisposing to dental infections, and the deleterious effects of teeth that are too large or too small reveal that the PME does not logically account for the reduction of tooth size. Given such information, this paper proposes models of dental reduction based upon natural selection, which, unlike the PME, are testable in both modern and archaeological populations. The integration of clinical and skeletal data permits a more thorough understanding of dental reduction in the hominid fossil record.

Over the past 40,000 years, human populations worldwide have experienced a reduction in tooth size. Data documenting this trend are now available from the continents of Africa (Calcagno, 1984, 1986; Greene, 1972; Smith, 1979), Europe (Brabant, 1967; Brabant and Twiesselmann, 1964; Brabant, 1971; Brace, 1979; Formicola, 1987; Frayer, 1977, 1978, 1980, 1984; LeBlanc and Black, 1974; y'Edynak, 1978; y'Edynak and Fleisch, 1983), Asia (Brace, 1978; Brace and Hinton, 1981; Brace and Nagai, 1982; Brace et al., 1984; Suzuki, 1969), Australia (Brace, 1980a,b) and North America (Hinton et al., 1980; Larsen, 1981; Sciulli, 1979; Smith et al., 1980). As a result, the phenomenon of dental reduction in *Homo sapiens sapiens* has provoked considerable commentary (e.g., Anderson and Popovitch, 1977; Bailit and Friedlaender, 1966; Brace, 1967; Brace and Mahler, 1971; Brace and Ryan, 1980; Brace et al., 1987; Brose and Wolpoff, 1971; Dahlberg, 1963; Garn et al., 1969; Macchiarelli and Bondioli, 1986; Smith, 1977a,b, 1982; Wolpoff, 1971, 1975; and others).

As is generally the case, documenting a trend is easier than accounting for the causal factors responsible for it. In the view of C. Loring Brace (1963, 1964), human dental reduction represents a cardinal example of a specific evolutionary mechanism, which he termed the probable mutation effect (PME). According to the PME model, structures that are no longer functional experience a relaxation in selection pressure. This permits mutations to accumulate in the population, with the result that the concerned structures reduce in size. The PME model has been applied to cases of structural reduction ranging

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from the loss of eyesight and pigmentation in cave fish (Brace and Montagu, 1965) to the loss of tails by anthropoid apes (Brace, 1963), and in Brace's view "the best illustration of structural reduction in man is in the face" (Brace, 1963:44). Simply stated, as tools and techniques of food preparation become increasingly sophisticated, large teeth are no longer needed. Hence, random mutations are free to accumulate, and "since the majority of such mutations will result in structural reduction" (Brace, 1963:44), the "probable effect" is decreased tooth size.

Even when first articulated, the PME model was based on questionable genetic and functional anatomical arguments (Bailit and Friedlaender, 1966; Brues, 1966, 1968; Byles, 1972; Holloway, 1966; Prout, 1964; Williams, 1978; Wright, 1964). Nevertheless, the model remains very much alive, as witnessed by a recent article (McKee, 1984) purporting to demonstrate mathematically that in the absence of natural selection, structures under multifactorial genetic control and subject to relatively high mutation rates will be expected to decrease in size over a period of 40,000 years.

Like other proponents of the PME model, however, McKee ignored critical genetic, clinical, and bioarchaeological data of relevance to the potential effects of dental mutations and to the meaning of tooth size. These data indicate that the PME model of reduced dentition is both unnecessary and untenable. Instead, the present paper suggests that teeth may have reduced in size by direct selection for smaller teeth. In addition, unlike the PME model, the selectionist model is directly testable in both modern and archaeological populations.

THE PME MODEL: GENETIC ASSUMPTIONS AND FALLACIES

The models of both Brace and McKee rest upon a number of genetic assumptions that appear invalid. McKee's model, for instance, demands very high rates of mutation of 2×10^{-4} to 2×10^{-5} per generation. Although the average mutation rate for the human genome is far from totally agreed upon, most geneticists would consider these figures to be exceptionally high. For example, Stevenson and Kerr (1967), Cavalli-Sforza and Bodmer (1971), and Nute and Stamatoyannopoulos (1984) report 1×10^{-6} as the best estimate of the average mutation rate for humans. The fact that many known mutation estimates, upon which McKee derives his figures, are often greater than this average may relate to the focusing upon associated disorders because of their greater medical attention and impact on society. When attempting to calculate mutation rates, disorders that are relatively frequent (having rates as high as those estimated by McKee) are selectively studied because they produce a sufficient number of cases to form the basis for reliable mutation estimates given the population size (Vogel and Motulsky, 1986). Thus, one cannot expect the average mutation rate to approach the rate of the most common genetic disorders. Indeed, it is likely that the median mutation rate, perhaps a better measure since the distribution of mutation rates is drastically skewed toward lower rates, is another magnitude smaller (1×10^{-7}) than the estimated mean, according to Cavalli-Sforza and Bodmer (1971:105).

If a mutation rate of 1×10^{-6} (20 to 200 times smaller) is substituted for the range of estimates McKee presents in Table 2 (1984:239), it becomes evident that more than 25 loci controlling tooth size would be necessary to account for phenotypic reduction throughout 90% of the population. If the median figure of 1×10^{-7} is used, the number of loci (acting solely upon tooth size) needed is further increased and even more unimaginable. It has been suggested that only three to seven independent genetic factors control tooth size (Lombardi, 1975; Potter et al., 1976).

In other words, what McKee has demonstrated is that if unknown mutation rates for reduced tooth size greatly exceed average mutation rates for other human genes and if the numbers of genes controlling tooth size greatly exceed those estimated by students of the subject, then PME could lead to reduced tooth size. It would seem, then, that rather than demonstrating the probability of the probable mutation effect, as he assumed, McKee actually demonstrated its improbability.

Other problems also exist with the genetic assumptions of the PME model. As initially proposed, Brace's model was based, in part, on a misinterpretation of Sewall Wright's concept of mutation pressure. Wright proposed that in the absence of selection, recurrent mutations may effect changes in gene frequencies within a population (Wright, 1929, 1931, 1964). Thus defined, the term mutation pressure did not refer to the "probable effect of mutations on characters" (Wright, 1964). Rather, Wright's explanation of structural reduction was placed within the context of the pleiotropic effects of genes, such effects being "practically universal;" conse-

quently "the momentary selective advantage of a gene over an allele is the resultant of components, positive or negative, from effects on numerous characters. Direction of change of gene frequencies is controlled almost wholly by the more important of these components." Hence, the reduction of structures "is due largely to selection for pleiotropic effects of newly favored alleles of the genes that had been involved in the development of the organ in question" (Wright, 1964: 66).

By contrast to Wright's model of near universal pleiotropy, the models of both Brace and McKee are based on concepts of no pleiotropy: "If a gene can be postulated which specifically affects only one character . . ." (Brace, 1964:453) or "None of the mutations in this polygenic system may have pleiotropic effects which would significantly alter relative fitness . . ." (McKee, 1984:233). Hence, if Wright's assumption of near universal pleiotropy is correct, then the PME model is flawed at its base.[1]

Wright did state in other contexts that mutations "tend to reduce structures," and it is accepted among geneticists that most mutations are deleterious (Wright, 1964). These concepts of structural reduction formed a second major component of Brace's model. Problems arise, however, in extending them to human evolution. Wright's observations were based on studies of *Drosophila* using the scientific technology of the 1920s and 1930s. At the time he worked, the sciences of human medical and dental genetics were in their infancy. In the intervening six decades, much has been learned about human genes. As of 1988, very few human genes are known that reduce or eliminate anatomical organs such as teeth or limbs. In humans, most spontaneously appearing organ reductions are traced to teratogenic effects. By contrast, hundreds, perhaps thousands, of genes are known whose phenotypic effects are more subtle and/or metabolic in nature. Given this modern knowledge, the concept that most human mutations tend to reduce structures would appear untenable, at least if structures are interpreted to mean anatomical organs.

Brace's concept of structural reduction was based on the fact that mutations often result in missing or defective enzymes. Defective enzyme function may manifest itself in several ways, one of which is the reduction or simplification of the morphological end product of enzyme-catalyzed reactions. If anatomical structures are the end products of such reactions, then it is logical to assume, as Brace did, that reduced enzyme function will lead to structural loss. Biochemical and clinical evidence, however, indicates that even when expressed in this more sophisticated manner, the concept is flawed. Enzymes do not usually code for the production of entire organs such as eyes, tails, or teeth, but rather for particular proteins, lipids, or other biochemical substances. As few, if any, anatomical organs are composed of a single biochemical substance, defects in single enzymes are more likely to result in defective organ structure than in diminution in organ size. For an organ to reduce in size while maintaining normal form, all involved enzymes would have to produce end products that are normal in structure and function but reduced in quantity.

An individual tooth, for instance, is not composed of a single protein, lipid, or other biochemical structure. Rather, it is composed of three main tissues: enamel, dentin, and cementum, each of which, in turn, has a complex biochemical matrix. Hence, developmental interactions of numerous enzyme-mediated processes form the basis of normal tooth formation. The loss of a single enzyme would selectively effect single biochemical components of each tooth and be expected to result in defective structure of specific dental components, such as enamel, rather than in an overall size decrement. Reduced size, coupled with normal structure, would require quantitative reductions in all tooth-related enzyme systems. As, however, some of the enzymes involved in the genesis of dental structures, such as alkaline phosphatase, are also required for the production of bone and other body structures, a generalized decrement in all enzymes affecting tooth structure would also affect other body parts. In this case, a basic assumption of the PME model, no pleiotropy, would be invalidated.

Actually, however, it is unnecessary to argue in the abstract about what mutant genes affecting dental structures might do. Many mutant genes are known from clinical experience. As Wright expected, based on the concept of near universal pleiotropy, many genetically mediated dental problems result from genes with multiple negative pheno-

[1]Wright's assumption may be invalid, for if pleiotropy is near universal within the organism, it becomes likely for any given mutation to be disadvantageous for most traits within the pleiotropic system. Wolpoff (1969) has stressed that this could only rarely lead to adaptive change in a particular trait, and thus the occurrence of evolution would be drastically restricted. However, most known human mutations are, in fact, pleiotropic. In either case (regardless of pleiotropic effects), the PME remains indefensible for reasons presented throughout this paper.

typic manifestations. Several hundred such genetic defects are known. Some genetic defects, however, do affect dental structures alone with few, if any, pleiotropic effects. These include genes producing at least eleven different forms of amelogenesis imperfecta (defective enamel), at least two forms of dentinogenesis imperfecta (defective dentin formation), and at least two forms of dentin dysplasias (defective dental roots). In addition, several single-gene-mediated enzyme deficiencies are known that affect the teeth indirectly through deleterious effects on the periodontium or gingiva. Hypophosphatasia, for instance, results in teeth of relatively normal form that are lost prematurely owing to presumed defects in the periodontal-cementum interface. Similarly, a deficiency of the enzyme catalase, acatalasia, results in severe periodontal disease with frequent tooth loss (Stewart and Prescott, 1976).

Although the genetic mechanisms that reduce tooth size without producing deleterious effects on teeth or other body structures are largely unknown, clues of their possible nature can be gained by examining developmental data. Such data indicate that tooth size is determined by the size of the developing tooth germ and by the amount of dentin and enamel matrix secreted by odontoblasts and ameloblasts, respectively (Martin and Boyd, 1984; Ten Cate, 1985). The size of the tooth germ is determined, in turn, by the number of mitotic cell divisions involved in the formation of the germ. Hence, tooth germ size reflects the rates of mitoses and the length of the mitotic period. Similarly, the amount of enamel and dentin matrix secreted reflects the rates of secretion and the length of the secretory period (Martin and Boyd, 1984). These considerations suggest that variations in tooth size reflect changes in developmental regulatory processes rather than in enzyme systems, as suggested two decades ago by Holloway (1966). Possibly, these changes in the developmental cycle result from changes in regulatory genes affecting the tooth buds themselves. Alternately, it is possible that the developing tooth germs compete for space within the mandible and may inhibit each other's growth when space is limited (Sofaer, 1969a,b, 1973; Sofaer et al., 1971a,b; Townsend and Brown, 1983). Hence, tooth size variations may be, in part, indirect secondary effects of genes or other developmental effects regulating fetal and infantile jaw size rather than direct effects of genes affecting the dental complex itself.

The genetic control of congenitally missing teeth, like that of tooth size, remains uncertain. Some have hypothesized that missing lateral incisors and missing third molars are controlled by dominant genes (Brown, 1983). It is known, however, that tooth germs that fail to reach a specific size may degenerate. Based on this information, others have suggested that the same genetic mechanisms that result in reduced tooth size are also responsible for congenitally missing teeth (Chosach et al., 1975; Grunberg, 1963; Suarez, 1974; Woolf, 1971).

The realization that decreased tooth size and/or dental agenesis probably results from altered developmental timing and/or from space competition within a smaller masticatory apparatus places the probable mutation model within a different light. Some might argue that decreases in the rates or lengths of the developmental processes are themselves the products of deleterious mutations resulting in the loss of regulatory gene end products. There is not evidence of this, however. Moreover, hypodontia occurs in Trisomy 21 (Townsend, 1983), clearly indicating that a reduced dentition can result from increased numbers of genes and gene interactions, rather than from loss of functional genes.

Alternatively, it might be argued that as tooth size is sometimes developmentally labile with respect to jaw size, genetic changes are not involved in dental reduction at all. Rather, teeth have reduced in size as a secondary consequence of environmental changes leading to reduced masticatory development. The high prevalence of dental crowding and malocclusion in modern societies contradicts this latter interpretation and clearly indicates that genetic, as well as developmental processes, are involved in the inheritance of tooth size. Moreover, tooth size is thought to be under more strict genetic control than jaw size (Greene, 1967; Kraus, 1957, 1962; Kraus and Jordan, 1965; Kraus et al., 1959; Krogman, 1967; Lasker, 1950; Moorress, 1962; Osborne, 1962, 1963; Osborne et al., 1958).

In addition, the PME can be used to explain tooth size, only if it is assumed that mutant genes controlling tooth development are unique among genes controlling developmental mechanisms in that they only mutate in the direction of reduced developmental rates and/or reduced developmental time frames. If, by contrast, genes regulating tooth development, like those in-

fluencing the development of other body parts, can mutate in the direction of increased rates or lengths of development, then there is no reason to assume that mutations will necessarily lead to reduction. Rather, mutations could cause increases, decreases, or no change in tooth size (Prout, 1964).

These data suggest that the genetic explanation for dental reduction must lie in something other than a random accumulation of mutant genes. If, as presented in the PME model, selection no longer acts to maintain teeth of a given size and structure (thus creating a vestigial, useless dentition), then mutants resulting in congenitally missing teeth, defective tooth structure, premature dental loss and even increased size would be as likely to accumulate in the population as mutants resulting in reduced tooth size. The evolutionary picture would be one of massive accumulation of dental abnormalities and progressive loss of a functional dentition, rather than of steady reductions in the size of otherwise normal teeth. Instead, dental reduction represents an increased incidence of a specific class of genes, those that influence dental size while maintaining normal structure and that have no deleterious pleiotropic effects on other organs. This suggests that teeth never became functionless or vestigial, rather there has been continuing selection for a fully functional, albeit smaller, dentition. That teeth have become progressively smaller in size rather than more variable in size is indicative of positive selection for smaller teeth, not selective neutrality of dental size. Reduction in tooth size becomes, then, a classic example of directional selection.

NATURAL SELECTION AND DENTAL REDUCTION: SOME PROBABLE MECHANISMS

Teeth that are small relative to environmental demand may be worn to the pulp by the age of forty or fifty years in humans. Such teeth not only exhibit reduced function, but are subject to loss through infections and abscesses as well. In many wild animals, loss of dental function will result in a loss of feeding efficiency or ability. A similar phenomenon may have characterized much of human evolution, especially in populations subsisting on hard and/or abrasive diets. That wear can act as a selective agent for large teeth under such circumstances is sufficiently intuitive that, to our knowledge, no one has ever postulated a specific evolutionary mechanism to account for dental enlargement.

That small teeth can also be selected for or, alternatively, that large teeth can be selected against, is apparently less intuitive; hence, the continuing popularity of the PME model. Clinical evidence, however, indicates that large teeth may have deleterious, even life-threatening effects on human health. Large teeth, for instance, are prone to many conditions that actually reduce, rather than enhance dental function. For example, impacted teeth may exert pressure upon the roots of adjacent teeth, thereby resulting in root resorption and loss. In addition, large teeth are more susceptible to dental caries than small teeth, in part, because large teeth have more pits and fissures in which caries tend to form (Anderson and Popovitch, 1977; Dirks, 1965; Grainger et al., 1966; Greene, 1970, 1972; Hanke, 1933; Hunter, 1967; Kamp et al., 1983; Keene, 1964, 1965, 1967, 1971; Klatsky and Fisher, 1953; Paynter and Grainger, 1961, 1962; Van Reenen, 1966). Even when teeth are not absolutely large, but merely relatively large by comparison to jaw size, susceptibility to caries is increased because relatively large teeth lead to dental crowding, malpositioned teeth, and/or impacted teeth. These often abut against each other in a manner that produces retentive areas favoring the accumulation of caries-causing bacteria. Before the advent of modern restorative dentistry of the last 150 years, severely carious teeth were rendered nonfunctional, thus representing one of several means whereby large teeth could lead to tooth loss.

Hence, paradoxically, large teeth may result in less functional dental surface than small teeth. If, however, reduced dental function were the only negative consequence of large teeth and, as Brace hypothesizes, if teeth have become functionless, then premature tooth loss would have no selective consequences.

However, modern clinical experience indicates that dental caries, dental crowding, dental impaction, and periodontal disease can all have life-threatening consequences. In particular, each can lead to abscesses and infections. Dental infections can, in turn, lead to gangrene, septicemia or osteomyelitis. Infections of the maxillary teeth or oral tissues can also readily pass into the cranial cavity via the facial vein or the internal pterygoid plexus of veins. Once there they can produce cavernous sinus thrombosis or meningitis. Mandibular infections (see Fig. 1A–C) can result in swellings of the throat and

submandibular region (Ludwig's angina), which may lead to asphyxiation, endocarditis, or pneumonia (Mead, 1933; Thoma, 1948).

While the actual incidence of these potentially fatal consequences of dental infection is not known on a population-wide basis, clinical experience indicates that it is not inappreciable. The Oral Surgery Department at the University of Texas Dental Branch at Houston, for instance, currently treats an average of four to five patients a week who have been admitted to university-serviced hospitals for severe infections of dental origin that have proven resistant to outpatient treatment with antibiotics (McFarland, personal communication).

Although the lives of nearly all of such patients can be saved with aggressive in-hospital surgical and medical treatment, this has not always been the case. As recently as the 1940s, for instance, it was not uncommon for patients to be admitted to hospitals in the United States with meningitis and cavernous sinus thrombosis subsequent to dental infection. Even with neurosurgical drainage, the death rate from cavernous sinus thrombosis was estimated at 50% to 90% in some hospitals (Childs and Courville, 1942; Dixon, 1929; Haymaker, 1945; Koepf et al., 1937; Stout, 1931). Similarly, the death rate from Ludwig's angina was estimated as approximately 40% to 50% in one series, mostly as a result of asphyxiation or secondary pneumonia (Thoma, 1948). Malnourished or debilitated patients generally exhibit increased susceptibility to infections, and it has been suggested that in the 1930s and 1940s the incidence of oral gangrene subsequent to dental infections was considerably higher in Asia than in the United States and may have been a fairly common cause of death among young children (Sung and Sung, 1947; Thoma, 1948).

Hence, it is clear from clinical data that teeth that are too large and those that are too small can both yield deleterious, even life-threatening, consequences. According to McKee (1984:240), "It is up to the selectionist to demonstrate that any such functional differences (associated with reduced tooth size) could be advantageous within a given environment and would be subject to natural selection." In the face of the evidence cited above, however, it would seem that the burden of proof lies with those who would claim that tooth size is selectively neutral and/or who postulate that natural selection does not act in favor of reduced tooth size.

BIOARCHAEOLOGICAL IMPLICATIONS OF SELECTIONIST MODELS OF DENTAL REDUCTION

Based on the clinical data cited above, it is clear that there is no optimum tooth size that would suffice for all populations. Rather, tooth size must be viewed in relationship to other factors, such as diet and size of the jaws.

Hard or abrasive diets, for instance, will produce more rapid wear than softer diets. Consequently, teeth that are sufficient in size to last an entire life span under some dietary conditions may evidence wear to the gum line by early middle age in others. Similarly, the cusp/groove configuration that renders the occlusal surface of large teeth, particularly large molar teeth, susceptible to caries may be completely obliterated by the teenage years in populations that experience heavy wear. However, even when the diet is sufficiently soft to permit the cusp/groove pattern to remain into the middle years, tooth decay is generally not a serious threat unless the diet is also cariogenic.

Problems such as dental crowding and impaction result not so much from teeth that are absolutely large as from those that are relatively large by comparison to the jaw size. While genetic factors may be involved in producing these discrepancies, it is also clear that environment plays a prominent role. Under conditions of heavy wear, for instance, the teeth will shorten in the mesiodistal direction subsequent to eruption, thereby preventing dental crowding and permitting a mesial migration of the teeth that will result in ample posterior space for the eruption of the third molars (Begg, 1954; Mucci, 1982).

The maxilla and mandible, like other bones, grow, in part, in response to muscular activity and can be modified by changes in such activity and/or in the resting position of the mandible (Avis, 1959, 1961; Barber et al., 1963; Ghafari and Heeley, 1982; Harvold, 1975; Harvold et al., 1973; Hinton, 1983; Hoyte and Enlow, 1966; Hunt, 1960; McNamara, 1975; Oppenheimer, 1964; Pratt, 1943; Scott, 1957; Washburn, 1947; Watt and Williams, 1951). Thus, whether drastically tested by surgically removing one of the masseter muscles from rats (Pratt, 1943) or simply by placing rats or monkeys on hard versus soft diets (Beecher and Corrucini, 1981; Corrucini and Beecher, 1982; Watt and Williams, 1951), bone resorption occurs under reduced demand and bone apposition results from periods of increased stress. Indeed,

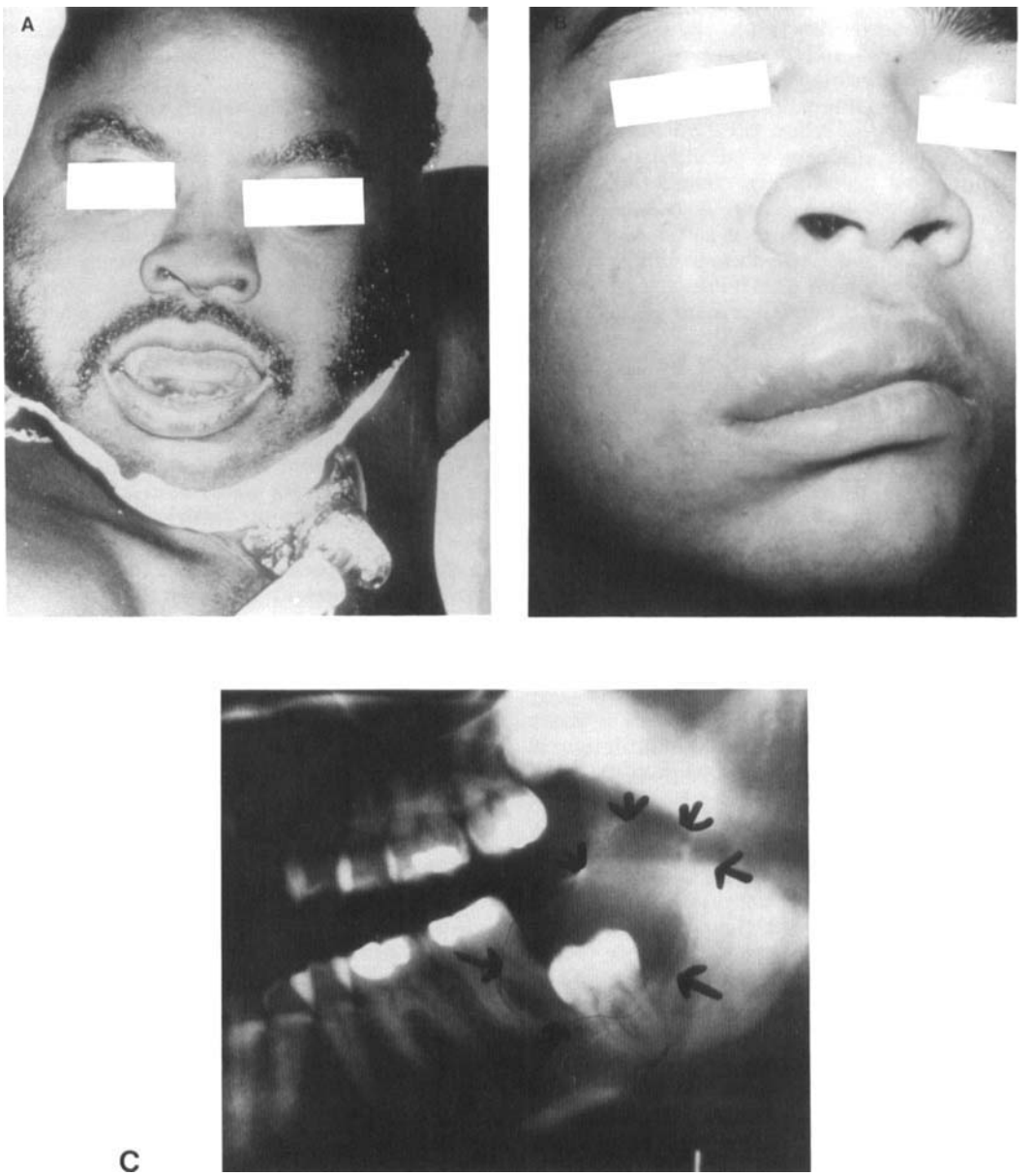

Fig. 1. Abscesses and infections stemming from factors such as dental caries, dental crowding, dental impacting, and periodontal disease can have life-threatening consequences. For example, in **A**, Ludwig's angina (swellings of the throat and mandibular region) can result from mandibular infections. Patients may be placed on artificial respiration systems to avoid blockage of the airway through asphyxiation. In **B**, infections such as cellulitis, after creating a large swelling of the face, can pass from facial region into the mediastinum and produce bacterial endocarditis. Such patients are routinely given prophylactic antibiotics, without which the infection can be fatal. As seen in **C**, it is not uncommon for dentigerous cysts to develop around impacted third molars. If surgically removed, these cysts progressively enlarge, resulting in weakening of the bony substance of the jaws and rendering them susceptible to fracture under minor stress. Such harmful and often life-threatening situations suggest that continuing positive selection for a fully functional, smaller dentition, rather than a continual build-up of random mutations, best accounts for dental reduction over time.

this interrelationship between bone growth and increased functional demand is not unique to jaws, but is in accord with Wolff's law of transformation (Wolff, 1892), which concerns the general response of bone to mechanical forces. When the two environmentally labile processes, dental wear and masticatory growth, are considered in conjunction with each other, it becomes clear that a simple change to a softer diet in the absence of any genetic change can produce a situation in which the teeth are relatively too large for the jaws (Corrucini and Whitley, 1981; Corrucini et al., 1983).

With these considerations in mind, two contrasting selective conditions can be envisioned. Under conditions of a hard diet producing heavy attrition and demanding heavy masticatory muscle activity, the maxilla and mandible would grow to their optimum size, thus providing ample room for large teeth. At the same time, early wear would result in the loss of the cusp/groove caries-susceptible anatomy and in mesiodistal shortening of the teeth. Neither dental crowding nor coronal caries would play a major selective role.

In such populations, however, teeth would be worn to a nonfunctional state by the early middle years, thereby resulting in a shortened life span. The selective challenge in these conditions would be to produce sufficiently large teeth that do not wear to the pulp before the individual has a chance to rear several viable offspring.

In populations subsisting on a soft diet, conditions would be very different. Cusps would remain unworn throughout much of the life span thereby predisposing to caries. Lack of interproximal wear would result in the loss of mesial drift. Lack of proper bone/muscle stimulation would result in defective growth of the jaws. Hence, a predisposition would exist for dental crowding and impaction.

The selective challenge in these conditions would be to avoid premature loss of teeth owing to caries, crowding, and impactions, as well as to avoid potentially life-threatening infections secondary to these conditions. Unlike the excess wear characteristic of teeth too small to meet environmentally imposed demands, the negative effects of big teeth could well make themselves felt in childhood and adolescence. Selection would work in the direction of smaller teeth and dental agenesis. Third molars would be under the heaviest negative selection pressure because, being the last to erupt, they are the most frequently impacted and because the absence of third molars frees additional space for the remaining teeth without creating developmental gaps between teeth.

In reality, of course, many populations probably were subject to selective pressures slightly different from either of these extremes. For example, both dental crowding and excessive dental wear were already present in australopithecines, suggesting that at least in some early hominid groups the demands for large teeth in later life stages exceeded the capacity of the jaws to accommodate them. It is inappropriate to treat the dentition as a single unit, assuming that all teeth need to be large to counteract heavy wear or small to fit into diminishing jaws. Both *Homo erectus* and Neandertals exhibit decreasing posterior tooth metrics in combination with increasing anterior tooth size. In each case, possible selective advantages for enlarging posterior teeth in australopithecines and anterior teeth in *Homo erectus* and Neandertals are rather casually and confidently offered, while advantages for the reduction in their counterparts often are not acknowledged. It is as though teeth that do not have to be larger are destined to reduce in size for no selective reasons, despite that, as previously stated, teeth too small to meet environmental demand present severe liabilities to human health.

NUBIA: A POSSIBLE EXAMPLE OF SELECTION IN ACTION

Because the dietary factors affecting tooth and jaw size will vary with culture, selective pressures mediating tooth size have not only changed with time, but vary even among extant populations. Determining the particular factors operable in any one culture demands a detailed study of the population in question. As Nubia is one of the most thoroughly studied skeletal populations, it provides a good model for the ways in which selection might act on dental size.

Reduction in the dentition and masticatory apparatus in Nubia: Temporal correlates and natural selection

The work of Carlson (1976a,b) and Carlson and Van Gerven (1977, 1979) indicates that the " progressive decrease in the size and robusticity of the mandible and masticatory apparatus is the dominant feature in the transition from the Mesolithic period through

the Christian horizon in Nubia" (Carlson and Van Gerven, 1977:502). Nine of the eleven craniofacial dimensions that decreased in size after the Mesolithic[2] were directly associated with masticatory function. Masseter origin length displayed the greatest dimensional change, experiencing a dramatic 21.6% reduction between the Mesolithic (ca. 12,000 B.C.) and Agriculturalist period (3,400–1,100 B.C.), when the majority of the change occurred for all variables.

Not all craniofacial measurements decreased in size over that time period, however, as might be expected if the size reductions simply represented a decrease in body size. Four variables pertaining to relative height of the cranial vault and face increased, resulting in a shifting in craniofacial shape to a more globular form and a less robust craniofacial appearance. These results are best explained by a decrease in masticatory functional demands related to a dietary shift. Mesolithic Nubia was characterized by intensive gathering and hunting with a focus upon seeds and large bovids, whereas the Agriculturalist period exhibits more reliance upon the cultivation of barley, millet, and sorghum, with some herding (Goodman et al., 1986). Based on the decreased robusticity of the masticatory apparatus, Carlson and Van Gerven (1977:504) predicted a "reduction in the size of the teeth and associated alveolar region due to the reduced anteroposterior growth of the maxillomandibular complex."

Mandibular data gathered by Calcagno (1984) substantiated these claims, leaving little doubt that a reduction in the size of the masticatory apparatus occurred in Nubia. Virtually every mandibular length, breadth, and height measurement decreased significantly from the Mesolithic to the Agriculturalist period in both sexes, including height and breadth of the corpus and ascending ramus, the breadth of the mandible and the maximum length of the mandible. Overall, males experienced a slightly greater decrease (10%) in these measurements than did females (7%). Much smaller decreases in size of the masticatory apparatus are evident between the Agriculturalist and Intensive Agriculturalist (A.D. 0–1,400) phases, with average reductions of only 1–2% for each sex.

Tooth size reductions parallel the reduced metrics of the masticatory apparatus. All teeth decreased markedly in size from the Mesolithic to Agriculturalist period in accordance with major reductions in jaw size, but only the molars reduced between the two agricultural phases (Calcagno, 1984, 1986).

The major decreases that occur in size of the masticatory apparatus between the Mesolithic and Agriculturalist periods suggest that teeth would have been under selective pressure to reduce in size to avoid the negative sequelae of dental crowding. The fact that all teeth reduced in size, coupled with data indicating a low incidence of caries at this time period, lends further support to crowding rather than caries as the selective mechanism. This hypothesis could be tested by correlating tooth size with detailed data on dental crowding and/or dental disease due to crowding during this time frame.

Between the two Agriculturalist periods jaw size changed minimally, suggesting at least a partial abatement of selection to avoid dental crowding. Increased reliance upon an agricultural subsistence pattern, however, would have resulted in an increasingly cariogenic diet (Brothwell, 1963a,b; Greene, 1970). As molar teeth are the most susceptible to caries, the decreased size of the molars observed during this time frame is as expected given such a dietary shift. Again, this model could be tested by collecting data on the incidence of caries in this population and, in fact, caries incidence has been found to have increased dramatically from the Mesolithic (1.01%) to the Intensive Agriculturalist period (12.2–18.0%) in Nubia (Armelagos, 1968; Greene et al., 1967). One might also predict a decrease in morphological complexity of the molars (less pits and fissures to harbor caries), and Greene (1972) has reported that molar morphology in the Agriculturalists is characterized by a much less complex cusp and fissure pattern than observed in the Nubian Mesolithic, even though discrete dental traits indicate biological continuity between these samples.

Hence, the tooth size reductions evident in Nubia tentatively conform to the predictions of selectionist models, which await further testing. In contrast, the pattern of reduced size of all teeth during the Mesolithic/Agriculturalist transition followed by reduced size of the molars alone would not be predicted by the PME model, since there would be no reason to predict differential reduction of any

[2]This material may be more accurately labeled as "Final Paleolithic;" but to maintain continuity with previously cited research on the subject, we will continue to use "Mesolithic" in reference to this time frame.

specific group of teeth. There is no rationale, under the PME, to assume that certain teeth were subject to accumulating mutations during one period of time and other teeth affected during another period, if a general relaxation of selection for tooth size had occurred. Moreover, if teeth are useless, we would predict positive selection for tooth loss, rather than for smaller teeth. Even relatively small teeth may become carious or crowded in small jaws, thereby producing disease. Thus, if teeth are not needed, it would be better to have no teeth than to have small teeth. If they are needed, natural selection should operate to maintain a particular range of variation in tooth size that is compatible with those needs.

CONCLUSIONS

Based on genetic, developmental, clinical, and bioarchaeological data, it is suggested that natural selection can better explain reductions in dental size than can the PME. However, as Endler (1986:97) so concisely understated, "natural selection is hard to detect." This paper offers possible selective forces accounting for the trend of reduced tooth size observed in many human groups and predicts associated increases in particular dental and gnathic abnormalities to be found in the skeletal record. Such selectionist models require additional and rigorous testing. Systematic analyses of dental size and pathology in skeletal populations are needed, as well as data from living populations having undergone documented and dramatic shifts in diet to gain levels of control on the relations of dental pathology, tooth size, and fitness that are not possible with fossil samples.

Although important questions remain unanswered in selectionist models, unlike the PME, these models are indeed testable. Under the PME, if random mutations are free to accumulate, one might anticipate increased variation in tooth size and increased dental asymmetry over time. Although Brace and McKee may be correct in noting that such diachronic changes are not necessarily relevant to the validity of the PME, what they have failed to indicate is any basis upon which the PME can be invalidated. Documenting dental reduction and then merely stating that the PME accounts for smaller teeth is not enough, for the model must somehow be tested. If it cannot be tested, its value as a scientific model is questionable.

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