

Muscling in on the truth

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“In spite of the evidence, the profession holds the concept of muscular causes of malocclusion at arm’s length. Because when it is acknowledged, then the moral imperative for big changes will be inescapable...”

Debate on the causes of malocclusion has been raging since the genesis of the orthodontic science, but has the answer already been found?

Various factions in the orthodontic tradition have declared the influence of both environmental and genetic determinants in malocclusion. Common consensus regards tooth position to be more environmentally influenced and skeletal development more genetically.

Genetic factors

Lauc et al¹ (2003) claim that genetic factors are significant in malocclusion, citing a number of twins studies. However, sibling genetic correlations are intrinsically fallacious in that they do not consider the influence of shared environments, which Garn et al² (1979) have termed the ‘co-habitational effect’.

Nonetheless, certain traits do seem to be characteristic among family members and a possible explanation is that all animals seem to inherit certain muscular functions; Wiley³ (1962) describes the mating ritual of the Three Spine Stickleback, stating “the pattern and sequence of these movements is just as much a part of the genetic make-up of the fish as its body shape”.

Epidemiological studies of malocclusion show it does not follow Mendelian laws of inheritance. Mew⁴ (1986) cites the example of sickle cell

anaemia, which provides near immunity to malaria. It has become endemic in populations where it is an asset for survival and it displays a progressive familial and geographic spread. Malocclusion, however, has shown no type of progressive spread, with every permutation found in every corner of the globe. An evolutionary change of this magnitude would also require millions of years, not one generation and furthermore, what genetic advantage has malocclusion provided for this supposed evolutionary change to materialize?

Certainly, there is a quality of irrationality to the genetic model for the aetiology of malocclusion, but what is the answer?

Environmental factors

Evidence for environmental causes is formidable. Weiland et al⁵ (1997) compared skulls from 19th century Austrian males with their contemporaries, finding that change in diet ensured the latter displayed significantly higher malocclusion scores. Corruccini and Lee⁶ (1984) reported that malocclusion was significantly worse in Chinese children born in the UK compared to their immigrant parents, raised in less developed areas. Since genetic factors remained unchanged, the malocclusion in the offspring was attributed to diet, premature deciduous tooth loss from caries and oral respiration. Corruccini and Beecher⁷ (1981, 1983, 1984) have also shown that a soft diet significantly



Figure 1. Before Treatment - December 2009.



Figure 2. May 2010.

increases dental and skeletal malocclusions in rats, macaques and primates. This is most likely due to less tonicity in muscles of mastication, resulting in compensatory overactivity in muscles of facial expression.

Perhaps most telling has been Harvold's series of experiments on primates⁸ in which induced oral respiration caused a range of malocclusions, but all included increased face height, steeper mandibular plane and larger gonial angle; in short, skeletal and dental discrepancies. Harvold's summation was that oral respiration was the trigger factor, but it is 'deviant muscle recruitment' which directly causes maldevelopment.

The weight of the evidence, be it from the genetic or environmental school, seems to rest with muscle dysfunction being the cause of malocclusion. Texture and nutri-

tional value of diet has been shown to have an impact on tonicity of facial muscles, oral respiration causes 'deviant muscle recruitment' and even from the genetic standpoint, the animal kingdom shows a marked tendency for muscle function (and dysfunction) to be inherited.

P.R. Begg's seminal 1954 manifesto⁹ asserted that a lack of grit in modern diets results in less interproximal wear and subsequently more crowding. Although Begg believed that this environmental factor caused dental crowding, his theory was predicated on the belief that skeletal form is inherited and unmalleable. Currucinni¹⁰ (1990), however, discredited this research and recognised that Begg's own figures render his theory redundant because both crowding and attrition increase with age.

Despite being roundly refuted, Begg's assertion still serves as the rationale and justification for orthodontists to shorten dental arches via extractions to this day.

In Figure 1, the 13-year-old girl's profile shows a severely underdeveloped mandible, with a subsequent overbite. The strain of the mentalis muscle also betrays a 'reverse swallow' with mentalis activity, which is the cause of this skeletal malocclusion. After 6 months of myofunctional appliance use and myofunctional therapy, the release of muscle tension has allowed the mandible to translate anteriorly, with seemingly spontaneous lower dental alignment also a happy bonus.

In spite of the evidence, the profession holds the concept of muscular causes of malocclusion at arm's length. Because when it is acknowledged, then the moral imperative for big changes will be inescapable.

That time is now.

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