

Genetics *versus* environment in the aetiology of malocclusion

A. McKeever¹

IN BRIEF

- Provides an evidence-based overview of the human occlusal arrangement as it usually occurs in nature.
- Addresses the genetics *versus* environment question as it relates to the aetiology of malocclusion.
- Suggests that the conflation of different classifications of malocclusion may stifle the debate on their phylogenesis.

The ongoing debate regarding the relative contributions of heredity and environment to the aetiology of malocclusion would benefit from both a more careful interpretation of the evidence and the abandonment of the tendency to conflate under one umbrella term distinct clinical entities, which may in turn have different aetiologies. D. Normando's letter of 24 February 2012 (*BDJ* 2012; **212**: 153) raises some interesting and surprising points, which deserve attention and comment.

INTRODUCTION

The question of genetics *versus* environment as it relates to the aetiology of malocclusion is a significant one, yet there is too often a tendency to bring together distinct clinical entities, which may have different aetiologies, to construct an argument. This evidence-based overview aims to breathe new life into the debate on the phylogenesis of malocclusion through a more open-minded and careful interpretation of the facts.

CROWDING WITH TOOTH WEAR

D. Normando writes that a study of isolated Amazonian communities shows that genetics is more important than environment in the aetiology of malocclusion and considers an individual with crowding in the presence of wear.¹ Most of the evidence on occlusal variation in either historical civilisations²⁻⁸ or in modern pre-industrial populations⁹⁻¹² demonstrates that the prevalence of malocclusion in these groups is very low and usually coupled with high levels of attritive tooth wear. Tooth wear is an important element in this discussion, as it is posited that once a Class I occlusion is established during childhood, it is maintained by a process of gradual mesial

drift, facilitated by interproximal and occlusal/incisal attrition.¹³ Older adults in these well-occluded populations therefore typically exhibit what would clinically be described as heavily worn Class III edge-to-edge malocclusions with a slight tendency towards posterior crossbites. Crucially, conventional features of Class II malocclusion such as overbite and overjet are almost always entirely absent. This and the observation that these individuals also exhibit larger mandibular dimensions¹⁴ – presumably through greater functional demands on the masticatory apparatus – provide the basis for the 'environment' argument. The findings of Normando *et al.*¹⁵ are therefore very interesting and appear contrary to the current thinking.

DISCUSSION

It is unfortunate, however, that the example provided by the author shows an individual with tooth substance loss more characteristic of chemical erosion, rather than attritional wear as exemplified in Figure 1 (from my own as yet unpublished research on a 16th century New Mexican population). Furthermore, Normando's own research described a two-centre cross-sectional study where the mean ages of the study subjects were too young to assess the effects or otherwise of attritional wear (12.5 and 10.2 years). The main findings of the paper appear to be statistically significant increases in anterior open bites and Class III malocclusions in the inbred population, which would indeed support



Fig. 1 Attritional tooth wear. Note that degree of wear is proportional to eruption sequence (reproduced with permission from the Duckworth Collection)

the hypothesis that genetics has a more important role than environment when all other factors are effectively controlled. A high prevalence of Class III malocclusion has previously been documented in an isolated archaic population⁵ – again contrary to the observations in their well-occluded contemporaries – also suggesting a genetic explanation. The authors also found a statistically significant increase in Class II malocclusions and overjet, although as there was no significant increase in overbite it is assumed that this represents anterior open bite individuals.

CONCLUSION

Perhaps it is time to appreciate the futility of presenting the debate over heredity *versus* environment in the aetiology of malocclusion as a simple dichotomy. What the evidence tells us is that Class II malocclusions with increased overbites never occur in the ancestral environment; however, within one

¹General Dental Practitioner, Whitetree Specialist Centre, 1-3 North View, Bristol, BS6 7PU
Correspondence to: Aidan McKeever
Email: aidan@gotadsl.co.uk

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or two generations of adopting a modern urban culture they are rife.⁹⁻¹² Class II anterior open bites and Class III malocclusions do occur and, as far as we know, always have occurred in nature. Might it not be reasonable to suppose that heredity plays a more important role in the aetiology of the latter two, and function/environment in the former? It may be that the term 'malocclusion' encompasses various clinical phenomena – as well as variations of normal – that occupy opposite ends of the diagnostic and phenotypic spectrum and might therefore be better considered unrelated for research purposes. When it comes to comparing notes we can then at least be sure we are talking about the same problem.

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