

Aetiology of Malocclusion Revisited

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The other day, while flipping through the latest edition of the textbook of *Contemporary Orthodontics* by Proffit¹, a pie chart caught my attention, which depicted that the aetiology of malocclusion is known for only 5% of the population. It was one of those déjà vu moments, taking me back to Proffit's 1986 paper² based on his lecture to the British Society, where the very same chart had appeared. That got me thinking, nearly four decades later, the figure has not changed. For all the advances in orthodontics over these years, we still know remarkably little about the aetiology of malocclusion.

A clear understanding of the aetiology of malocclusion is central to orthodontics because it underpins every decision, from diagnosis to treatment planning and retention. However, it has been a debated topic, with some attributing the aetiology to genetics and others to environmental factors. Proffit's seminal paper² seemed to have settled this dispute by acknowledging the role of both genetic and environmental factors in the aetiology of malocclusion. Yet, we still lack clarity regarding the relative contributions of these factors and how their influence changes over time.

Generic discussions of genetic and environmental factors as causes of malocclusion offer little clinical value. We need answers with direct practical implications. The good news is we now have the tools to investigate the aetiology of malocclusion in much greater detail. Three-dimensional imaging and geometric morphometrics allow us to capture dental and craniofacial structures with a precision that was unimaginable just a few decades ago. Twin studies employing genetic modelling can help disentangle the contributions of genetic, epigenetic, and environmental factors to the development of dentofacial traits.³ Longitudinal twin studies take this further,

revealing how these influences change with time.⁴ Add genomics to the mix, and we are finally in a position to address this critical question that has long eluded us.

By integrating precise three-dimensional measurements of twins with genomic data, we may finally uncover the aetiology of specific malocclusion traits. This understanding can refine diagnosis, guide targeted treatment plans, and address relapse, which often stems from our incomplete understanding of underlying causes. We can move beyond merely correcting malocclusion and start preventing it. By tailoring interventions to each patient's unique genetic and environmental profile, we can provide orthodontic treatment that is fully personalised.

Proffit's pie chart should make us pause and reflect. The next major breakthrough in orthodontics may not be another appliance or technique, but a comprehensive understanding of the aetiology of malocclusion. With such understanding, we may be able to prevent malocclusion or achieve more stable treatment outcomes. Until then, we continue treating the effects rather than the causes.

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