Letters to Editor

Etiological factors of temporomandibular joint disorders

Sir,

We were happy to see an article on "etiological factors of temporomandibular joint disorders" in Jul-Dec issue (Sharma S, Gupta DS, Pal US, Jurel SK. Etiological factors of temporomandibular joint disorders. *Natl J Maxillofac Surg* 2011; 2:116-9). We congratulate these authors for providing state-of-art review on the etiology of temporomandibular disorders but genetic basis of temporomandibular joint disorders (TMD) was not discussed in this paper. Previously, as in this paper, orthodontic treatment has been variously cited as both a protective and/or a harmful factor in TMD etiology. In a meta-analysis in 2002, a systemic review of 31 studies by Dr. Kim, Dr. Graber, and Dr. Viana drew no definitive conclusion about the relationship, and found that the data "do not indicate that traditional orthodontic treatment increased the prevalence of TMD."^[1]

Recently, a search has begun for a genetic influence on TMD etiology. Now, researchers and clinicians are becoming increasingly aware of the possibilities that genetic variation may play a role in the pain perception and onset of TMD. In 2003, Zubietta and coworkers reported that a common variant of the gene that codes for the enzyme catechol-O-methyltransferase (COMT) was associated in human beings with diminished activity of pain regulatory mechanisms in the central nervous system (CNS).^[2] In a study, it was reported that subjects with pain-sensitive haplotypes of COMT have elevated responses to standardized noxious stimuli compared with people who have pain-resistant haplotypes.^[3] Hence, it seems likely that people with pain-sensitive haplotypes would have experienced relatively greater discomfort or pain when undergoing procedures used during fixed orthodontic treatment. And, there is additional experimental evidence that people with genetically downregulated COMT have reduced analgesic effects of endogenous opioid systems within the CNS.^[2] Taken together, these findings provide some biological plausibility to support an interpretation that orthodontic treatment could be a risk factor for TMD. In 2008, Slade et al. reported the findings from a prospective cohort study of 186 females that illustrate an example of gene-environment interaction in TMD onset.^[4] However, important methodological features of this study should also be considered. Final conclusion in this recent genetic-based study^[4] was drawn as, "On balance, therefore, it would be premature to propose that orthodontia is a risk factor for TMD, even among the subgroup of females with pain-sensitive haplotypes of COMT." However, based on current evidence about biological processes involved in pain regulation, it seems plausible that there could be a subset of the population that is relatively sensitive to noxious stimuli, and for those individuals pain experienced during orthodontic treatment may interact with that pain sensitivity. Furthermore, the combined attributes of COMT pain-sensitive haplotypes and the receipt of orthodontic care were useful markers to identify a subgroup with particularly high risk of developing TMD. While further studies are needed to investigate TMD etiology, this genetic variant potentially could help to identify patients, whose risk of developing TMD is heightened following orthodontic treatment, hence serving as a risk marker useful in planning orthodontic care.

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