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LETTER TO THE EDITOR

# Does the level of salivary nitric oxide independently estimate to which extent periodontium is affected?

Dear Editor,

Recently, an article entitled 'Salivary nitric oxide levels in inflammatory periodontal disease – A case–control and interventional study' by Parwani *et al.* (1) is published in the present journal. I read the article with interest. There is some controversy that how periodontitis is correlated with salivary level of nitric oxide. As my colleagues and I have the same experience (Khosravi M, Poorsattar Bejeh Mir A, Kashiri A, Quejeq D, unpublished data), I have some comments and suggestions.

Dr Parwani conducted a case–control and interventional (before–after) study, comparing the level of nitric oxide level between the healthy individual and patients with affected periodontium and assess the outcome of periodontal therapy by changes in salivary NO content. The main believed reason to rationale the decrease of NO, after the appropriate periodontal treatment, is that of attenuation of pro-inflammatory cytokines with resultant suppression of iNOS expression (inducible isoform, NOS2). This distinct isoform of NO synthase enzyme family is not expressed considerably within normal endothelial layers by un-stimulated macrophages. On the contrary, eNOS (endothelial type, NOS3) is regularly expressed in healthy endothelium in a calcium-dependent manner to a lesser amount that lasts shorter when compared to NO produced by NOS2 (2). As far as I know, NO produced from either NOS2 or NOS3 cannot be distinguished from each other when assessed by Griess reaction. The success rate is also dependent on the extent of improvement that conferred by treatment and is properly detectable by tracing the eNOS expression rather than measuring total NO. Some authors agreed upon and some argued to this finding that periodontal therapy decreases the salivary NO (3).

Based on existing literature, I believe that the casual relationships between NO and severity or progression of periodontitis should be sought elsewhere. To my opinion, amount of produced nitric oxide may not independently determine or estimate the periodontal tissue health status. The amount of concurrent free radical species (ROS) dictates whether existing NO exhibits bio friendly characteristics or react with ROS and peroxynitrite formation leading to tissue destruction. (4, 5) The latter is produced when NOS is uncoupled. Furthermore, ROS itself can uncouple the NOS. On contrary to this, when essential substrates (e.g. $O_2$ and arginine) are available, NOS can maintain its proper spatial structure, coupled form, which may be a better estimation of prognosis for produced NO. More precisely, maintaining capacity may be tested by incubation with inhibitors of electron transmission from NAPDH-FAD-FMN chain to $O_2$ and arginine as substrates [e.g. N-nitro-l-arginine (L-NNA) or, N-nitro-l-arginine methyl-ester (L-NAME)] (5).

Conclusively, a small amount of NO with large amount of ROS may disturb periodontium homeostasis more than large amount NO that is produced from coupled form of NOS in the absence of large body of ROS.

## References

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4 Channon KM. Tetrahydrobiopterin, regulator of endothelial nitric oxide synthase in vascular disease. *Trends Cardiovasc Med* 2004; **14:** 323–327.

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