ERRATUM

Owing to the omission of two reference citations, the article titled "Expression of Toll-like Receptor 2 and 4 in Dental Pulp" published in J Endod 2007;33 (10): 1183-1186 published with several reference citations misnumbered.

References 8-12 should have been cited as follows:

However, according to more recent data, the TLR-2 recognizes unknown cell-wall components of *P gingivalis* rather than LPS itself (8) strengthening the hypothesis that TLR-4 is the principal signal transducer for LPS and TLR-2 is a signal transducer for other bacterial components, such as peptidoglycan and lipoprotein.

Recently, Dillon et al (9) suggested a critical role for the extracellular signalregulated kinase activation after signaling through TLR-2 via a mechanism that is independent of c-Fos and showed that odontoblasts activated through TLR-2 are able to initiate an innate immune response by secreting chemokines that recruit immature dendritic cells while downregulating their specialized functions of dentin matrix synthesis and mineralization (10). Odontoblast-like cells express TLR-4, and LPS-induced VEGF expression is mediated, at least in part, by TLR 4 signaling (11).

Oral microorganisms induce the dental caries and pulpitis as demineralization of enamel, which constitutes an impermeable barrier that protects the underlying dentin and pulp in the center of the tooth. Once the enamel barrier is disrupted, oral microorganism products diffuse through the dentinal tube toward the pulp (12).

And references 13-17 should have been cited as follows:

In this study, we established a murine dental caries model with pulpitis by using exposed cavity preparation and showed the expression of TLR-2 and 4 mRNA in the inflammatory pulp cells by using real-time PCR and immunohistochemistry. The major findings in our study are as follows: (1) the TLR-2– and TLR-4–expressed cells were induced on mainly macrophage and dendritic-like cells in the early stage of pulpitis; (2) the expression of TLR-2 mRNA in pulp cells begins to increase 3 hours after infection, reaching a maximum level at 9 hours and gradually decreasing from 9 hours to 72 hours; and (3) the mRNA level of TLR-2 mRNA was 30-fold higher than that of TLR-4 mRNA at 9 hours after infection. The significantly higher levels of TLR-2 mRNA 9 hours after infection may have resulted from the stimulation of dental pulp cells by gram-positive bacteria. Matsuguchi et al (13) speculated that, when gram-negative bacteria invade a host, macrophages first recognize LPS through TLR-4, whereas TLR-2 is induced later, either directly by LPS or indirectly through secondary cytokines; they also suggested that macrophages respond better to LPS or other bacterial components such as lipoproteins, which are membrane components of both gram-positive and gram-negative bacteria through the newly synthesized TLR-2 in the cell-level analysis. This difference in stimulation may explain the higher level of TLR-2 at 9 hours after infection. The reason for the discrepancy was explained by the species of infiltrated microorganisms into the dentinal tube. Almost all of the infiltrated species were grampositive microorganisms in the early stage of pulpitis in the murine caries model such as in human caries (14). Figure 2*G* showed that numerous gram-positive microorganisms (cocci) infiltrated into the dentinal tube and nearly reached the odontoblast layer at 24 hours. The TLRs are important factors in innate immune responses because they mediate signals from microorganisms products in relation to inflammatory reactions. In this study, the TLR 2 mRNA level was 30-fold higher than the TLR-4 mRNA level, 9 hours after infection, although anti–TLR-2– and anti–TLR-4–positive cells were observed in and around the odontoblast layer and center of inflammatory pulp. TLR-2 and TLR-4 are expressed at high levels in cells that respond to LPS, such as peripheral blood leukocytes, macrophages, and monocytes (15). TLR-4 mRNA and protein are expressions that were both present in cells of the odontoblast layer and pulp tissues, and TLR-4 expression has been shown in odontoblasts and some pulpal vascular endothelial cells (16). Therefore, it has been suggested that various cell types express different combinations of TLRs in order to recognize microorganisms pathogens. Anti– TLR-2–positive cells may be identified to almost macrophage and dendritic cells because the distribution of anti–TLR-2–positive cells and CD64-positive cells were almost similar in the inflammatory pulp at 9 hours in this study. This study also showed the presence of diverse inflammatory and immunocompetent cells in pulpitis. These observations support the view that a wide range of defense reactions, including humoral immunity, cell-mediated immunity, and nonspecific inflammatory reactions, function locally in pulpitis. It is not yet known which of the mechanisms are more operative in eliminating the continuously invading bacterial antigenic substances derived from infected root canals. Changes in the cellular composition may reflect the status of the defense reactions functioning during each phase of pulpitis.

Our quantitative data indicated that the expression levels of TLR-2 and TLR-4 are associated with the early pulpitis stage at the same time of bacterial inversion to dentinal tube. The results also support the concept that early treatment of a carious lesion will be a biologically rational approach because it may help the tooth to use its innate immune defense potential to overwhelm the carious antigenic challenges(17).

The Journal of Endodontics regrets these errors.

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