

Cellular crosstalk mechanism of Toll-like receptors in gingival overgrowth (review)

ABSTRACT

Gingival overgrowth is an undesirable outcome of systemic medication and is evidenced by the accretion of collagenous components in gingival connective tissues along with diverse degrees of inflammation. Phenytoin therapy has been found to induce the most fibrotic lesions in gingiva, cyclosporine caused the least fibrotic lesions, and nifedipine induced intermediate fibrosis in drug-induced gingival overgrowth. In drug-induced gingival overgrowth, efficient oral hygiene is compromised and has negative consequences for the systemic health of the patients. Toll-like receptors (TLRs) are involved in the effective recognition of microbial agents and play a vital role in innate immunity and inflammatory signaling responses. TLRs stimulate fibrosis and tissue repairs in several settings, although with evident differences between organs. In particular, TLRs exert a distinct effect on fibrosis in organs with greater exposure to TLR ligands, such as the gingiva. Cumulative evidence from diverse sources suggested that TLRs can affect gingival overgrowth in several ways. Numerous studies have demonstrated the expression of TLRs in gingival tissues and suggested its potential role in gingival inflammation, cell proliferation and synthesis of the extracellular matrix which is crucial to the development of gingival overgrowth. In the present review, we assessed the role of TLRs on individual cell populations in gingival tissues that contribute to the progression of gingival inflammation, and the involvement of TLRs in the development of gingival overgrowth. These observations suggest that TLRs provide new insight into the connection among infection, inflammation, drugs and gingival fibrosis, and are therefore efficient therapeutic target molecules. We hypothesize that TLRs are critical for the development and progression of gingival overgrowth, and thus blocking TLR expression may serve as a novel target for antifibrotic therapy.

Keyword: Gingival overgrowth; Cellular crosstalk; Toll-like receptors; Medication