

Nickel (II) Effects on NFκB Subtype P52

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Abstract

Objectives: Driven by its low cost and availability, nickel alloys are used extensively in dentistry, but their use is controversial due to release of Ni(II) corrosion products. Recent literature reports that Ni(II) increases inflammatory cytokine secretion from LPS-activated monocytes, but does not alter NFκB p65 levels or translocation. Yet, little is known about the effects of Ni(II) on other NFκB subtypes such as p52, which serves regulatory roles in NFκB function. Our goal was to assess the effect of Ni(II) on p52 in monocytes exposed to Ni(II) with or without LPS, which is an inflammatory component of dental plaque.

Methods: Human THP-1 monocytes were exposed for 72 h to Ni(II) (0, 30, and 50 μM), ± LPS (1 μg/mL) for the last 0.5, 2, and 6 h. Levels of p52 were measured using immunoblots of whole-cell or cytosolic proteins, probing for p65, p52, β-actin, and IκBα. Protein levels were detected via chemiluminescence captured to radiographic film.

Results: In whole-cell lysates, p52 was marginally detectable without LPS, increased markedly by LPS, and unaffected by Ni(II). In cytosolic fractions, no significant p52 was observed with or without LPS, regardless of Ni(II) concentration. p65 and IκB controls suggested that the NFκB pathway was intact and functioning. β-actin controls suggested repeatable, equivalent gel-lane loading.

Conclusion: Ni(II) did not alter THP-1 p52 levels in the cell or in the cytosol. Under +LPS conditions, we speculate that increased p52 levels primarily occur in the nucleus. Immunoblots of nuclear protein are underway to confirm our speculation.