CONTACT DERMATITIS AND OCCUPATIONAL DERMATOSES

ANALYSIS OF TOLL-LIKE RECEPTORS AND INFLAMMASOMES IN ALLERGIC CONTACT DERMATITIS TO METHYLCHLOROISOTHIAZOLINONE / METHYLISOTHIAZOLINONE

Heliana Freitas De Oliveira Góes [(1)] - Anangélica Rodrigues Virgens [(1)] - Maria Notomi Sato [(1)] - Vitor Manoel Silva Dos Reis [(1)] - Gabriel Costa De Carvalho [(1)] - Luanda Mara Da Silva Oliveira [(1)] - Anna Claudia Calvielli Castelo Branco [(1)] - Anna Julia Pietrobon [(1)]

University Of São Paulo, Dermatology, São Paulo, Brazil [(1)]

INTRODUCTION: Allergic contact dermatitis (ACD) to methylchloroisothiazolinone / methylisothiazolinone (MCI/MI) has been increasing in recent years. It is related to cosmetic products, preservatives, among others.

OBJECTIVE: Evaluating the participation of innate immunity to MCI/MI.

MATERIALS AND METHODS: Subjects with positive Patch test for MCI/MI (DCA +) (n = 13) and negative (Control) (n = 11) were evaluated: in vitro to analyse the effect of MCI/MI and MI on inducing cytokines by peripheral blood mononuclear cells (CMNs) by flow cytometry and in situ by real time PCR.

RESULTS: MCI / MI induced low responsiveness to the CMNs of the individuals tested, but at higher levels of TNF-α, IL-6 and IL-1β in DCA + cases whose cytokines were inhibited by the IKK2 inhibitor (TPCA). LPS-RS inhibited IL-1β secretion. MCI/MI test biopsies showed low expression of TLR4 and Arginase-1, high production of IL-6, IL-4, IL-13, Foxp3, IL-10, decreased M2 macrophages, and possible presence of M1 by expression of CXCR3A and CXCL10.

CONCLUSIONS: The unpublished results show that MCI/MI in vitro induces low responsiveness to the CMNs of the tested individuals, however at higher levels of TNF-α, IL-6 and IL-1β in DCA + cases. It is noteworthy that those responding to the compound, cytokines were inhibited by the IKK2 inhibitor (TPCA), evidencing that the compounds activate this pathway for induction of the proinflammatory cytokines. In addition, it was observed that the competitive inhibitor of TLR4, LPS-RS, is able to inhibit IL-1β secretion, suggesting that the MCI/MI activates via TLR4. Test-contact biopsies showed low expression of TLR4 and Arginase-1 in relation to the control, but high production of IL-6

(proinflammatory), IL-4, IL-13 (cytokines related to the Th2 profile), Foxp3, IL-10 (regulators). In addition, the data suggest that there is a decrease in M2-type macrophages, and the possible presence of M1 by the expression of CXCR3A and CXCL10.