Effects of thymol and carvacrol on T-helper cell subset cytokines and their main transcription factors in ovalbumin-immunized mice

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Abstract

Thymol and carvacrol, two main components of thyme, have several valuable effects on the immune system. This study aims to evaluate the effects of these components on T-helper (Th) cell responses and their subsets in mice immunized with ovalbumin. The effects of these components on: a specific in vivo immune response were evaluated by assessing changes in delayed-type hypersensitivity (DTH); ex vivo splenocyte proliferative responses were evaluated using a BrdU assay gene expression of cytokines and key transcription factors involved in T-cells subset differentiation among the mouse splenocytes were assessed using real-time polymerase chain reaction (PCR); and splenocyte cytokine formation (ex vivo) and levels of the cytokines in mouse sera were measured by ELISA. Mice treated with thymol or carvacrol had reduced DTH responses (26% and 50%, respectively) compared with control mice. Thymol and carvacrol each diminished splenocyte proliferation to nearly 65–72% of control levels (p < 0.01). These agents also led to decreased Th1 [interleukin (IL)-2, interferon (IFN)-γ], Th2 (IL-4) and Th17 (IL-17A) levels in the splenocyte cultures and in the sera of mice but increased levels of IL-10 and transforming growth factor (TGF)-β. Treated immunized mice showed significantly reduced T-box 21 (T-bet) expression from 3.8 [±0.3]-fold in untreated ovalbumin-immunized mice to 0.9 [±0.4]-fold (thymol) and 0.8 [±0.2]-fold (carvacrol) (p < 0.01). GATA binding protein 3 (GATA-3) expression declined from 3.4 [±0.4]- to 0.5 [±0.3]-fold (thymol) and 0.6 [±0.4]-fold (carvacrol), whereas RORγt decreased from 13.4 [±1.6]- to 1.5 [±0.6]-fold (thymol) and 0.8 [±0.4]-fold (carvacrol) (p < 0.001). As carvacrol and thymol each suppressed the antigen-specific immune response by reducing Th cell-related cytokines/specific transcription factors, this indicated their potential to modulate destructive immune responses attributed to T-cells over-activation. © 2016 Informa UK Limited, trading as Taylor & Francis Group