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Suppression of IP-10/CXCL10 gene expression in LPS- and/or IFN-γ-stimulated macrophages by parasite-secreted products

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Abstract

T helper (Th)2 polarized immune responses are characteristically dominant in helminth infections. The gene expression of interferon (IFN)-γ-inducible protein 10 (IP-10/CXCL10), which promotes Th1 responses, in mouse macrophages stimulated with lipopolysaccharide (LPS) and/or IFN-γ was suppressed by excretory/secretory (ES) products of *Spirometra erinaceieuropaei* plerocercoids. ES products suppressed LPS- and/or IFN-γ-induced transcriptional activities of a luciferase reporter gene under the control of a 243-bp fragment of the IP-10 gene promoter/enhancer, which contains an IFN-stimulated response element (ISRE) and two κB elements. Consistent with this result, ES products inhibited ISRE-dependent heterologous promoter activities and LPS- or IFN-γ-induced ISRE-binding activity. ES products also suppressed LPS-induced IFN-β gene expression. Furthermore, ES products suppressed nuclear factor (NF)-κB RelA (p65)-dependent transcriptional activity, whereas ES products had no effect on the κB-binding activity. These results suggest that ES products suppress the IP-10 gene expression by inhibiting the ISRE- and RelA-dependent transcriptional activities in mouse macrophages.

Highlights

► The IP-10/CXCL10 gene expression in macrophages was suppressed by a parasite. ► The parasite-secreted products suppressed LPS-induced IFN-β gene expression. ► The parasite-secreted products inhibit LPS- or IFN-γ-induced ISRE-binding activity. ► NF-κB RelA (p65)-dependent transcriptional activity