Effect of Various Cytokines, Lipopolysaccharide, and Soluble Immune Complexes on the Release of Interleukin-8 by Monocytes from Patients with IgA Nephropathy

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Dear Sir,

Dysregulated cytokine expression has been implicated in the pathogenesis of IgA nephropathy (IgAN) [1]. Interleukin-8 (IL-8) is a chemotactic cytokine with proinflammatory and growth-promoting activities [2]. In order to identify mediators that might induce the release of IL-8 in IgA N, peripheral blood monocytes (PBM) were cultured in the presence of a variety of cytokines. In addition, soluble immune complexes (IC) derived from human glomerular basement membrane, and lipopolysaccharide (LPS) were used as stimuli.

As shown in figure 1, in the PBM of IgA N patients significantly elevated levels of IL-8 were determined compared to normal con-

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• Normals ■ IgAN

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Fig. 1. Effect of various cytokines, LPS, and IC on release of IL-8 by PBM (1×10^5 /ml). PBM from 15 normal individuals (•) and 19 IgAN patients (■) were cultured with or without the indicated agent for 24 h. Single values and means (columns) are shown. As shown by the release after 24 h, IL-1 β , TNF α , GM-CSF, LPS and IC are powerful inducers of IL-8 in PBM both from normal individuals and IgAN patients, $+p < 0.01$; $++p < 0.05$. Furthermore, IL-8 release from PBM of IgA N patients was significantly higher than from PBM of healthy individuals, $*p < 0.01$.

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trois by means of a sensitive and specific enzyme-linked immunosorbent assay (Toray Industries, Inc., Tokyo, Japan). The kit showed no cross-reactivity with human interleukin-1β (IL-1β), interleukin-2, interleukin-6, tumor necrosis factor-α (TNFα), and granulocyte-macrophage colony-stimulating factor (GM-CSF). Furthermore, IL-1β, TNFα, GM-CSF, LPS, and IC were found to be very effective inducers of IL-8 release.

From these results, it may be assumed that some cytokines and IC are potent activators of IL-8 release in IgA N. These results are in good agreement with a recently published report on elevated TNF production in vitro by PBM in IgAN [3]. However, further studies are needed to define the role of IL-8 and other molecules described above in the pathogenesis of IgAN.

References

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