G303 MACROPHAGE-DERIVED TGF-β1 ENHANCES IgA ISOTYPE SECRETION BY LPS-ACTIVATED MURINE B LYMPHOCYTES

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We examined the role of endogenously secreted TGF-β1 in the IgA isotype synthesis by murine B cells. LPS-activated whole spleen cell population was cultured in the presence of anti-TGF-β1 antibody for 5 to 7 days. The number of Ig secreting cells and total secretion was measured by ELISPOT assay and ELISA, respectively. The degree of IgA response in whole spleen cell culture treated with LPS and anti-TGF-β1 antibody was 2-5 fold less than those treated with LPS alone. Similarly, treatment of whole spleen cells with anti-TGF-β1 antibody decreased 5 fold in number of IgA secreting cells. The IgA response in the culture of whole spleen cell population minus macrophages was diminished 2-6 fold as compared to the culture of intact whole spleen cell population. Further, LPS could activate macrophage cell line, P388D1 to secrete an active TGF-β1. Under the same conditions, the induced TGF-β1 transcripts was also detectable by RT-PCR. Finally, the supernatant from activated P388D1 substantially increased IgA secretion by spleen B cells.

Taken together, these results suggest that macrophages coexisted in whole spleen cell population has important IgA modulating activity.

G801 Lymphokine Gene Expressions in Mouse Mast Cell

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It is suggested that the regulations of interleukin-3 (IL-3) and granulocyte-macrophage colony-stimulating factor (GM-CSF) genes expression are mediated through common pathway. To understand signal pathway of lymphokine gene expression, we screened IL-2, IL-3, IL-4, and GM-CSF gene expression in activated mouse mast cell, PB-3c. In PB-3c cells activated by PMA and A23187, lymphokine (IL-2, IL-3, IL-4, and GM-CSF) gene expressions were detected from 15 min. Cycloheximide did not affect on expressions of the gene in activated PB-3c cells. IL-2 gene expression was induced in TPA/A23187-activated PB-3c cells only, but IL-4 gene expression was induced in A23187-activated PB-3c cells. IL-3 and GM-CSF gene expressions were induced in A23187-activated PB-3c cells, and the inductions of these gene were increased about 2.5 fold in TPA/A23187-activated PB-3c cells. In the pretreatment of protein phosphatase(PP) inhibitors, okadaic acid (PP2A and PP1 inhibitor) did not block the induction of the gene expreession. But in the pretreatment of cyclosporin A (PP2B inhibitor), inductions of IL-2 and IL-4 gene expressions were blocked completely, but IL-3 and GM-CSF gene expressions were decreased to about 20%. In the pretreatment of protein (ser/thr) kinase inhibitor, IL-2 and GM-CSF gene expressions were blocked completely, but IL-3 and IL-4 gene expressions were not affected. These results indicate that the induction of lymphokine gene expressions in activated PB-3c cells are mediated by different signal mediators. Moreover PB-3c cells will provide a useful system to investigate the signal transduction pathway for induction of lymphokine gene expression.