G103 NO and TNF-α Secretion in LPS-stimulated Murine Macrophage Are Inhibited by the Stress Response

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To elucidate the role of stress response during macrophage activation, we examined the effects of heat shock (HS) or amino acid analog, 5-Aza, on the secretion of nitric oxide (NO) synthesis and tumor necrosis factor-alpha (TNF-α) secretion from IFN-γ plus lipopolysaccharide (LPS)-stimulated macrophages. HS or 5-Aza markedly inhibited the induction of NO synthesis from IFN-γ plus LPS-stimulated macrophages. While HS or 5-Aza inhibited the amount of NO released, we also examined the discrepancy between HS and 5-Aza on the level of TNF-α secreted from cells. 5-Aza reduced the level of TNF-α in the culture medium at 24 h after cell stimulation. But, HS only reduced TNF-α secretion at the initiation stage of macrophage stimulation. In addition, when cells were stimulated with IFN-γ plus LPS after various time of recovery at normal culture conditions at 37°C, the reversibility of the HS-induced inhibition of NO release by increasing of recovery time was interestingly examined progressively. These results suggested that the cause of inhibition of NO secreted from macrophages by HS may be the modification by crosstalk of signal pathways between NO and HSPs synthesis rather than the increment of cellular contents of HSPs such as HSP72.

G301 Regulation of IgA secretion by CD5 B cells : Possibile role of IL-5 and macrophage-derived TGF-β1

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CD5 B cell (B-1 cell) population is, ontogenically distinguished from conventional B cells (B-2 cells), resides primarily in peritoneal cavity. These B-1 cells are known to be relocated into lamina propria of intestinal tract. However, it is unknown what factors induce B-1 cells to secrete IgA isotype. Previously, we have shown that TGF-β1, in the presence of IL-2 or IL-5, specifically increases IgA isotype production by conventional B cells. Thus, in the present study, we examined the effect of TGF-β1 and IL-5 on Ig expression by murine B-1 cells. TGF-β1 in the presence of IL-5 increased antigen-nonspecific IgA isotype production by LPS-stimulated peritoneal B-1 cells by 2-3 fold but not IgM isotype. Under the same conditions, number of IgA secreting cells and surface IgA expression increased ≈10-fold and 2-3 fold, respectively. Further, LPS-stimulated peritoneal macrophage produced TGF-β1 which, in the presence of IL-5, could also induce B-1 cells to secrete IgA isotype. Next, we examined the antigen-specific IgA regulation by TGF-β1 and IL-5. Mice were immunized intraperitoneally with VP8, which is a part of human rotavirus. TGF-β1 plus IL-5 markedly increased VP8-specific IgA response by B-1 cells (≈90-fold). Finally, we found that peritoneal immunization with BSA confers the intestine to secrete BSA-specific IgA.

Taken together, the results from the present study indicate that macrophage-derived TGF-β1 in the presence of IL-5 can modulate B-1 cells to produce IgA antibody and presumably, antigen-specific B-1 cells in peritoneum repopulates into the intestine.