INTRODUCTION

Systemic lupus erythematosus (SLE) is an autoimmune disorder characterized by B cell hyperactivity and impaired cell mediated immunity (CMI) (Decker et al., 1979 ; Kunkel, 1983). T cells proliferation (Horwitz, 1975 ; Delfraissy et al., 1980), cytotoxic T cells activity (Charpenter et al., 1979 ; Tsokos and Balow, 1983), NK cells cytotoxicity (Tsokos et al., 1985), interleukin 1 production (Alcocer-Varela et al., 1984), and in vivo delayed-type hypersensitivity to various antigens such as tuberculin, trichophyton, and other bacterial and fungal extracts (Rosenthal et al., 1975), are depressed in SLE patients. Depression of CMI function may lead to the reduction of host resistance and increased susceptibility to various infections such as viral and fungal infections (Drutz and Graybill, 1987). These events are often seen in SLE patients (Hamaguchi et al., 1970 ; Staples et al., 1974). However, the early events responsible for these CMI abnormalities in SLE are poorly understood.

Interleukin 2 (IL-2), another CMI parameter, is a product of mitogen or antigen stimulated T cells (Gillis, 1978a). It has an important role in immunoregulation (Robb, 1984 ; Smith, 1984). IL-2 could enhance cytotoxic T cells (Erad et al., 1985) and NK cells activity (Henney et al., 1981) and could regulate IFN $-\gamma$ production (Vilcek et al., 1985). IL-2 was required for clonal expansion of T cells (Kurnick et al., 1979). Thus, deficiency of

IL-2 activity may contribute to various abnormality in CMI function. Studies from several laboratories demonstrated deficiency of IL-2 activity in SLE patients (Alcocer-Varela et al., 1982 ; Linker-Israeli et al., 1983 ; Miyasaka et al., 1984 ; Murakawa et al., 1985; Huang et al., 1986). The decreased IL-2 production in SLE has been explained by excessive suppressor function of $CD8^+$ cells (Linker-Israeli et al., 1985). This was supported by raising the IL-2 production to normal or above normal levels after removal of $CD8^+$ cells from PBMC of SLE patients. Whereas removal of $CD8^+$ cells from PBMC of normal subjects did not significantly increase IL-2 activity. The $CD8^+$ enriched cells of SLE, but not of normal person, decreased IL-2 production when added back to autologous CD8-depleted cells. However, recent finding suggested that active suppressor ($CD8^+$) cells of SLE patients was not always detected *in vitro* by co-culture method (Huang et al., 1986). He and his coworkers demonstrated that adding freshly isolated autologous $CD8^+$ enriched cells to rested $CD4^+$ enriched cells did not lead to suppression of the IL-2 secretion in SLE. The data suggested that *in vitro* deficiency of IL-2 activity may be resulted from exhaustion of T cells that have been continuously activated *in vivo* and deficiency of IL-2 production should be restored when T cells were rested for 2 to 3 days in culture medium prior to be stimulated with mitogen.

As described, in vitro IL-2 production of circulating T lymphocytes in response to motogenic stimulation from patients with active SLE is strongly reduced. It is still not clear whether this abnormality is caused by a defect in the producer lymphocytes or by excessive suppression. By using the autologous erythrocyte rosette technique, Con A-induced suppressor cells can be identified and separated from nonsuppressor cells (Sakane et al., 1981). Suppressor cells regardless of either $CD4^+$ or $CD8^+$ phenotype are confined to the autorosetting T cell populations (Takada et al., 1983). Suppressor cells separated by this method have more potent suppressor activity than $CD8^+$ cells. Thus, suppressor cells separated by this method will be selected to suppress IL-2 secretion.

The main objective of this study are :

1. To compare the ability of IL-2 production between freshly isolated and rested PBMC in patients with active SLE.
2. To study the ability of autorosetting T cells from patients with active SLE to suppress autologous rested PBMC and heterologous rested normal PBMC to secrete IL-2.