LIST OF ILLUSTRATIONS

Fig. 1. Expression of human house keeping gene R18 rRNA in monocytes of patients with SLE, TB and SLE-TB as well as in normal healthy individuals. (Page No: 74)

Fig. 2. Expression of human TNF-α mRNA in monocytes of patients with SLE, TB and SLE-TB as well as in normal healthy individuals. (Page No: 75)

Fig. 3. Expression of secreted TNF-α protein in 24 hr culture supernatants of monocytes of patients with SLE, TB and SLE-TB as well as in normal healthy individuals. (Page No: 76)

Fig. 4. Expression of iNOS mRNA in monocytes of patients with SLE, TB and SLE-TB as well as in normal healthy individuals. (Page No: 77)

Fig. 5. Expression of secreted IFN-γ protein in 24 hr culture supernatants of monocytes of patients with SLE, TB and SLE-TB as well as in normal healthy individuals. (Page No: 78)

Fig. 6. Determination of glutathione peroxidase (GPx) activity in monocytes of patients with SLE, TB and SLE-TB as well as in normal healthy individuals. (Page No: 79)

Fig. 7. Dose response effect of NAC (0-10 mM) on the expression of secreted TNF-α in 24 hr culture filtrates of monocytes of patients with SLE, TB and SLE-TB as well as in normal healthy individuals. (Page No: 80)

Fig 8: MTT cell viability assay for dose-response (0 – 25 ug/ml) effect of ECGC on 24 hr monocytes cultures of patients with SLE, TB and SLE-TB. (Page No: 81)

Fig. 9. Expression of human house keeping gene R18 r18 mRNA in 24 hr monocyte cultures treated with 5 μg/ml of EGCG. The monocytes were of patients with SLE, TB and SLE-TB Monocytes from normal healthy individuals served as controls. (Page No: 82)
Fig. 10. Dose response effect of EGCG (0-25 μg/ml) on the expression of secreted TNF-α protein in 24 hr culture supernatants of monocytes of patients with SLE, TB and SLE-TB as well as in normal healthy individuals. (Page No: 83)

Fig. 11. Effect of SN50 (black bars; 100 μg/ml SN50M) and control peptide SN50M (square bars; 100 μg/ml) on the expression of secreted TNF-α protein in 24 hr culture supernatants of monocytes of patients with SLE, TB and SLE-TB as well as in normal healthy individuals. (Page No: 84)

Fig. 12. NAC (10 mM)-induced modulation of glutathione peroxidase (GPx) activity (U/mg protein) in 24 hr monocytes cultures of patients with SLE, TB and SLE-TB as well as in normal healthy individuals. (Page No: 85)

Fig. 13. EGCG (5 μg/ml)-induced modulation of glutathione peroxidase (GPx) activity (U/mg protein) in 24 hr monocytes cultures of patients with SLE, TB and SLE-TB as well as in normal healthy individuals. (Page No: 86)

Fig. 14. Expression MDA in 24 hr culture supernatants of monocytes of patients with SLE, TB and SLE-TB as well as in normal healthy individuals. (Page No: 87)

Fig. 15. Modulation of the expression MDA levels by 10 mM NAC in 24 hr culture supernatants of monocytes of patients with SLE, TB and SLE-TB as well as in normal healthy individuals. (Page No: 88)

Fig. 16. Modulation of the expression MDA levels by 5 μg/ml EGCG in 24 hr culture supernatants of monocytes of patients with SLE, TB and SLE-TB as well as in normal healthy individuals. (Page No: 89)

Fig. 17. Direct binding ELISA on microtitre ELISA plates that were coated with purified calf thymus dsDNA against anti-DNA antibodies. Normal human sera served as controls. (Page No: 90)

Fig. 18. Direct binding ELISA on microtitre ELISA plates that were coated with Mycobacterial total sonic extract against anti-DNA antibodies. Normal human sera served as controls. (Page No: 91)
Fig. 19. Direct binding ELISA on microtitre ELISA plates that were coated with Mycobacterial total culture filtrate (MTCF) against anti-DNA antibodies. Normal human sera served as controls. (Page No: 92)

Fig. 20. Direct binding ELISA on microtitre ELISA plates that were coated with Mycobacterial total culture filtrate (MTCF) against Sepharose 4B purified SLE IgG i. e. purified anti-DNA antibodies. Normal human sera served as controls. (Page No: 93)

Fig. 21. Inhibition ELISA on microtitre ELISA plates that were coated with dsDNA and ssDNA against Sepharose 4B purified SLE IgG i. e. purified anti-DNA antibodies. The inhibitors were dsDNA and ssDNA. The immune complex prepared was used in place of antibody as described in methods. (Page No: 94)

Fig. 22. Inhibition ELISA for RNA on microtitre ELISA plates that were coated with dsDNA against Sepharose 4B purified SLE IgG i. e. purified anti-DNA antibodies. The inhibitor used to prepare immune complex was RNA. The immune complex was used in place of antibody as described in methods. (Page No: 95)

Fig. 23. Inhibition ELISA for MTCF on microtitre ELISA plates that were coated with dsDNA against Sepharose 4B purified SLE IgG i. e. purified anti-DNA antibodies. The inhibitor was MTCF. The immune complex prepared with varying doses of MTCF was used in place of antibody as described in methods. (Page No: 96)

Fig. 24. Inhibition ELISA for MTCF on microtitre ELISA plates that was coated with dsDNA against immunoaffinity purified anti-DNA antibodies (on DNA-polylysyl-sepharose 4B column) i. e. immunoaffinity purified SLE IgG. The inhibitors were dsDNA and MTCF. The immune complex prepared with varying doses of dsDNA or MTCF, and was used in place of antibody as described in methods. (Page No: 97)

Fig. 25. Direct binding ELISA on microtitre ELISA plates that were coated with dsDNA against TB antibodies (TB sera). Normal human sera served as controls. (Page No: 98)
Fig. 26. Inhibition ELISA against antibodies in sera of patients with TB on plates coated with 30 kDa Ag85B isolated from mid-logarithmic phase cultures of *Mycobacterium tuberculosis* H$_{37}$Rv. The inhibitor used here was varying concentrations of 30 kDa Ag85B. (Page No: 99)

Fig. 27. Comparative Inhibition ELISA against autoantibodies in patients with SLE on plates coated with native dsDNA (non mycobacterial), where the inhibitors used were varying concentrations of native dsDNA versus mycobacterial 30 kDa Ag85B. (Page No:100)

Fig. 28. Comparative Inhibition ELISA against antibodies in patients with TB on plates coated with mycobacterial 30 kDa Ag85B, where the inhibitors used were varying concentrations of mycobacterial 30 kDa Ag85B versus non-mycobacterial native dsDNA. (Page No:101)

Fig. 29. Comparative Inhibition ELISA against antibodies in patients with TB-SLE on plates coated with mycobacterial 30 kDa Ag85B, where the inhibitors used were varying concentrations of mycobacterial 30 kDa Ag85B versus non-mycobacterial native dsDNA. (Page No:102)

Fig. 30. Inhibition ELISA against antibodies in sera of patients with TB on plates coated with total protein lysates obtained from human monocytes isolated from blood of TB patients where the inhibitor used were varying concentrations of the above mentioned total protein lysates obtained from monocytes of TB patients. (Page No:103)

Fig. 31. Inhibition ELISA against antibodies in sera of patients with SLE on plates coated with non-mycobacterial native dsDNA and the inhibitors used were lysates obtained from monocytes isolated from blood of SLE patients where the varying inhibitor concentrations were 0.01, 0.1, 1.0, 10 and 20 µg/ml. (Page No:104)

Fig. 32. Inhibition ELISA against antibodies in sera of patients with TB-SLE on plates coated with non-mycobacterial native dsDNA and the inhibitors used were lysates obtained from monocytes isolated from blood of SLE patients where the varying inhibitor concentrations were 0.01, 0.1, 1.0, 10 and 20 µg/ml. (Page No:105)
Fig. 33. Modulation of TB monocytes with reduced glutathione: Inhibition ELISA against antibodies in sera of patients with TB \((n=5)\) on plates coated with total protein lysates obtained from TB patient monocytes that were co-cultured for 24 hrs with 10 nM of reduced glutathione where the varying inhibitor concentrations used of the above mentioned total protein lysates were 0.01, 0.1, 1.0, 10 and 20 ug/ml. (Page No:106)

Fig. 34. Modulation of SLE monocytes with reduced glutathione: Inhibition ELISA against antibodies in sera of patients with SLE on plates coated with total protein lysates obtained from SLE patient monocytes that were co-cultured for 24 hrs with 10 nM of reduced glutathione where the varying inhibitor concentrations used of the above mentioned total protein lysates were 0.01, 0.1, 1.0, 10 and 20 ug/ml. (Page No:107)

Fig. 35. Modulation of TB-SLE monocytes with reduced glutathione: Inhibition ELISA against antibodies in sera of patients with TB-SLE on plates coated with total protein lysates obtained from TB-SLE patient monocytes that were co-cultured for 24 hrs with 10 nM of reduced glutathione where the varying inhibitor concentrations used of the above mentioned total protein lysates were 0.01, 0.1, 1.0, 10 and 20 ug/ml. (Page No:108)

Fig. 36. Anti-TNF-α antibody Activity: Inhibition ELISA against monoclonal anti-TNF-α antibody on plates coated with total protein lysates obtained from human monocytes isolated from blood of TB patients where the inhibitor used were varying concentrations of the above mentioned total protein lysates obtained from monocytes of TB patients. The inhibitor concentrations were 0.01, 0.1, 1.0, 10 and 20 ug/ml. (Page No:109)

Fig. 37. Anti-TNF-α antibody activity: Inhibition ELISA against monoclonal anti-TNF-α antibody on plates coated with total protein lysates obtained from monocytes isolated from blood of SLE patients where the inhibitor used were varying concentrations of the above mentioned total protein lysates obtained from monocytes of SLE patients. The inhibitor concentrations were 0.01, 0.1, 1.0, 10 and 20 ug/ml. (Page No:110)
Fig. 38. Anti-TNF-α antibody activity: Inhibition ELISA against monoclonal anti-TNF-α antibody on plates coated with total protein lysates obtained from monocytes isolated from blood of TB-SLE patients where the inhibitor used were varying concentrations of the above mentioned total protein lysates obtained from monocytes of TB-SLE patients. The inhibitor concentrations were 0.01, 0.1, 1.0, 10 and 20 μg/ml. (Page No:111)

Fig. 39. Modulation of TB monocytes with reduced glutathione versus Anti-TNF-α antibody Activity: Inhibition ELISA against antibodies in sera of patients with TB on plates coated with total protein lysates obtained from TB patient monocytes that were co-cultured for 24 hrs with 10 nM of reduced glutathione where the varying inhibitor concentrations used of the above mentioned total protein lysates were 0.01, 0.1, 1.0, 10 and 20 μg/ml. (Page No:112)

Fig. 40. Modulation of SLE monocytes with reduced glutathione versus Anti-TNF-α antibody Activity: Inhibition ELISA against antibodies in sera of patients with SLE on plates coated with total protein lysates obtained from SLE patient monocytes that were co-cultured for 24 hrs with 10 nM of reduced glutathione where the varying inhibitor concentrations used of the above mentioned total protein lysates were 0.01, 0.1, 1.0, 10 and 20 μg/ml. (Page No:113)

Fig. 41. Modulation of TB-SLE monocytes with reduced glutathione versus Anti-TNF-α antibody Activity: Inhibition ELISA against antibodies in sera of patients with TB-SLE on plates coated with total Protein lysates obtained from TB-SLE patient monocytes that were co-cultured for 24 hrs with 10 nM of reduced glutathione where the varying inhibitor concentrations used of the above mentioned total protein lysates were 0.01, 0.1, 1.0, 10 and 20 μg/ml. (Page No:114)

Fig. 42. Expression of secreted pro-inflammatory cytokine TNF-α in supernatants of monocytes that were cultured for 24 hrs with and without 10 nM of reduced glutathione. The different monocytes cultured were obtained from blood of patients having TB, SLE and TB-SLE respectively. Also in each group, three categories were undertaken in this study. They were: Category I: Patients in the age group of 20 yrs to 35 yrs, Category II: patients in the age group of 20
yrs to 35 yrs and Category III: patients in the age group of 20 yrs to 35 yrs. (Page No:115)

Fig. 43. Inhibition ELISA against anti-DNA antibodies in sera of patients with SLE on plates coated with dsDNA obtained from SLE patient monocytes that were co-cultured for 24 hrs with without or with 10 mM of NAC, where the varying inhibitor concentrations used were 0, 0.01, 0.1, 5 and 10 μg/ml. (Page No:116)

Fig. 44. Inhibition ELISA against anti-DNA antibodies in sera of patients with SLE on plates coated with dsDNA obtained from SLE patient monocytes that were co-cultured for 24 hrs with without or with 5 μg/ml of EGCG, where the varying inhibitor concentrations used were 0, 0.01, 0.1, 5 and 10 μg/ml. (Page No:117)

Fig. 45. Inhibition ELISA against anti-DNA antibodies in sera of patients with SLE on plates coated with dsDNA obtained from SLE patient monocytes that were co-cultured for 24 hrs with without or with 100 μg/ml SN50 or 100 μg/ml SN50M, where the varying inhibitor concentrations used were 0, 0.01, 0.1, 5 and 10 μg/ml. (Page No:118)

Fig. 46. Inhibition ELISA against anti-DNA antibodies in sera of patients with SLE on plates coated with dsDNA obtained from TB patient monocytes that were co-cultured for 24 hrs with without or with 10 mM of NAC, where the varying inhibitor concentrations used were 0, 0.01, 0.1, 5 and 10 μg/ml. (Page No:119)

Fig. 47. Inhibition ELISA against anti-DNA antibodies in sera of patients with SLE on plates coated with dsDNA obtained from TB patient monocytes that were co-cultured for 24 hrs with without or with 5 μg/ml EGCG, where the varying inhibitor concentrations used were 0, 0.01, 0.1, 5 and 10 μg/ml. (Page No:120)

Fig. 48. Inhibition ELISA against anti-DNA antibodies in sera of patients with SLE on plates coated with dsDNA obtained from TB patient monocytes that were co-cultured for 24 hrs with without or with 100 μg/ml or 100 μg/ml SN50, where the varying inhibitor concentrations used were 0, 0.01, 0.1, 5 and 10 μg/ml. (Page No:121)
Fig. 49. Inhibition ELISA against anti-TB antibodies in sera of patients with TB on plates coated with dsDNA obtained from untreated SLE patient monocytes that were cultured for 24 hrs, where the varying inhibitor concentrations used were 0, 0.01, 0.1, 5 and 10 μg/ml.

Fig. 50. Inhibition ELISA against anti-TB antibodies in sera of patients with TB on plates coated with dsDNA obtained from SLE patient monocytes that were co-cultured for 24 hrs with either 10 mM NAC, 5 μg/ml EGCG, 100 μg/ml SN50 and 100 μg/ml SN50M. The varying inhibitor (above respective dsDNA from treated cells) concentrations used were 0, 0.01, 0.1, 5 and 10 μg/ml.