Introduction

Tuberculosis (TB) is a growing global health problem both in terms of disease burden and in terms of resistance to conventional chemotherapy ***(Hazbón et al., 2000).***

According to current estimates of the World Health Organization (WHO), 8 million cases of TB occur each year, resulting in 3 million deaths. Almost one-third of the earth's population are believed to be infected by the causative organism *Mycobacterium tuberculosis* (*M.TB*) ***(Portales- Pérez et al., 2002 and Bauman, 2004).***

Tuberculosis has reemerged in several industrialized countries. The cause of the global resurgence can be ascribed to the changes in the dynamics of the age–old battle between *M. tuberculosis* and the human host. Human immunodeficiency virus (HIV) infection is not the only contributing influence; Socioeconomic poverty, homelessness, overcrowding and malnutrition have historically been associated with TB infection ***(Joloba et al., 2000 and Tortora et al., 2004).***

The problem is compounded by the rising incidence of drug resistance and particularly the emergence of multidrug-resistant TB (MDR–TB).More than 50 million people may be already infected with MDR–TB which is associated with a mortality of 50% ***(Kam et al., 2002).***

Rifampicin (RIF) and Isoniazid (INH) are the cornerstone of short course chemotherapy regimens for TB, so the resistance of *M. tuberculosis* to these drugs is usually associated with resistance to all other first line agents ***(Grange, 2002).*** Early recognition and appropriate

treatment have been proven to be one of the most effective strategies to control MDR–TB ***(Hazbón et al., 2000)***.

Protective immunity against *M.TB* essentially depends on cell mediated immunity (CMI), There are several types of cells involved in the expression of cell mediated reactions of which T-lymphocytes and macrophages are the most important ***(Hertoghe et al., 2000).*** T cell function in protection against disease through activation of antimicrobial activities in macrophages by T-cell cytokines such as interferon γ (IFN γ), a major macrophage activating cytokines, tumour necrosis factor α (TNF-α) and interleukin 2 (IL-2), and other Th1 cytokines ***(Nirmala et al., 2001).***

A strict association exists between apoptosis and intracellular killing of mycobacteria, as evidenced by the finding that ATP–induced apoptosis is responsible for inhibition of Bacillus Calmette Guerin (BCG) multiplication in monocytes ***(Santucci et al., 2000).***

During active tuberculosis, exposure of T cells to *M. tuberculosis* in situ at a sites of active M. tuberculosis replication may lead to T cell activation and prime *M. tuberculosis*-responsive T cell to die through apoptotic mechanisms. Loss of *M. tuberculosis* reactive T cells throughout apoptosis might play a role in limiting the cellular response in this infection ***(Pearl et al., 2000 & Worku and Hoft, 2003).***