# CHAPTER I

## INTRODUCTION

Epstein-Barr virus (EBV) is a ubiquitous virus infecting more than 90% of adult people worldwide. EBV is transmitted mainly via oropharyngeal secretion and preferentially infects nasopharyngeal epithelial cells (1). The virus will also invade B-lymphocytes and stay in latency state in the B cells for life. Even though the virus is well control and causes only mild disease in immunocompetent hosts (2), it can be associated with more severe disease, such as post-transplant lymphoproliferative disease (PTLD) (3) and T cell lymphoma in immunocompromised host (4). In relatively immunocompromised HIV-infected patients, for example, EBV could cause a number of malignancies such as Burkitt's lymphoma (5) and primary central nervous system lymphoma (PCNSL) (2).

The cytotoxic T lymphocytes were believed to be a protective agent against viral infection for many years. The protective role of EBV-specific cytotoxic T lymphocyte was also study and reported by several scientists in many ways including in vivo, animal model and human. The evidence ,for example, was done by Rickinson et al 1984 who found that cell mediated immune processes play a pivotal role in controlling the number of EBV infected B cells (6). Some of the early *in vitro* experiments demonstrated that T cells inhibit the proliferation of EBV infected B cells (7). In human many groups have reported the role of EBV specific CTL in decreasing EBV viral load (8, 9), tumour regression (10, 11) and therapy (12, 13). Moreover EBV-DNA together with EBV-specific $CD8^+$ T cells was recently used in preemptive and therapeutic treatment of many diseases such as Hodgkin's disease (14-16), NPC (15) and PTLD (16).

In the present study, we observed the relationship between EBV-specific CTL response and EBV-DNA of HIV patients in our study to elucidate the role of EBV-specific CTL in EBV pathogenesis.We were also developed quantitative real-time PCR technique for quantitate EBV load in peripheral blood mononuclear cell (PBMC) of healthy donors and HIV infected patients. We found EBNA-3 family was the immunodominant protein. In addition, we also demonstrated the inverse correlation trend of EBV-specific T cell responses

and EBV-DNA in HIV-infected patients which might support the role of EBV-specific CTL in controlling EBV infection.