Abstract

Cervical cancer is the second most common cause of cancer mortality among women worldwide (Franco et al, 2003). Epidemiological studies have shown a strong link between human papilloma virus (HPV) infection and the development of cervical cancer (Franco et al, carcinogenic process (Chan et al, 2005). Most HPV infections are transient and regress spontaneously and only a minority of women develops persistent infection that with time may evolve into cervical intraepithelial neoplasia and/or progress to invasive cervical cancer(Villa, 1997; Franco et al, 1999). Given that host immune response to HPV is thought to be an important determinant of HPV acquisition and progression to high-grade cervical lesions and cancer, it is plausible that human leucocyte antigen (HLA) variations may affect pathogenesis of cervical neoplasia (Beskow et al, 2005; Clerici et al, 1997; Hildesheim et al, 1997). The major histocompatibility complex is a highly polymorphic gene cluster on the short arm of chromosome six. The genes in this cluster are divided into three classes with different roles in immune responses. HLA gene polymorphisms result in variations in peptide-binding cleft, therefore influencing the antigens bound and presented to T cells (Beskow et al, 2005; Wang e al, 2005). The HLA class I genes (HLAA, -B, and –C) present foreign antigens to CD8+ Cytotoxic T lymphocytes, while class II genes (HLA-DR, - DQ and –DP) present antigenic peptides to CD4+ T helper cells and are important in host immune responses to viruses and other pathogens (Wang et al, 2001).