

Abstract

Cervical cancer is an important global public health problem and a common cause of death among women, and it is attributable to human papillomavirus (HPV) (Walboomers et al, 1999; Parkin et al, 2008). In a large series of invasive cervical cancer from around the world, HPV-DNA was detected in 99.7% of the tumors, leading to the conclusion that HPV was a necessary cause of cervical cancer (Bosch et al, 1995; Walboomers et al, 1999; Bosch et al, 2007). The identification of HPV's role in cervical cancer has led to important advances in primary prevention through vaccination and diagnosis through HPV detection (Stanley et al, 2008; Bosch et al, 2008). However, tangible reduction in the incidence of cervical cancer and the impact on global public health will probably take decades. As HPV types are divergent, efficacy of current vaccines is type-restricted, and therefore development of the next generation of HPV vaccines will require inclusion of relevant antigens from several HPV types (Lowy, 2008). Geographical profiling of HPV type distribution will be important in making vaccines more relevant for target populations. Most women will be infected with HPV sometime in their lifetime. Results from large meta-analyses studies indicate that at any given point in time, 10.4% (95% confidence interval (CI) 10.2-10.7) of women worldwide are positive for cervical HPV DNA (Bosch et al, 2008). The prevalence of HPV is higher in less developed regions (13.4%; 95% CI: 13.1-13.7) than in the more developed regions (8.4%; 95% CI: 8.3-8.6) (Bosch et al, 2008). The same studies indicate that African women at 22.1% (95% CI: 20.9-23.4) and East African women in particular, have the highest HPV prevalence rates (31.6%; 95% CI: 29.5-33.8) (Bosch et al, 2008). HPV type 16 is the most common in all continents, with an estimated point prevalence of 2.6% (95% CI: 2.5-2.8) worldwide, and HPV type 18 the second most frequently detected type (Clifford et al, 2005). Regional differences are thought to be related to geographical and immunogenetic factors, such as defects in cellular immunity through chronic cervical inflammation, malnutrition and more recently, HIV infection; Type 16 though appears to be less influenced by immune impairment than other types (Clifford et al, 2005). Although many women get infected with HPV, most do not develop cervical cancer. Several co-factors are postulated to influence the disease process. The potential co-factors include exogenous factors such as tobacco smoking, hormonal contraceptives, and coinfections with other sexually transmitted infections (Munoz et al, 2006). In addition, viral co-factors, such as specific HPV types, viral load, and viral integration, as well as host co-factors such as endogenous hormones, genetic factors, and factors related to the immune response may variably influence the course of HPV infection (Munoz et al, 2006). Women with HIV infection have been shown to be more likely not only to have a concurrent HPV infection but also to have an increased risk for a high grade cervical squamous intraepithelial lesion (La Ruche et al, 1998; Temmerman et al, 1999; Womack et al, 2000; Baay et al, 2004; Hawes et al, 2006; Didelot-Rousseau et al, 2006; Ngándwe et al, 2007). HPV is the commonest sexually transmitted infection, with more than 75% of sexually active adults acquiring one or more genotypes in their lifetime (Bosch et al, 2008). However, by age 30 years, most women clear the infection due to an effective cell-mediated immune response, and only a small number thereafter are diagnosed with a HPV-associated lesion (Schiffman, 1992). It is thought that it is through its effect on CD4+ cells and regulation of immune responses to a variety of antigens that HIV attenuates the systemic response to HPV (Palefsky, 2006). The prevalence of HIV among adult Kenyan women was 13% in 2003 with trends reported to have decreased to 5.1% by 2006 (KDHS, 2003). The high prevalence of HIV may increase the incidence of cervical pre-cancer and potentially, of cervical cancer. Gichangi et al (2002),

however, demonstrated that a two to three-fold increase in HIV prevalence did not translate to a proportionate increment in incidence of cervical cancer. They hypothesized that HIV-infected women die from HIV-related opportunistic infections before they develop invasive cervical cancer. The mean survival time for women with HIV in 2008 was reported to be 5 years (Yamada et al, 2008) while typically more than 10 years elapse before the development of cervical cancer after HPV infection. Yamada et al (2008) also advanced the possibility that sub-clinical cervical cancer may be missed in many women dying prematurely from AIDS-related opportunistic infections. This study was carried out to establish whether the coinfection of HIV and HPV has an influence on HPV genotype distribution and on the prevalence and grade of cervical neoplasia.