To the Editor—In a recent article, Tracy et al [1] use mathematical modeling to predict the potential impact of human papillomavirus (HPV) vaccination programs in developing countries; HPV is the primary causative agent for cervical cancer. The analysis is timely, given the recent announcement that Rwanda will soon launch Africa’s first HPV vaccination program [2]. Tracy and colleagues focused on challenges associated with HPV vaccination in Mali (eg, female circumcision, marriage at younger ages, polygamy, cultural and economic factors), but they did not assess the potential effect of human immunodeficiency virus (HIV) on HPV vaccination programs.

Rwanda and many other countries in Sub-Saharan Africa have a significant

Human Papillomavirus Vaccination Programs and Human Immunodeficiency Virus Epidemics
The prevalence of high-risk HPV subtypes increases a woman’s susceptibility to HPV infection, boosts the chances that infection is from high-risk subtypes [3] (ie, those that cause cervical cancer), hinders the clearance of HPV infection, and accelerates progression to cervical cancer [4]. Consequently, the effect of HPV vaccination programs on reducing the size of the population at risk for developing cervical cancer will depend on the prevalence of HIV.

To illustrate the potential effect of HPV vaccination programs in countries with HIV epidemics, we used a published model of HPV vaccination [5] and modified the values of its parameters to reflect the influence of HIV. The efficacy of the HPV vaccine is ~100%, and effectiveness is equivalent to the prevalence of the high-risk HPV subtypes it protects against (subtypes 16 and 18). The prevalence of these subtypes is 66% in HIV-positive women [3] and 5% in HIV-negative women [5]. Addressing the effect of HIV on HPV clearance, we note that in most HIV-negative women (~80%) HPV is cleared within 1 year [5], whereas few HIV-positive women ever achieve HPV clearance and if they do it takes considerably longer than a year. Based on unpublished data from Botswana, we assume that the clearance rate for HPV in HIV-positive women is 10% per year. With these modified parameter values, the model [5] can be used to predict the potential impact of HPV vaccination programs in reducing the size of the population at risk for developing cervical cancer as a function of HIV prevalence and vaccine coverage (Figure 1).

Figure 1 shows the potential impact of HPV vaccination programs in Sub-Saharan African countries, where HIV prevalence ranges from low (eg, 2% in Mali and 4% in Rwanda) to high (eg, ~30% in Botswana). If coverage is high (eg, 80%) the reduction in the size of the population at risk for developing cervical cancer is between 15% and 26% (Figure 1). However, if coverage is low (eg, 20%), the reduction is only 4%–6.5%. Notably, the higher the prevalence of HIV (for any given vaccination coverage) the greater the reduction in the size of the at-risk population. For example, achieving a 15% reduction in the at-risk population would require 80% vaccine coverage if HIV prevalence is ~2%, as in Mali, but only 46% coverage if HIV prevalence is ~30% as in Botswana. Therefore vaccinating even a small proportion of women in countries with severe HIV epidemics could substantially reduce the number of women at risk for developing cervical cancer.

Figure 1. Impact of human immunodeficiency virus (HIV) epidemics on the effectiveness of human papillomavirus vaccination programs in reducing the number of women at risk of developing cervical cancer; HIV prevalence is plotted versus coverage, and curves show the percentage reduction in the number of women at risk (numbers on right axis).

Notes

Thanks to Dr Diana Dickinson, MB ChB, and staff at Independence Clinic, Gaborone, Botswana for access to clinical data, and Iqbal Chand at Diagnofirm Laboratory, Gaborone, Botswana for providing pathology reports.

Financial support. This work was supported by the National Institutes of Health [RO1 AI041935] and the National Institute of Allergy and Infectious Diseases [R21AI086701].

Potential conflicts of interest. All authors: No reported conflicts.

All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

Seema Yasmin, David J. Gerberry, and Sally Blower
Center for Biomedical Modeling, Semel Institute of Neuroscience and Human Behavior, David Geffen School of Medicine, University of California, Los Angeles, California

References


Clinical Infectious Diseases 2011;53(8):845–846

Correspondence: Seema Yasmin, BSc, MB BChir, Center for Biomedical Modeling, Semel Institute of Neuroscience and Human Behavior, David Geffen School of Medicine, University of California, Los Angeles, CA 90024 (seemayasmin@cantab.net).

Clinical infectious diseases are on behalf of the Infectious Diseases Society of America. All rights reserved. For Permissions, please e-mail: journals.permissions@oup.com.

1058-4838/2011/538-0016$14.00

DOI: 10.1093/cid/cir514