Publisher: CDC; Journal: Emerging Infectious Diseases
Article Type: Research; Volume: 13; Issue: 1; Year: 2007; Article ID: 06-0438
DOI: 10.3201/eid1301.060438; TOC Head: Research

Appendix[1]

[1]Refer to the Appendix References, below, for citations in this Appendix.

Demographic Model

The demographic model stratifies the population by gender and 17 age groups (12–14, 15–17, 18–19, 20–24, 25–29, 30–34, 35–39, 40–44, 45–49, 50–54, 55–59, 60–64, 65–69, 70–74, 75–79, 80–84, and ≥85 years). This age grouping permits age-specific inputs for patterns of sexual activity and cervical cancer screening and allows for age-specific outputs such as rates of cervical human papillomavirus (HPV) disease among girls and women, and genital warts among both males and females. Similar age groupings have been used by other sexually transmitted disease models (*1,2*). We further stratified each age group into 3 sexual activity groups (high, medium, low). We defined sexual activity according to the rates of sex partner change per year: low (0–1 per year), medium (2–4 per year), and high (≥5 per year). The number and the initial distribution of new entrants into the population by each gender were chosen to satisfy the Lotka characteristic equation with zero population growth (*3*). This allowed for variation in results across strategies to primarily be due to epidemiologic and program model features and not to changes in the demographic characteristics of the population over time (*3*).

The model starts with 12-year-olds entering the population at a gender-specific and sexual activity–specific rate, and transfers persons between successive age groups at an age- and gender-specific rate per year. The transfer rate depends on the rate of population growth, age- and gender-specific per capita mortality rate, and the number of years within an age group (*3*). We assumed equilibrium in the age distribution with zero population growth.

We set the population size in the model to 100,000 persons divided equally between females and males. Death rates for males and for females without cervical cancer were obtained from Vital Statistics data on gender- and age-specific mortality rates across all races for 2002 (*4*). Death rates among adolescent girls and women with cervical cancer were obtained from Surveillance Epidemiology and End Results (SEER)

Program data for 1997–2002 (*5*). Other demographic data were obtained from US Vital Statistics and the 2000 Census (*4,6*).

Epidemiologic Model

The epidemiologic model simulates HPV infection and occurrence of HPV disease (cervical intraepithelial neoplasia [CIN], cervical cancer, and genital warts) in the population. The acquisition of infection and progression from infection to disease follow a similar natural history structure, as assumed in previous models for HPV 16 and 18 (*7*). Building on these previous models, we also incorporated HPV 6 and 11 infection and genital warts and modeled infection by using 3 groups of HPV types (HPV 16/18, HPV 6/11, or HPV 6/11/16/18).

To simulate the occurrence of CIN, genital warts, and cervical cancer among those infected with HPV, we divided the population into distinct epidemiologic categories, according to the population's susceptibility to infection or the population's status with respect to infection, disease, screening, and treatment. These categories were similar to what has previously been defined in other models (*7*). The following, along with Figure 1, describes the movement of the population through these categories.

HPV Infection: Acquisition and Transmission

The epidemiologic model begins with 12-year-olds entering into the susceptible category *X*. Susceptible persons acquire HPV infection with a given type (HPV 16/18 infected only, HPV 6/11 infected only, or HPV 6/11 and HPV 16/18 infected) at a rate dependent upon gender, sexual activity group, age, and time. The rate at which persons of a given gender, sexual activity group, and age class at a given time acquire infection with a certain type (per capita force of infection) depends on the number of sexual partnerships and how these persons form partnerships with persons of the opposite sex, the fraction of infected sex partners, and the transmission probability per partnership. The formation of sexual partnerships is governed by a conditional probability sexual mixing matrix. Each cell in the mixing matrix represents the probability of a person of a given gender, sexual activity group, and age class having a sexual activity group, age-class specific partner from the opposite gender. In generating the mixing matrix, we used 2 parameters to depict the degree of mixing between age and sexual activity groups. This strategy

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allowed us to represent a wide range of mixing patterns in the matrix, from fully assortative (as for persons with like persons when parameter is zero) to proportionate (random partners when parameter is 1) mixing (*1,2,8,9*). The baseline parameter values for the rate of sexual partner change, stratified by gender, sexual activity, and age, were calculated by using data from the National Health and Social Life Survey (*10*) and methods outlined in Garnett and Anderson (*2*) (Appendix Table 1).

Once HPV transmission occurs, susceptible persons enter the category of infected persons, *Y*. Persons leave this category when the infectious period for HPV ends and enter the category of recovered persons with a fixed duration of immunity, *Z*. In the base case, we assumed that duration of natural immunity is lifelong. Unvaccinated infected persons clear infection at a type-specific per capita rate. Persons in the immune (*Z*) category who are susceptible to only 1 type can be infected with that type and move to another infected/immune category, *U*.

A fraction of susceptible persons are vaccinated and move into the vaccination category *V*. The movement of those vaccinated through the model is similar to the movement of those unvaccinated, shown in Figure 1A. The remaining fraction of persons who are not vaccinated remains in the susceptible category *X*. The vaccine-induced immunity of those in the vaccinated category may wane over time. As a result, persons can eventually move to the susceptible category *S* at an age- and gender-dependent rate. We assumed that when a person loses vaccine-derived immunity, he or she becomes susceptible to infection with any of the types. In the base case, the duration of vaccine-derived immunity is assumed to be lifelong. Vaccinated persons can also experience a breakthrough infection and enter the category of infectious persons, *W,* at a per capita rate that depends on the degree of protection offered by the vaccine. Vaccinated persons can recover from an HPV infection at an age- and gender-specific rate by a factor that is different from the recovery rate for unvaccinated infected persons. Vaccinated persons then move to a category with fixed duration of immunity, *Q*. Persons in this category who are susceptible to 1 type can be infected with that type and move to another vaccinated infected/immune category, *P*.

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No epidemiologic studies have estimated the probability of HPV infection transmission per partnership and by type. We assumed that this probability is higher for transmission from males to females (0.8) than that for transmission from females to males (0.7) (*12–15*). Using data on participants in the placebo arm of Merck's HPV vaccine clinical trials, we estimated mean duration of HPV infection before progression to CIN, or regression, at 1.2 years for HPV 16/18 and 0.7 years for HPV 6/11 (R. Insinga, unpub. data).

CIN, Cervical Cancer, and Genital Warts

CIN develops in infected girls and women at a specified rate and moves to the HPV disease categories of the model (Figure 1B). Several categories represent the true histologic health status of a woman: CIN grade 1 (CIN 1), CIN grade 2 (CIN 2), CIN grade 3 (CIN 3), localized cervical cancer (LCC), regional cervical cancer (RCC), distant cervical cancer (DCC), and cervical cancer survivors who are free from cancer. Women with CIN and cancer were further classified into undetected, detected, or treated categories. Two additional absorbing categories are for women who are no longer at risk for cervical cancer (*16*). These include the following: 1) women who have had a benign hysterectomy for reasons other than cervical cancer (at an age-specific rate) and 2) women treated and cured for cervical cancer. Finally, infection with the low-risk type can result in genital warts in females and males and move to the genital warts category, *GW* (*17*). We assumed women with benign hysterectomies can be infected and are at risk for genital warts (*18*). Women and men recovering from genital warts move to category *Z*.

We assumed all progression and regression rates to HPV and cancer states to be independent of age (*19–23*). Annual transition rates from HPV infection to clinically detectable CIN were calculated from studies by Winer et al. (*17*) and Insinga (R. Insinga, unpub. data). Several published reports were also used to estimate annual rates of CIN regression and progression to cervical cancer (*24–31*) (Merck, unpub. data). Incidence and regression rates for genital warts were obtained from Winer et al. (*17*) (Appendix Table 2). Hysterectomy rates; cervical cancer screening coverage, sensitivity, and specificity; and treatment efficacy were derived from several published studies (*32–40*) (Appendix Table 3).

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Economic Parameters

All model costs were updated to 2005 US dollars by using the medical care component of the Consumer Price Index (*41*). The direct medical costs for screening and treatment for CIN, genital warts, and cervical cancer were based on administrative claims data and other sources (*42–44*). We measured the cost of cytology screening per unit time as the product of the cost per test, the test compliance rate, the frequency of administering the test per unit time, and the size of the unidentified population that is eligible for screening. We estimated the cost of following up on false-positive results of the cytology test as a function of the specificities of the cytology test and colposcopy procedure and the costs of colposcopy and biopsy. The cost of the HPV vaccine for 3 doses was assumed to be $360, which was consistent with HPV vaccination costs used in previous cost-effectiveness analyses (*7*). Productivity losses as a result of HPV disease or death were not included in the analyses (*45*).

Quality adjusted life years (QALYs) were measured by weighting survival time by the quality-of-life adjustment weights associated with each health state and integrating the sum of adjusted time in all these health states over the planning horizon. We measured survival time as the total number of years spent alive by the active population during a given period. The health utility values used to estimate QALYs were derived from various sources (*46–48*). Health utility values for diagnosed invasive cancer states were estimated by Myers et al. (*47*) at 0.76 for localized cancer and 0.67 for regional cancer; these values were derived from Gold et al. at 0.48 for distant cancer (*46*). We assumed that the quality of life for cervical cancer survivors after successful treatment would continue to be lower (0.76) than that of healthy women (*49,50*). Diagnosed and treated CIN 1 and CIN 2/3 states were assumed to have quality weights of 0.91 and 0.87, respectively (*47,48*). We assumed the quality weight for genital warts to be 0.91 (*47*) (Appendix Table 4).

Undiagnosed and asymptomatic HPV, CIN, and cancer states and successfully treated CIN states were assumed to have a quality-of-life weight similar to those of persons without these conditions. Gender- and *46*age-specific quality weights for non-HPV disease states were also derived from Gold et al. (). Time in these states was

multiplied by the age- and gender-specific weights to reflect the variation of quality of life by age and gender groups. We assumed that quality of life did not vary by sexual activity groups. Finally, all costs and effects were discounted to present value at a rate of 3%.

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Appendix Table 1. Baseline behavioral parameter values for the sexually active population*

	Proportion of population, %		
Activity group	Male	Female	Relative partner acquisition rate
1 (highest)	2.56	2.56	11.29
2	11.47	11.47	2.96
3 (lowest)	85.97	85.97	1.0
Age group, y	Relative partner acquisition rate	Overall mean partner acquisition rate	
12–14	0.11	0.1	
15–17	1.18	0.3	
18–19	2.42	1.3	
20–24	2.61		
25–29	2.55		
30–34	1.72		
35–39	1.65		
40–44	1.53		
45–49	1.38		
50–54	1.25		
55–59	1.00		
60–69	0.61	0.5	
≥70	0.44		

*Sources: Lauman et al. (*10*), Abma and Sonenstein (*11*).

Appendix Table 2. Baseline biologic parameter values for HPV disease categories*

Parameter	Base-case estimate	Source†

Progression in the presence of HPV 16/18 per year, %		
Normal to CIN 1	9.4	(RI)
Normal to CIN 1 to CIN 2	5.8	(*17*,RI)
Normal to CIN 1 to CIN 2 to CIN 3	3.5	(*17*,RI)
CIN 1 to CIN 2	13.6	(MRK)
CIN 2 to CIN 3 (severe dysplasia)	14.0	(*26,27*)
CIN 3 - severe dysplasia to CIN 3 - CIS 1	42.0	(*26,28*)
CIS 1 to CIS 2	5.0	
CIS 2 to LCC	18.0	
LCC to RCC	10.0	(*16,24,25,31*)
RCC to DCC	30.0	(*16*)
Progression in the presence of HPV 6/11 per year, %		
Normal to CIN 1	9.5	(RI)
Normal to CIN 1 to CIN 2	1.9	(RI)
Normal to CIN 1 to CIN 2 to CIN 3	0.0	(RI)
CIN 1 to CIN 2	0.0	(MRK)
Normal to genital warts	57	(*17*)
Mean duration of acute HPV infection, y		
HPV 16/18 infection	1.2	(RI)
HPV 6/11 infection	0.7	(RI)
Regression of HPV 16/18+ disease per year, %		
CIN 1 to normal/HPV	32.9	(MRK,*29*)
CIN 2 to normal/HPV	21.0	(*26,27,30*)
CIN 2 to CIN 1	13.3	(*27*)
CIN 3 (severe dysplasia) to normal/HPV	11.0	(*26*)
CIN 3 (severe dysplasia) to CIN 1	3.0	(*26,27*)
CIN 3 (severe dysplasia) to CIN 2	3.0	(*26,27*)
Regression of HPV 6/11+ disease per year, %		
CIN 1 to normal/HPV	55.2	(MRK)
Genital warts to normal/HPV	87.5	(*17*)
Age (y) and stage-specific cervical cancer mortality rates per year, 1997–2002, %		(*5*)
For LCC		
15–29	0.7	
30–39	0.6	
40–49	0.8	
50–59	1.9	
60–69	4.2	
≥70	11.6	
For RCC		
15–29	13.4	
30–39	8.9	
40–49	11.0	
50–59	10.1	
60–69	17.6	
≥70	28.6	
For DCC		
15–29	42.9	
30–39	41.0	
40–49	46.7	
50–59	52.7	
60–69	54.6	
≥70	70.3	

*HPV, human papillomavirus; CIN, cervical intraepithelial neoplasia; CIS, carcinoma in situ; LCC, localized cervical cancer; RCC, regional cervical cancer; DCC, distant cervical cancer.
†RI, R. Insinga, unpub. data; MRK, Merck, unpub. data.

Appendix Table 3. Hysterectomy, screening, and treatment parameters*

Parameter	Base-case	Source

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	estimate	
Hysterectomy rate, % per year		(*32*)
15–24 y	0.02	
25–29 y	0.26	
30–34 y	0.53	
35–39 y	0.89	
40–44 y	1.17	
45–54 y	0.99	
≥55 y	0.36	
Cervical cytology screening, excluding those with hysterectomy, % per year		(*33*)
10–14 y	0.6	
15–19 y	21.0	
20–24 y	44.8	
25–29 y	61.6	
30–34 y	54.9	
35–39 y	50.5	
40–44 y	48.1	
45–49 y	49.1	
50–54 y	51.1	
55–59 y	46.7	
60–64 y	42.5	
65–69 y	38.9	
70–74 y	29.6	
75–79 y	20.1	
80–84 y	11.1	
≥85	5.5	
Females never screened, %	5.0	
Liquid-based cytology specificity, %	94	(*34,35*)
Colposcopy sensitivity, %	96	(*36*)
Colposcopy specificity, %	48	(*36*)
GW patients seeking physician care, %	75	(*37*)
Symptom development, % per year		Assumed
Localized cervical cancer	4	
Regional cervical cancer	18	
Distant cervical cancer	90	
Eradication with treatment, %		
For CIN 1	96	(*38*)
For CIN 2	92	(*38*)
For CIN 3, CIS	92	(*38*)
For localized cervical cancer	92	(*39*)
For regional cervical cancer	55	(*39*)
For distant cervical cancer	17	(*39*)
Persistence of HPV after treatment for CIN or GW, %	34	(*40*)

*HPV, human papillomavirus; GW, genital warts; CIN, cervical intraepithelial neoplasia; CIS, carcinoma in situ.

Appendix Table 4. Cost and quality-of-life parameters*

Parameter	Base-case estimate	Source
Costs of diagnosing and treating HPV disease		*(42–44)*
Genital warts	$489	
Liquid-based cytology screening	$99	
Colposcopy and biopsy	$318	
CIN 1	$1,554	
CIN 2/3, CIS	$3,483	
Localized cervical cancer	$26,470	

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Regional cervical cancer	$28,330		
Distant cervical cancer	$45,376		
Quality-of-life weights (0–1 scale)			
CIN 1	0.91		*(47)*
CIN 2/3, CIS	0.87		*(47)*
Localized cervical cancer	0.76		*(47)*
Regional cervical cancer	0.67		*(47)*
Distant cervical cancer	0.48		*(46)*
Cervical cancer survivor	0.84		*(47,49,50)*
Genital warts	0.91		*(47)*
No condition	F	M	*(46)*
12–17 y	0.93	0.93	
18–34 y	0.91	0.92	
35–44 y	0.89	0.90	
45–54 y	0.86	0.87	
55–64 y	0.80	0.81	
65–74 y	0.78	0.76	
≥75 y	0.70	0.69	

*HPV, human papillomavirus; CIN, cervical intraepithelial neoplasia; CIS, carcinoma in situ; F, females; M, males.