Beyond Hetero

The Impact of Disregarding Non-Heterosexual Partnerships in Mathematical Models of Human Papillomavirus

Caleb Easterly[1], Fernando Alarid-Escudero, PhD[1], Shalini Kulasingam, PhD[2], Eva Enns, PhD[1]

[1]Division of Health Policy and Management
University of Minnesota School of Public Health

[2]Division of Epidemiology
University of Minnesota School of Public Health

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Epidemiology of HPV

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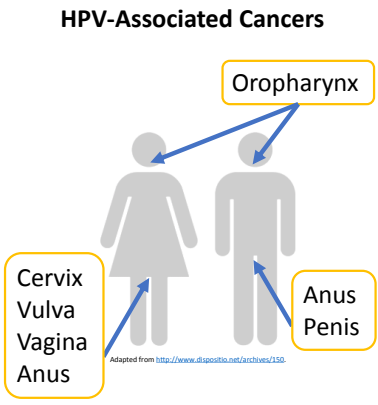
- High-risk HPV causes cancer at several anatomical sites



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  - other evidence

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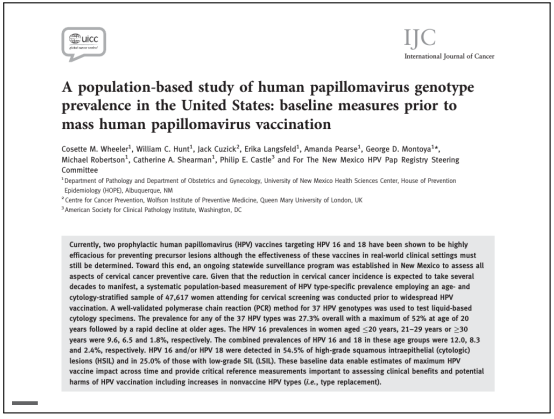
- Match population to calibration targets



UICC

IJC
International Journal of Cancer

A population-based study of human papillomavirus genotype prevalence in the United States: baseline measures prior to mass human papillomavirus vaccination

Cosette M. Wheeler[1], William C. Hunt[1], Jack Cuzick[2], Erika Langsfeld[1], Amanda Pearse[1], George D. Montoya[1]*, Michael Robertson[1], Catherine A. Shearman[1], Philip E. Castle[3] and For The New Mexico HPV Pap Registry Steering Committee

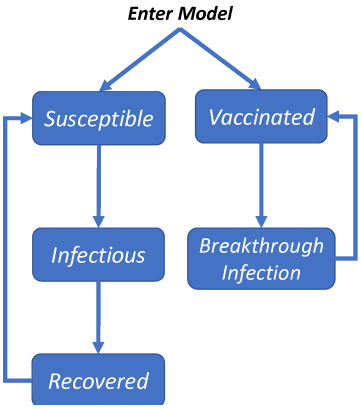
[1]Department of Pathology and Department of Obstetrics and Gynecology, University of New Mexico Health Sciences Center, House of Prevention Epidemiology (HOPE), Albuquerque, NM

[2]Centre for Cancer Prevention, Wolfson Institute of Preventive Medicine, Queen Mary University of London, UK

[3]American Society for Clinical Pathology Institute, Washington, DC

Currently, two prophylactic human papillomavirus (HPV) vaccines targeting HPV 16 and 18 have been shown to be highly efficacious for preventing precursor lesions although the effectiveness of these vaccines in real-world clinical settings must still be determined. Toward this end, an ongoing statewide surveillance program was established in New Mexico to assess all aspects of cervical cancer preventive care. Given that the reduction in cervical cancer incidence is expected to take several decades to manifest, a systematic population-based measurement of HPV type-specific prevalence employing an age- and cytology-stratified sample of 47,617 women attending for cervical screening was conducted prior to widespread HPV vaccination. A well-validated polymerase chain reaction (PCR) method for 37 HPV genotypes was used to test liquid-based cytology specimens. The prevalence for any of the 37 HPV types was 27.3% overall with a maximum of 52% at age of 20 years followed by a rapid decline at older ages. The HPV 16 prevalences in women aged ≤20 years, 21–29 years or ≥30 years were 9.6, 6.5 and 1.8%, respectively. The combined prevalences of HPV 16 and 18 in these age groups were 12.0, 8.3 and 2.4%, respectively. HPV 16 and/or HPV 18 were detected in 54.5% of high-grade squamous intraepithelial (cytologic) lesions (HSIL) and in 25.0% of those with low-grade SIL (LSIL). These baseline data enable estimates of maximum HPV vaccine impact across time and provide critical reference measurements important to assessing clinical benefits and potential harms of HPV vaccination including increases in nonvaccine HPV types (i.e., type replacement).

Wheeler, et al. 2013



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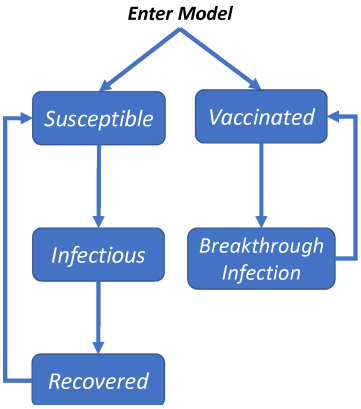

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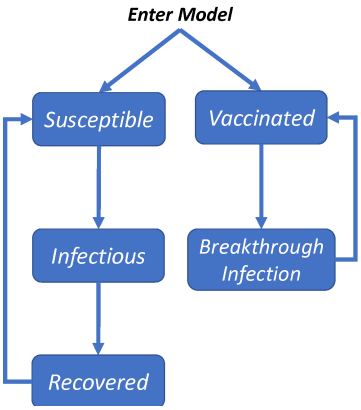

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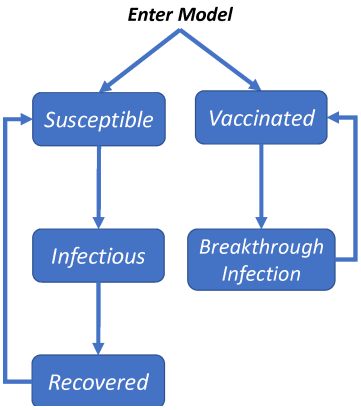

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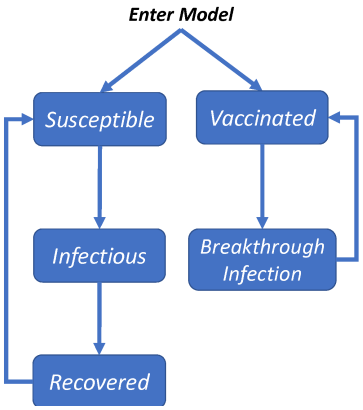

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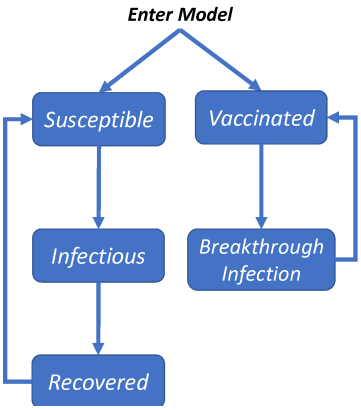

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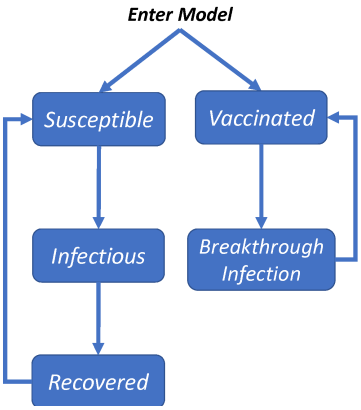

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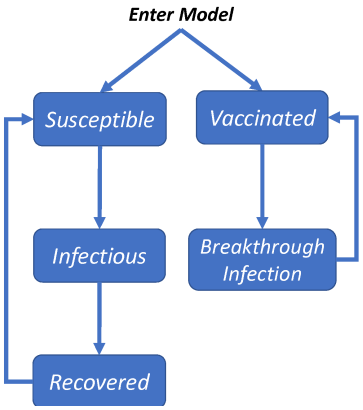

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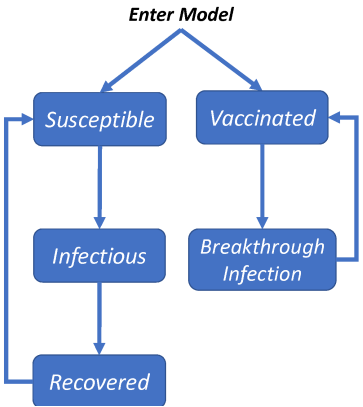

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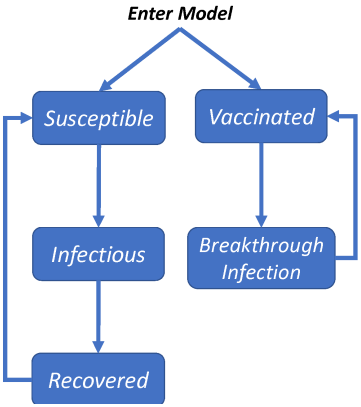

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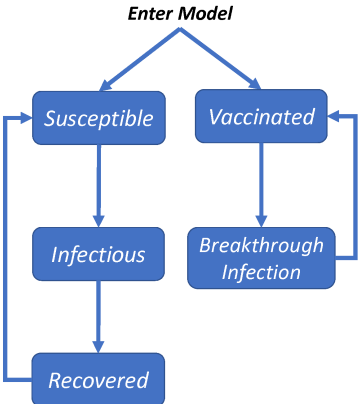

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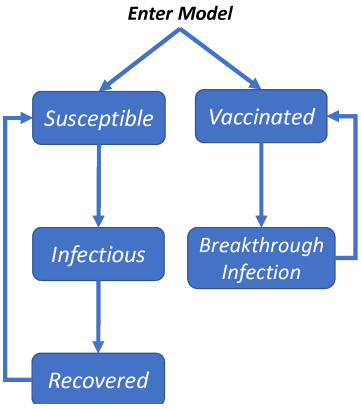

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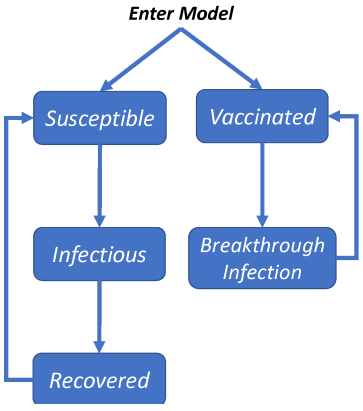

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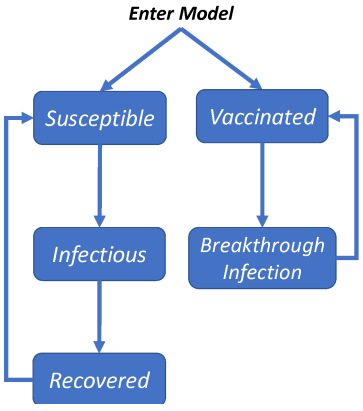

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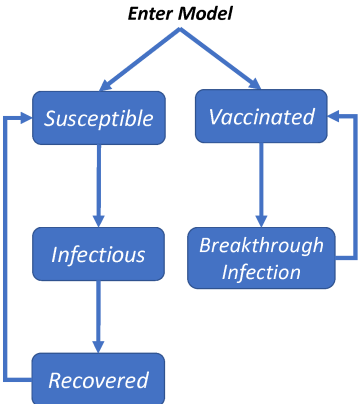

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- Number of infections prevented by male vaccination, over 20 years

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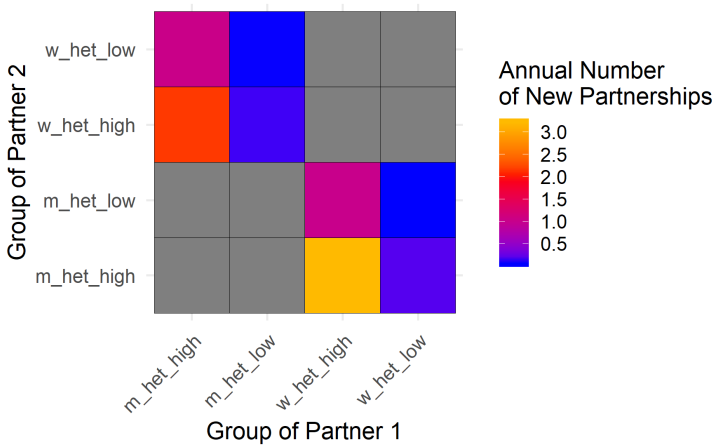

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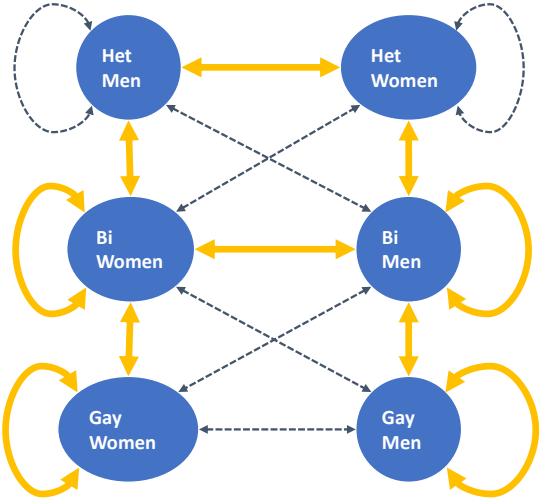

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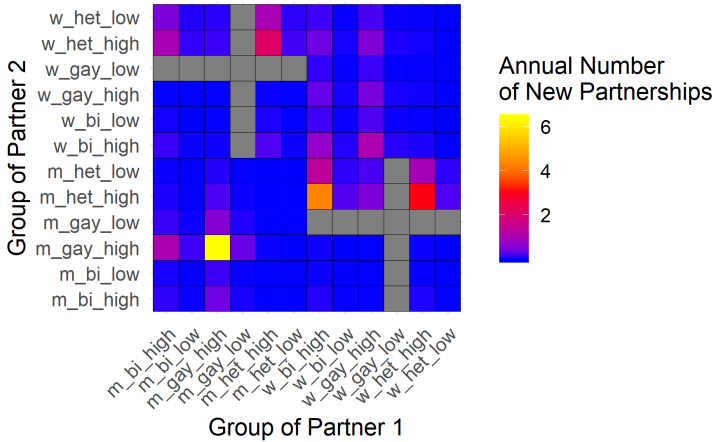
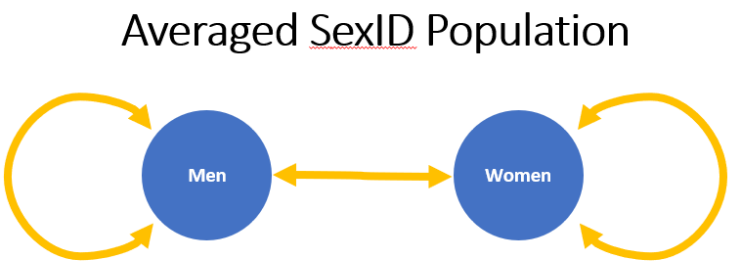
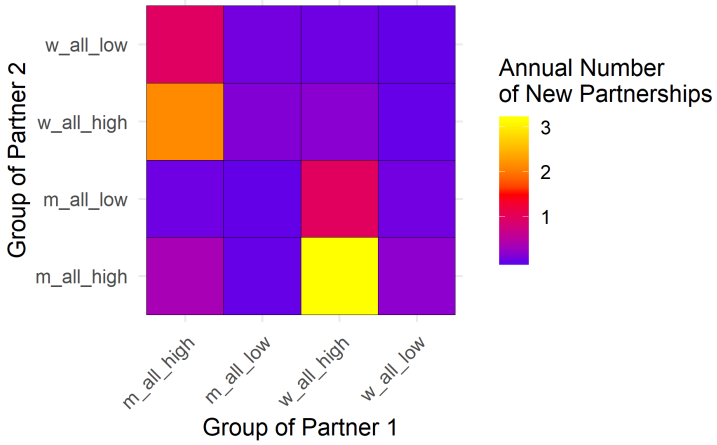
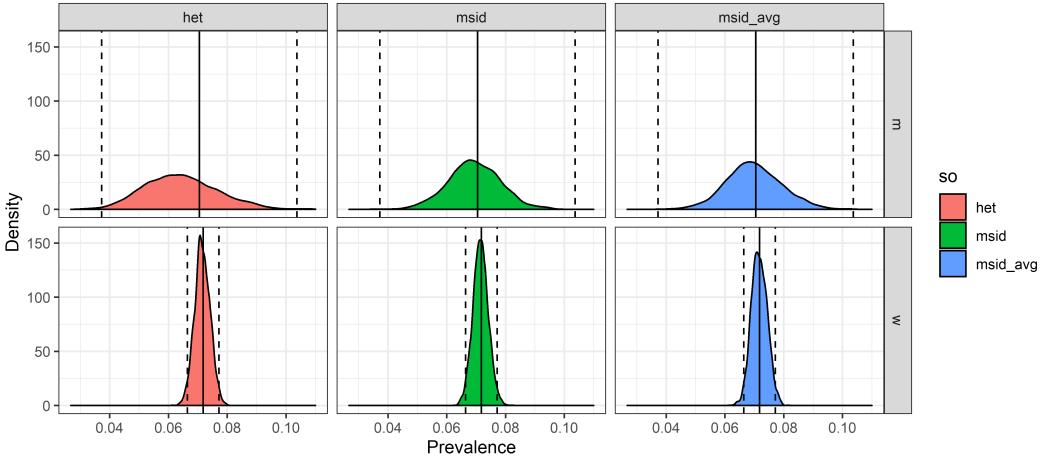
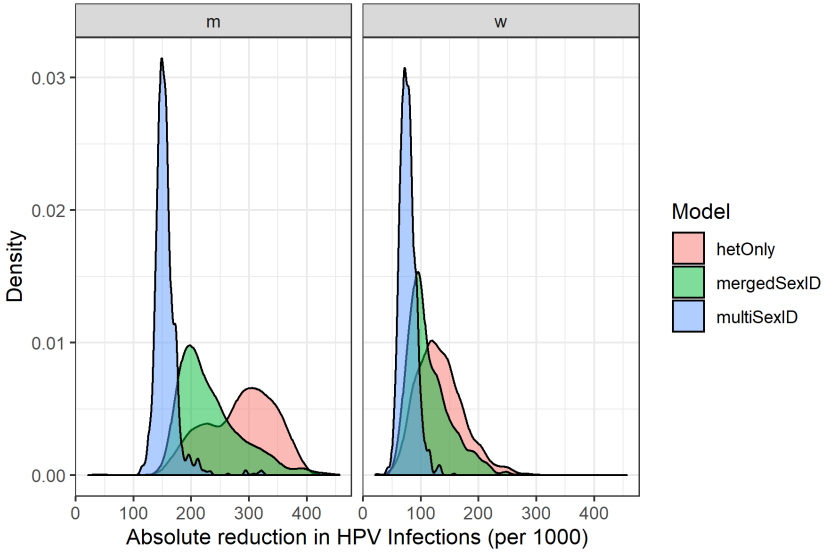



Averaged SexID Population
Men
Women

Contact Between Groups: Averaged Multiple Sex ID




het
msid
msid_avg
m
w
Density
150
100
50
0
0.04
0.06
0.08
0.10
Prevalence
so
het
msid
msid_avg


m
w
0.03
0.02
0.01
0.00
Density
0
100
200
300
400
Absolute reduction in HPV Infections (per 1000)
Model
hetOnly
mergedSexID
multiSexID

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- Including multiple sexual orientations is more representative of true population, and provides a 'seat at the table' for those who do not identify as heterosexual
- As in other study types, modeling studies should strive to ensure that their model population is as representative of the target population as possible, to facilitate valid generalization of results

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- Compare cost-effectiveness estimates of different vaccination strategies under each model

Any Questions?