

# Agent-Based Modeling of HPV Transmission and Vaccination



## Impact In Atlanta Metro Area

Anish Arora, Hailey Toepfner, Scott Watanuki

School of Computational Science Engineering, Georgia Institute of Technology



### Background

HPV is a highly prevalent STI. Vaccination of preadolescents and adolescents substantially reduces acquisition of high-risk types which lead to various cancers.

HPV (16/18) accounts for 70% of cervical cancer, the 5th leading cause of death for women. HPV also causes other cancers as well.

### Motivation

How do vaccination coverage and efficacy scenarios change HPV prevalence and incident infections in the heterogeneous population over time in the Atlanta Metropolitan Area?

To answer this we, built an Agent Based Model (ABM) with heterogeneous mixing to better understand HPV and Cervical cancer in Atlanta.

### Some Key Data

1. Atlanta Metro Demographic data (US Census 2024)
2. Current HPV 16/18 Infection Rates (McQuillan et Al)
3. Screening Rates by demographic (Spencer et Al. 2023)
4. Sexual behaviors of US adults (Santelli et Al. 2017)
5. SEER Cervical Cancer Incidence rates

### Methodology

Agents were broken up by stratified census data along with screening and vaccination data.

For the sexual model based on literature by Walker et Al. the agents were stratified by age and “sexual activity class,” which allowed partners to randomly choose other partners based on sexual activity levels and age.

Probability of interaction equation:

$$\rho_{i,h \rightarrow j,m} = \epsilon_A \epsilon_S \delta_{ij} \delta_{hm} + \epsilon_A (1 - \epsilon_S) \delta_{ij} \frac{P_{j,m}}{\sum_{\beta} P_{j,\beta}} + (1 - \epsilon_A) \epsilon_S \delta_{hm} \frac{P_{j,m}}{\sum_{\alpha} P_{\alpha,m}} + (1 - \epsilon_A) (1 - \epsilon_S) \frac{P_{j,m}}{\sum_{\alpha} \sum_{\beta} P_{\alpha,\beta}}$$

Figure 1: Probability of Contact Eq. From Walker et Al.

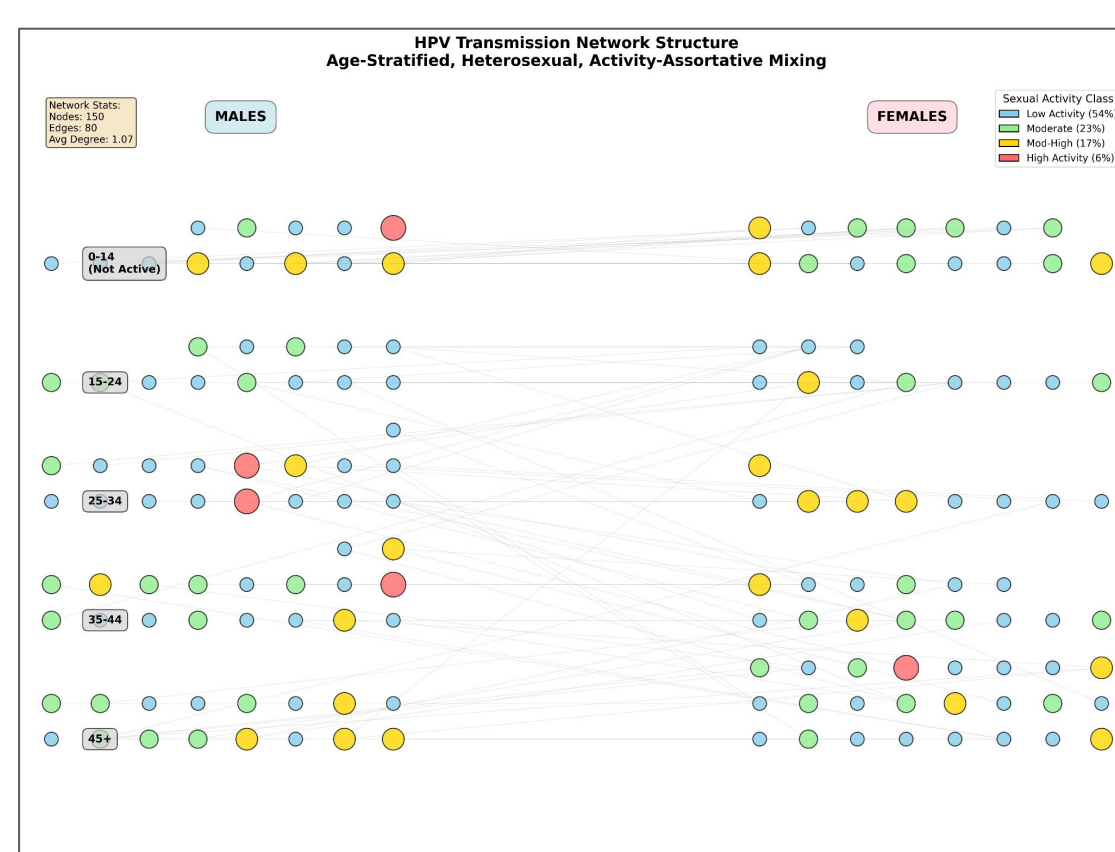


Figure 2: Simple Depiction of Sexual Network

Figure 2 shows a simple sample depiction of the sexual model and how partners connected. This affected contact rates based on each specific partner.

The disease progression states are:  $S \rightarrow I, I \rightarrow S, I \rightarrow C$

Cancer progression probability was calibrated to match studies by SEER.

### Validation & Verification

**Verification:**

- 1) Sensitivity Analysis  $\rightarrow$  Partnership Patterns
- 2) Sensitivity Analysis  $\rightarrow$  Beta Scaling

Adjusted an internal parameter  $\beta$  until the model's output (HPV rates) aligned with external empirical findings.

**Validation:**

Validated model against NSFG and HPV prevalence from NHANES

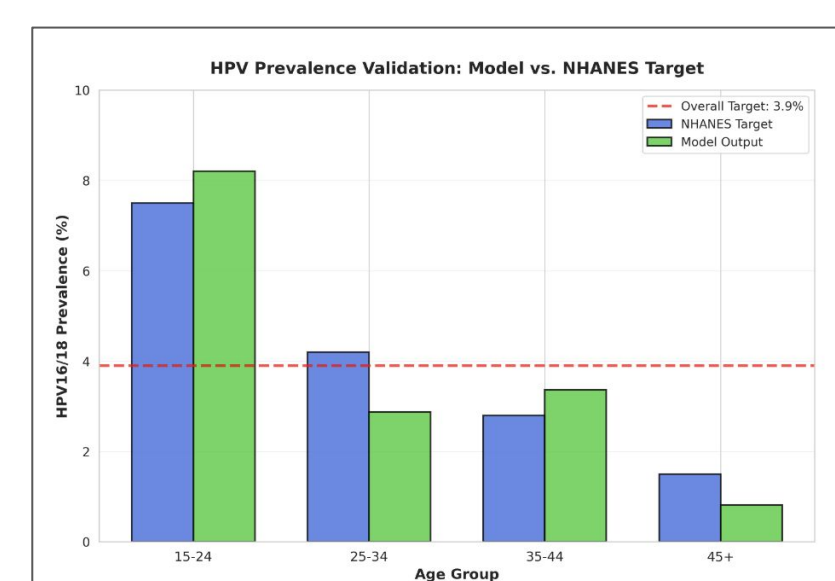


Figure 3: HPV Prevalence Validation Findings

### Results

Graphs depicting vaccination coverage impacts on general population infection:

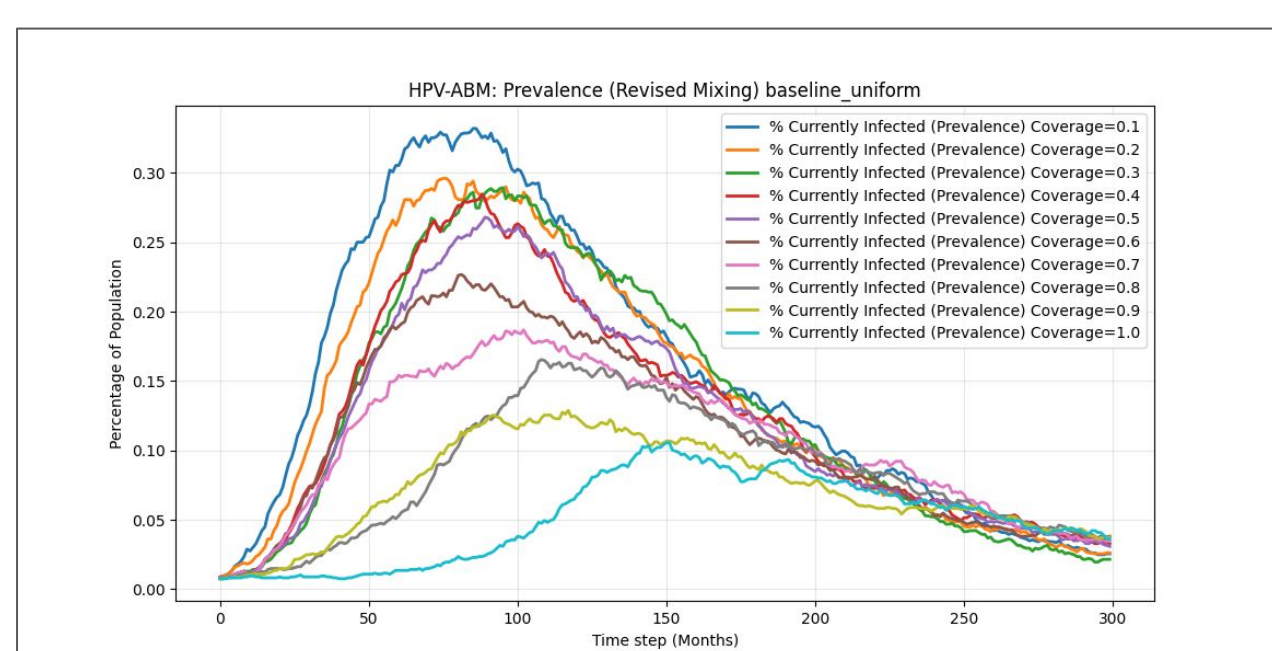


Figure 4: HPV Prevalence Baseline by Vaccination Coverage

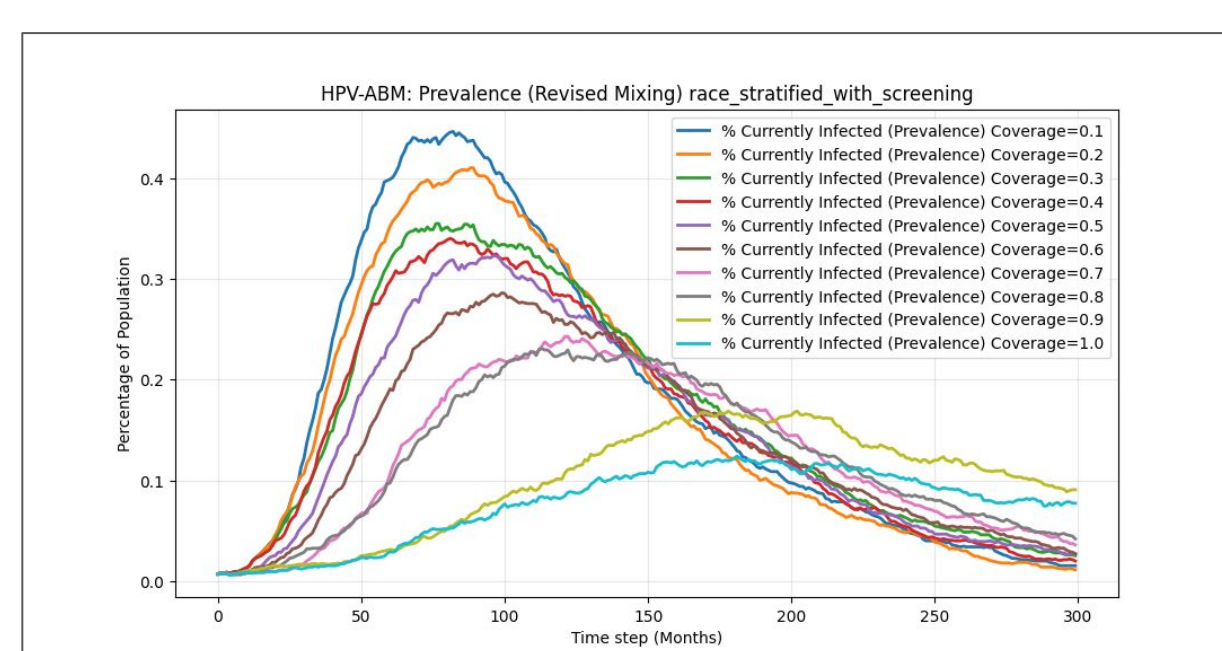


Figure 5: Race Stratified HPV Prevalence by Vaccination Coverage

Results continuously showed vaccination increases dramatically had effects in decreasing the spread of HPV. Disparities in race-specific vaccination coverage are sufficient to sustain higher long-term endemic HPV rates.

### Conclusions

Future improvements require two main approaches: enhancing data linkage and optimizing computational efficiency. We must better link HPV infections to all associated cancer types and utilize more specific local data for screening rates, which would lead to a more straightforward and accurate model for Atlanta.

Furthermore, increasing the model's parallelization is necessary to efficiently run the compute-intensive simulations, allowing for more trials to generate confident, interpretable outcomes for rare events like cancer incidence.