

Comparing Structural Determinants, Psychosocial Factors, and Individual-Level Risk Indicators for Women's HIV Prevention in the Urban South

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Running Head: Structural, Psychosocial, and Individual HIV Risk in Women

ABSTRACT

Background: Cisgender women, particularly Black women in the Southeastern U.S., experience substantial HIV risk. While current prevention strategies emphasize individual-level risk indicators, understanding the relative contributions of structural determinants and psychosocial factors is essential. This study aimed to evaluate the effects of structural determinants, psychosocial factors, and individual indicators on HIV risk among women in a high-incidence U.S. region.

Methods: Retrospective cohort of adults assigned female at birth, with ≥ 1 encounter at Grady Health, a safety-net system serving two of Atlanta's Ending the HIV Epidemic counties (2012–2022). New HIV diagnoses were identified using state surveillance. Logistic regression estimated associations between structural, psychosocial and individual-level indicators and HIV. Population attributable fractions (PAFs) were derived from multivariable models.

Results: Of 333,263 women, 617 (0.19%) acquired HIV; 88% were Black, mean age was 41. Structural determinants were common and strongly associated with HIV, specifically above-median SVI (aOR=1.7 [1.5–2.0]) and housing insecurity (5.2 [3.9–6.7]). Psychosocial factors such as substance use disorder (3.8 [3.1–4.6]) and individual-level indicators like STIs [3.1 [2.2–4.4]) were also associated. PAF analyses indicated that structural determinants, specifically residence in high-prevalence HIV zip codes (PAF=63% [57%–70%]) and above-median SVI (21% [14%–27%]), accounted for a larger share of population-level HIV risk than individual-level indicators like STIs (5% [3%–7%]).

Conclusion: Structural determinants were more strongly associated with HIV and explained a greater share of population-level risk than individual-level indicators. Integrating community context into risk tools may better identify PrEP candidates and support prevention in high-incidence settings.

KEY WORDS: HIV prevention, Women's health, Social determinants of health, PrEP implementation, Epidemiology

INTRODUCTION

Cisgender women account for a growing proportion of new HIV diagnoses, and ending the epidemic requires a clearer understanding of risk in this population. Atlanta has the second-highest HIV diagnosis rate among women of any U.S. metropolitan area, and Georgia ranks second among states, underscoring the city and the state as epicenters of the epidemic^{1,2}. Black women bear a disproportionate burden, comprising 50% of new HIV diagnoses among women in 2022, despite representing only 13% of the U.S. female population²¹. Since Black women in the South carry the highest HIV burden nationally, this group is crucial for national HIV prevention efforts.

Despite this burden, current risk screening and assessment approaches are not well-suited to identify women prior to HIV acquisition. Existing frameworks rely on self-reported sexual and substance use behaviors or diagnosis of bacterial STI³. While these risk factors are well established,^{3,4} they are often absent, unidentified or undisclosed in clinical settings prior to diagnosis, hindering accurate risk assessment and HIV Pre-Exposure Prophylaxis (PrEP) uptake^{5,6}. Self-report is further constrained by community stigma around sexuality and LGBTQ+ status, especially in the South, where stigma is high^{7,8}. As a result, HIV frequently occurs among women without clinically identifiable individual risk indicators, not because they are absent, but because the tools designed to detect them are insufficient⁹.

HIV vulnerability in women is also shaped by community-level and structural determinants such as neighborhood-level deprivation, limited healthcare access, and medical mistrust¹⁰⁻¹³. These social determinants of health (SDoH) may amplify individual-level risk factors, making individuals more susceptible or independently contribute to vulnerability, often doing so in a

dose-dependent manner¹², thereby contributing to HIV acquisition that current screening approaches fail to anticipate.

The resulting complexities of risk assessment contribute to low HIV risk perception and persistently low PrEP uptake among women, particularly Black women, who would benefit from PrEP^{14–17}. Current U.S. PrEP guidelines emphasize offering PrEP in response to self-reported sexual and substance use behaviors and diagnosis of a bacterial STI, yet many women who acquire HIV are not identified through these criteria⁹. To address this, we evaluated how individual-level indicators, psychosocial barriers, and structural determinants contribute to HIV risk at both the individual and population levels. By integrating adjusted associations and population attributable fractions in a large health-system cohort of women in Atlanta, we aimed to inform clinical risk assessment and guide prevention strategies that better reflect women's lived risk in high-incidence settings.

METHODS

Study Design and Setting

This retrospective cohort study was conducted at Grady Health System (GHS), an urban safety-net hospital in Atlanta, Georgia, that serves a catchment area of >6 million people living in Fulton and DeKalb counties¹⁸, which are two of the 42 Ending the HIV Epidemic Initiative counties¹⁹. The system runs a Level 1 trauma center and offers comprehensive emergency and primary care services across 16 locations in Atlanta. It cares for over 700,000 patients each year, with more than 250,000 emergency and over 400,000 primary care and medical specialty visits annually. GHS performs more than 42,000 HIV tests per year, is the largest reporting site for new HIV diagnoses in Georgia^{20,21}, and is home to the Ponce De Leon Center, one of the

nation's most comprehensive outpatient HIV clinics, providing care for over 6,000 individuals with HIV annually^{22,23}.

Data Sources and Collection

Clinical and demographic data were extracted from the GHS Epic electronic health record (EHR) from 2012 to 2022. Extracted variables included demographic characteristics, social determinants of health (SDOH), ICD diagnosis codes, medication data, healthcare encounters, and laboratory results. Data was cleaned and processed to create analytic variables. The Emory University Institutional Review Board and the GHS Research Oversight Committee approved this study.

Study Population

The cohort included female individuals aged 18 years or older with at least one healthcare encounter at GHS between 2012 and 2022. Females were defined as individuals with documented legal sex or sex assigned at birth as female, with no record indicating male sex assigned at birth or a transgender female gender identity. Both legal sex and sex assigned at birth were used to accurately capture female individuals, as systematic documentation of sex assigned at birth was not implemented in the GHS EHR until 2018. The selection of participants into the study cohort is illustrated in **Figure 1**.

Primary Outcome: HIV

The primary outcome was HIV diagnosis during the study period, identified through linkage with the Georgia Department of Public Health secure database²⁴. An anchor date served as a reference point to provide a consistent temporal framework for time-dependent variables.

For PWH, the anchor date was the first documented HIV diagnosis between 2012 and 2022; for PWoH, it was the most recent healthcare encounter during the study period. Participants were

excluded if their only healthcare encounters occurred after their HIV diagnosis, if their diagnosis date fell outside of 2012-2022, or if they had visited the GHS Ponce HIV Clinic without a corresponding HIV diagnosis in the registry, suggesting likely misclassification.

Risk Factors: Structural Determinants, Psychosocial Factors, and Individual-level

Indicators

We organized risk factors into three domains: structural and community-level determinants (e.g., social vulnerability, high-incidence residence, housing insecurity), psychosocial factors (e.g., depression, substance use disorder), and individual-level risk indicators (e.g., bacterial STIs, HIV screening). This classification reflects the syndemic literature [^{11,12,25–27}], which positions mental health conditions and substance use as factors shaped by upstream structural inequities that in turn mediate individual-level HIV risk.

Structural and community-level determinants were grouped into three major categories: (1) geographic and community risk, (2) housing and socioeconomic instability and (3) healthcare utilization. For geographic and community exposures, we utilized dichotomous indicators. These included whether a patient lived in a ZIP code with cumulative HIV incidence above the median or in the top quartile for metro Atlanta between 2012 and 2022 ²⁸. We also assessed their residential area's ranking within the top half or top quartile of the Social Vulnerability Index (SVI). The SVI, a census-tract-level measure ²⁹, was derived from patient addresses listed at the anchor date via geocoding to provide insight into neighborhood-level vulnerability. Housing and socioeconomic instability was indicated by housing insecurity (ICD-10 codes), residential mobility (four or more address changes within a year), and a broader SDoH composite diagnosis from ICD codes including food insecurity, illiteracy, unemployment, and lack of social support (which has been linked to housing instability³⁰). Healthcare utilization was assessed through

encounters at GHS emergency departments, primary care clinics, or women's clinics over a two-year period.

Psychosocial factors included mental health conditions (depression, bipolar disorder, and all mental health diagnoses) as well as substance-related disorders (ICD-10 codes). We grouped mental health with substance use since they commonly co-occur and share common upstream structural risk factors, and function as an intermediary between structural conditions and HIV vulnerability³¹.

Individual-level risk-indicators were grouped into three categories: (1) STIs and sexual health labs, (2) hepatitis and HIV screening, and (3) reproductive and women's clinical risk indicators. Reproductive and women's clinical risk indicators included pregnancy, PID, non-occupational post-exposure prophylaxis (nPEP), and sexual assault. These represent clinical encounters or events that may reflect circumstances of HIV vulnerability occurring in women, rather than volitional behaviors. Bacterial STI results, Hepatitis C, and HIV screening were ascertained from laboratory results within two years of anchor date. Pregnancy, pelvic inflammatory disease (PID), non-occupational Post-Exposure Prophylaxis (nPEP) use, and sexual assault were identified via ICD codes. Trichomonas ascertainment combined laboratory and ICD-10 codes, as the diagnostic standard changed from wet mount to nucleic acid amplification during the study period.

Confounder Selection

Directed acyclic graphs (DAGs) were created using prior knowledge and literature to model hypothesized causal links between exposures and HIV^{32–34}. They clarified the assumed causal structure, identifying mediators, potential confounders, and distinguishing covariates that modify

effects from those that bias estimates if unadjusted. This guided variable selection and adjustment. DAGs were refined through expert input and data.

Across domains, demographic characteristics such as age, race, ethnicity, and an SDoH diagnosis were potential confounders, influencing both exposures and outcome pathways. Substance use exposures had mental health, healthcare utilization, and STIs as mediators. Mental health exposures were influenced by substance use, healthcare utilization, and STIs acting as mediators. Positive STI labs were affected by STI testing frequency, other STIs, and substance use, which were additional confounders. In healthcare utilization, mental health and substance use were potential confounders, while STIs were mediators. Final DAGs for each domain are shown in **supplemental digital content 1**.

Statistical Analysis

Statistical analyses were conducted in the R statistical software, version 4. Missingness was assessed; SVI was missing 5% of data. Descriptive statistics for continuous variables are presented as means with standard deviations (SD), and dichotomous variables as frequencies and column percentages (%). Univariate logistic regression models were used to estimate the crude association between each exposure and HIV status (dichotomous outcome). Multivariable logistic regression models were fitted for each exposure to estimate adjusted associations, controlling for confounders identified from the DAGs. Odds ratios (ORs) and adjusted ORs (aORs) with 95% confidence intervals (CIs) and p-values were obtained; statistical significance was defined as $p < 0.05$ or a 95% CI not including one.

To assess the population-level impact of potentially modifiable factors, population-attributable fractions (PAFs) were derived from adjusted ORs and the prevalence of each exposure in the full cohort [35]. PAF % and 95% CI from 500 bootstrap replications were reported, capturing both the

strength of association and the prevalence of the exposure. A mediation analysis wasn't performed. PAFs estimate the proportion of cases due to each exposure, but do not distinguish direct and indirect effects. They assume causality and no residual confounding, serving as exploratory population impact estimates rather than precise causal measures.

RESULTS

Demographics

In the cohort of 333,263 female individuals, 617 (0.19%) were newly diagnosed with HIV (**Table 1**). The mean age of individuals without HIV (PWoH) was 43 years (SD 18), whereas those newly diagnosed with HIV (PWH) had a mean age of 41 years (SD 13.3). Compared to individuals aged 18–34, those aged 35–49 years had increased odds of HIV (aOR=1.5, 95% CI [1.3–1.8]), while those aged 50 years or older had reduced odds (aOR=0.73 [0.60–0.87]). The majority of the cohort identified as Black (89% of PWH vs. 69% of PWoH), and Black race was significantly associated with HIV (aOR=2.6 [1.9–3.4]). Additionally, 97% of PWH were non-Hispanic, which was also linked to higher odds of HIV (aOR=2.9 [1.8–4.9]).

Structural Determinants

Geographic/Community Risk

A higher proportion of PWH lived in zip codes within the top 25% of HIV incidence in Atlanta, compared to PWoH (73% vs. 38%) as well as high HIV incidence zip codes, which are zip codes above the median HIV incidence (83% vs 48%, **Table 2**). Among PWH, 47% had an SVI above the median, and 11% were in the highest SVI quartile, compared to 36% and 6.9% of PWoH, respectively. Both living in a high HIV incidence zip code and having an SVI above the median were strongly associated with HIV (aOR=4.5 [3.7–5.6] and aOR=1.7 [1.5–2.0], respectively).

Housing/Socioeconomic Instability

PWH experienced significantly greater housing and socioeconomic instability compared to PWoH. Housing insecurity was reported in 10% of PWH versus 2.1% of PWoH, with a strong association with HIV (aOR=5.2 [3.9–6.7]). Residential mobility, defined as having four or more addresses within one year, was also more common among PWH (19% vs. 8.2%) and associated with more than double the odds of HIV (aOR=2.2 [1.8–2.7]). Additionally, 13% of PWH had a documented SDoH diagnosis, compared to 4.8% of PWoH (aOR=2.9 [2.3–3.7]).

Healthcare Utilization

Frequent emergency room visits (≥ 3 visits in the two years prior) were more common among PWH than PWoH (30% vs. 12%, aOR=2.3 [1.9–2.8]). In contrast, engagement in primary care and women's healthcare services was similar between groups.

Psychosocial Factors

Mental health diagnoses were more common among PWH compared to PWoH (**Table 3**). Specifically, bipolar disorder, depression, and schizophrenia were documented in 12%, 17%, and 10% of PWH, respectively, versus 4%, 11%, and 3% of PWoH. Both bipolar disorder and schizophrenia were strongly associated with HIV (aOR=3.4 [2.6–4.3] and aOR=4.1 [3.2–5.3] respectively). Additionally, PWH had higher odds of documented alcohol abuse (aOR=2.8 [2.2–3.6]) and substance use (aOR=3.8 [3.1–4.6]) compared to PWoH.

Individual-Level Risk Indicators

Laboratory-confirmed STIs and HIV Screening

STIs were more common among PWH. In the two years before, 9.4% of PWH tested positive for an STI compared to 2.2% of PWoH (aOR=3.1 [2.2–4.4]). Notably, trichomonas and chlamydia were also associated with HIV risk (aOR=3.2 [1.9–5.1], aOR=1.6 [1.0–2.5], respectively). Consistent with previous studies, lab-confirmed syphilis and gonorrhea in the past two years

were each associated with increased odds of HIV (aOR=8.7 [2.9–25.1], aOR=2.0 [1.5–2.6], respectively); however, only 8 (1.3%) women with HIV had lab-confirmed syphilis. More frequent HIV screening during the two years before diagnosis was also linked to HIV acquisition (aOR=1.7 [1.5–1.8]).

Population Attributable Fractions

PAFs were examined to quantify the population-level impact of modifiable factors strongly associated with HIV from the multivariable analysis. The structural determinants of living in a high HIV incidence zip code (PAF=63% [57%–70%]) and SVI above the median (PAF=21% [14%–27%]) explained the largest proportion of risk for HIV acquisition (**Figure 2**).

Psychosocial factors, such as substance use disorder (PAF=13% [10%–17%]), depression (PAF=8% [3.9%–12%]), and bipolar disorder (PAF=8% [5.4%–11%]) accounted for smaller but still meaningful proportions. Individual-level indicators, such as STIs, contributed marginally (PAF=5% [3.0%–7.1%]).

DISCUSSION

In this ten-year retrospective cohort study of over 333,000 women in Atlanta, structural and community-level barriers were disproportionately concentrated among women who acquired HIV and accounted for the largest share of population-level HIV risk. Nearly half of the women diagnosed with HIV had an above-median Social Vulnerability Index, and more than 80% lived in a high HIV incidence zip code, compared with fewer than 10% who had a bacterial STI before diagnosis. Multivariable regression confirmed that structural determinants were more strongly associated with HIV risk than most individual-level risk indicators, and population attributable fractions showed that high HIV incidence residence and high SVI explained the greatest share of population-level risk. By comparison, psychosocial factors such as substance use disorder and

mental health conditions contributed more modestly, and bacterial STIs explained little at the population level despite high odds ratios.

These findings highlight an essential mismatch between guideline-defined HIV risk and women's lived risk in high-incidence settings. In another recent cohort in Atlanta, many women who acquired HIV lacked risk factors identified through conventional risk screening assessments before diagnosis⁶. Instead, structural and community contexts, such as living in poverty or within areas with higher prevalence of HIV within sexual networks, were the primary drivers of population risk. This disparity may help explain persistently low PrEP uptake among women through two pathways: 1) *providers* may not identify women as PrEP candidates when conventional clinical markers are absent, and 2) *women* do not perceive themselves at risk if frameworks and messaging center on behavioral profiles that they do not identify with^{17,36–38}. These dynamics underscore the need to shift frameworks to integrate both individual-level indicators *and* structural barriers when identifying women who could benefit from prevention. Although bacterial STIs were associated with HIV risk, their prevalence was very low, consistent with several other extensive, population-based studies showing that few women have bacterial STIs before diagnosis^{39,40,40}. Notably, chlamydia and trichomonas infections were significantly associated with HIV and showed higher population impact than gonorrhea or syphilis. Although this association has been described previously, it has primarily been documented in international settings or among populations engaged in sex work^{41,42}. In contrast, our findings demonstrate this association in a representative U.S. cohort of women living in a high-incidence setting, where these infections may serve as markers of recent sexual activity or partner factors. While specific STI associations are clinically relevant, structural and psychosocial risk factors emerged as stronger predictors of individual risk than many individual risk indicators, a pattern recently

observed in a large HIV risk prediction study in the Southern US⁴³. Our study adds to the literature by extending the impact of individual associations to demonstrating how structural exposures account for the greatest share of women's risk at the population level using PAFs. Even though PAFs are rarely used in U.S. HIV studies because of the relatively low incidence in most regions, their application in Atlanta, where incidence in women is among the highest in the country, offers meaningful insight into the population-level contributions of structural and community determinants.

Current CDC and IAS-USA PrEP guidelines recommend discussing PrEP with all sexually active individuals, but focus on individual-level risk indicators for PrEP eligibility^{3,44}. Despite the recommendation for universal counseling, practice often remains indicator-driven; in our cohort, only 1 of 617 women who acquired HIV received PrEP beforehand. This gap shows the need to refine risk assessment strategies to ensure women in high-prevalence communities are not overlooked. Our findings suggest that incorporating structural determinants, such as residence in high-incidence areas or social vulnerability, could broaden PrEP candidate identification. Additionally, our work translating individual risk to the population level using PAFs reveals additional insights that could inform guidelines: risk factors that appear modest at the individual level can still have large population impact if they're prevalent, and conversely, factors with dramatic odds ratios (e.g., syphilis) but low prevalence contribute little at the population level. Incorporating structural context *and* applying population-level insights to risk assessment could help move practice towards the universal PrEP counseling that guidelines already recommend, particularly in communities where structural vulnerability and HIV prevalence are concentrated.

The three-domain classification used in this study (structural/community > psychosocial > individual-level risk indicators) aligns with the modified socio-ecological model proposed by Baral et al, (2013)⁴⁵, which positions individual-level risks within a broader community, network, and policy context. This framework argues that while individual-level risks directly mediate acquisition, higher-order structural factors mitigate or facilitate transmission at the population level. Capturing these upstream risks is arguably more actionable than individual data alone. Our study adds to the body of literature investigating these complex, interwoven risk factors using multilevel frameworks, including syndemic frameworks, mediation, and intervention studies. HPTN 064 demonstrated a dose-response relationship between concurrent syndemic domains (psychosocial, individual-level structural, and neighborhood-level structural) and HIV risk behaviors, and found that being in multiple syndemic groups increased the prevalence of behavioral risk factors¹². This supports our findings that structural determinants, psychosocial factors, and individual-level indicators operate along a gradient of population-level impact. HPTN 064 qualitative data identified multiple syndemic factors, particularly financial insecurity, as a prominent theme underlying risk-taking pathways^{11,46–48}, paralleling our findings that housing insecurity and SVI were among the strongest determinants. A conceptual model describes a structural-to-individual cascade, suggesting that HIV risk begins at the structural level and cascades to community, social, and individual levels, with downstream effects compounding structural vulnerability using a positive feedback loop⁴⁹. For example, housing instability and food insecurity have been linked to survival or transactional sex⁴⁸, which worsen structural vulnerability. The complexity of this intersectional framework is why we chose DAGs to guide confounder selection. This study provides empirical support, through PAFs, for the

gradient of population-level impact that previous frameworks have described but rarely quantified in U.S. women.

Strengths and Limitations

Strengths of this study include utilizing the Georgia state database from the Georgia Department of Health, which improved the identification of women with newly diagnosed HIV beyond what can be captured with a single health system. Additionally, GHS is the largest reporting site for new HIV diagnoses in Georgia and serves a catchment area of 6 million people in 2 of the 42 counties targeted by the Ending the HIV Epidemic initiative. This underscores the importance of this cohort to high-risk HIV populations, especially socioeconomically vulnerable women in the Southeastern U.S. This study period spans 10 years and includes over 330,000 women, providing a large and diverse cohort. Finally, the analytic approach was guided by DAGs, which helped guide thoughtful variable and confounder selection and reduce the risk of over-controlling. Despite the cohort's representativeness, several limitations exist. This was a retrospective, non-controlled study based on data from a single health system, which may limit generalizability; however, the size, various healthcare settings, and regional scope support its relevance to other Southern urban settings. Although this study captures lived risk in a socioeconomically vulnerable safety-net population, our cohort may underrepresent the lived experience of Black women with higher financial resources. Replicating this study among Black women across the socio-economic spectrum could assess the generalizability of these findings. Reliance on EHR data also introduces the potential for missing or incomplete information, particularly regarding socio-structural variables, where stigma and time constraints may impact disclosure or documentation. Additionally, 5% of the SVI data was missing, and STI-specific surveillance databases were not utilized, although laboratory results partially mitigated this gap. Interpreting

PAFs requires caution. They assume causality and no uncontrolled confounding, risking overstatement. As non-sequential and non-additive, pathways overlap and cannot be summed. PAFs are thus exploratory indicators of population impact, rather than precise estimates. Finally, while our use of DAGs provides transparency, they rely on assumptions that may not accurately capture the complexities of socio-structural frameworks. Incorrect selections can introduce undue bias, even as they help mitigate the risk of overcontrolling. It is also important to note that while our DAGs helped identify potential mediation pathways, we did not formally evaluate mediators in our analysis.

Conclusion

In this large, socioeconomically vulnerable population of women living in a high-incidence region in the southern US, we evaluated HIV risk factors at the individual and population levels. Structural determinants accounted for the largest share of population-level risk; psychosocial factors contributed modestly; and individual-level risk indicators, such as bacterial STIs, explained little at the population level despite strong individual associations. Yet in practice, PrEP discussions remain driven by individual-level indicators, and many women who acquire HIV are not identified as candidates. Our findings suggest that incorporating structural context in PrEP decision-making could broaden identification of women who would benefit from prevention.

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AUTHOR CONTRIBUTIONS

Concept and design: All authors.

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CONFLICT OF INTEREST DISCLOSURES

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DATA SHARING STATEMENT

Deidentified and date shifted final dataset and a data dictionary will be made available with publication. Data will be accessible to anyone upon request by contacting megan.schwinne@emory.edu. The data may be used for any purpose, and access will be granted following approval of a proposal. No additional supporting documents are available.

SDC final ---<https://links.lww.com/QAI/C777>

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ACCEPTED

FIGURE LEGENDS

Figure 1. Breakdown of Patient Cohort by Inclusion and Exclusion Criteria

No legend.

Figure 2. Population Attributable Fraction (PAF) of HIV Risk Factors Among Women

Figure 2A. PAF Bar Plot by Domain. Bars show the estimated proportion of new HIV diagnoses linked to each exposure, assuming causality, sorted from largest to smallest PAF. Bars are colored by domain: structural/community-level determinants, psychosocial factors, and individual-level risk indicators. Error bars indicate 95% confidence intervals. Larger PAFs indicate greater potential population-level impact if the risk factor were reduced. The PAF ranking differs from odds ratio ranking, showing that common risk factors with modest individual effects can contribute more to HIV incidence than rare risk factors with strong individual effects. PAFs are nonadditive because underlying pathways overlap; they cannot be summed.

Figure 2B. Bubble Plot of Population Attributable Fraction (PAF) Estimates and Adjusted Odds Ratios (OR). Each bubble represents one risk factor, positioned by its prevalence of exposure (horizontal axis) and adjusted odds ratio (vertical axis). Domain is indicated by both color and shape: structural and community determinants (circles), psychosocial factor (triangles), and individual-level risk indicators (squares). Risk factors are numbered 1 to 14 in descending order of PAF; the key lists each factor. The horizontal dashed line marks an adjusted odds ratio of 1.0 (no association). Risk factors with high ORs do not necessarily produce large PAFs if they are rare, while more

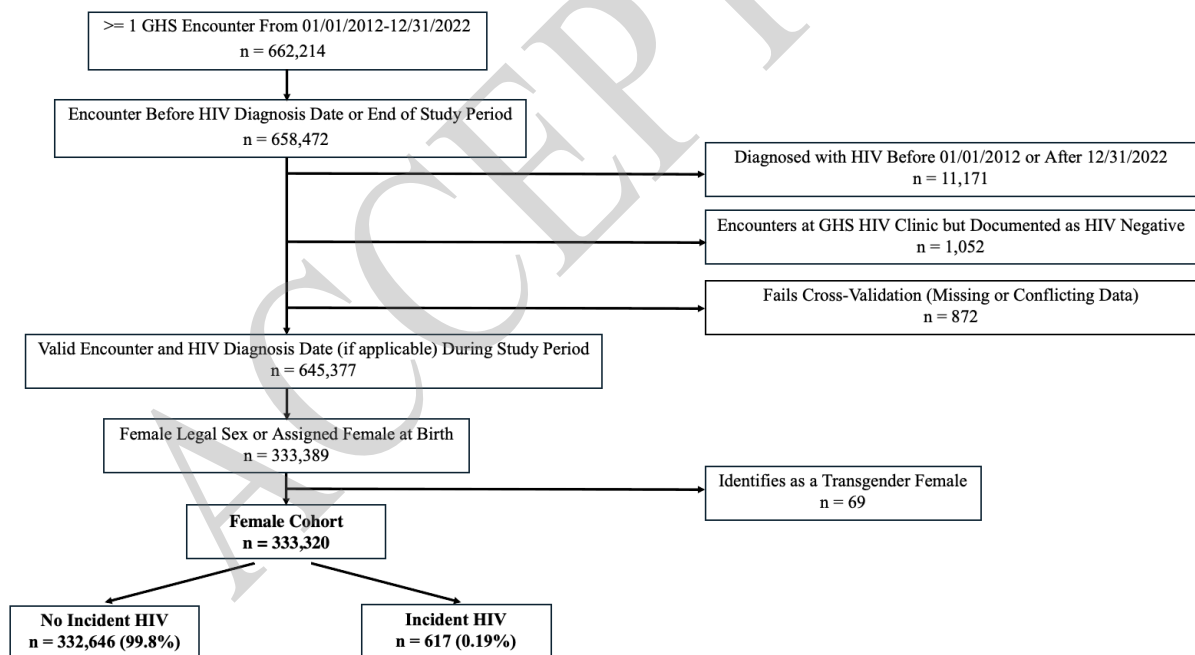
common exposures with modest ORs can account for a greater share of population-level risk.

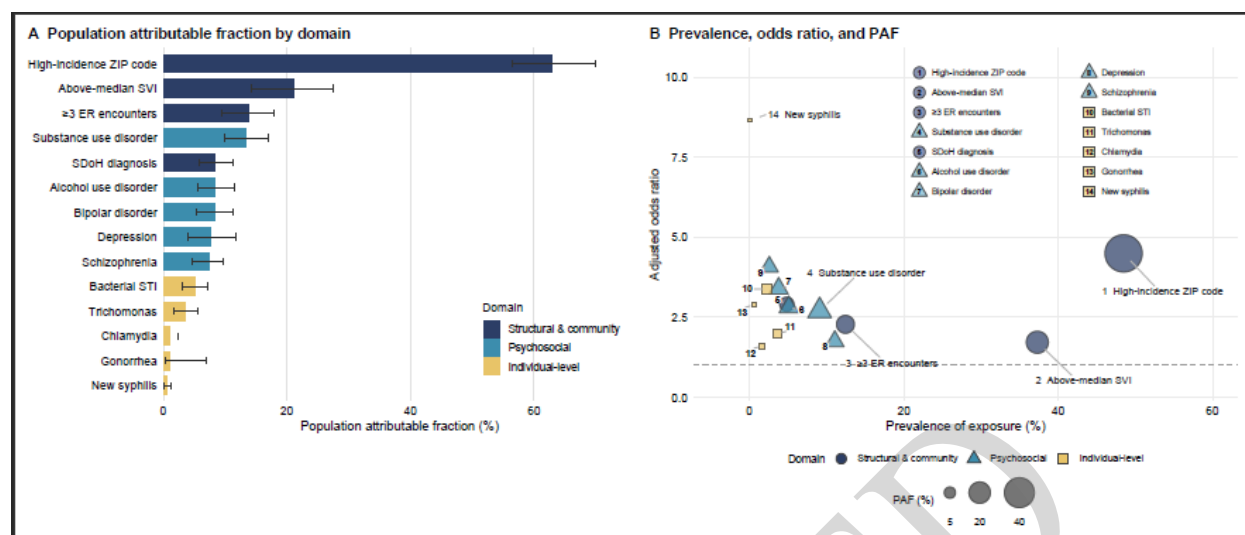
SUPPLEMENTAL DIGITAL CONTENT

Supplemental Digital Content 1.pdf

Contains supplemental figures 1-4 which show Directed-Acyclic Graphs (DAGs) to identify potential confounders.

Figure 1. Breakdown of Patient Cohort by Inclusion and Exclusion Criteria





TABLES

Table 1. Demographics of the Study Population

Demographic	n (%)		Odds Ratio (95% CI)	
	Incident HIV (617; 0.20%)	No Incident HIV (332,646; 99.8%)	Unadjusted	Adjusted
Age, years ^a	41.0 (13.3)	43.0 (18.0)	0.99 (0.99, 1.0)	0.99 (0.99, 1.0)
Age Group				
18-34	240 (39%)	138,876 (42%)	0.87 (0.74, 1.0)	0.92 (0.78, 1.1)
35-49	204 (33%)	83,303 (25%)	1.5 (1.2, 1.7)	1.5 (1.3, 1.8)
50+	173 (28%)	110,467 (33%)	0.78 (0.66, 0.93)	0.73 (0.60, 0.87)
Race				
White	42 (6.8%)	44,219 (13%)	0.48 (0.34, 0.64)	0.57 (0.41, 0.77)
Black	551 (89%)	229,883 (69%)	3.7 (2.9, 4.9)	3.3 (2.5, 4.3)
Asian	1 (0.2%)	7,306 (2.2%)	0.07 (0.00, 0.32)	0.07 (0.00, 0.30)
Other or Unknown	8 (1.3%)	20,508 (6.2%)	0.2 (0.09, 0.37)	0.58 (0.21, 1.3)
Ethnicity				
Hispanic	14 (2.3%)	31,440 (9.5%)	0.22 (0.12, 0.36)	0.48 (0.26, 0.84)
Non-Hispanic	598 (97%)	284,027 (85%)	5.4 (3.5, 8.8)	2.9 (1.8, 4.9)

^aPresented as mean (SD).

Table 2. Structural and Community Determinants and Psychosocial Factors

Variable	n (%)		Odds Ratio (95% CI)	
	Incident HIV (617; 0.20%)	No Incident HIV (332,646; 99.8%)	Unadjusted	Adjusted
Geographic / Community Risk				
Top 25% HIV Incidence Zip Code ^a	453 (73%)	127,249 (38%)	4.5 (3.7, 5.4)	3.9 (3.3, 4.7)
Top 50% HIV Incidence Zip Code ^a	510 (83%)	160,984 (48%)	5.1 (4.1, 6.3)	4.5 (3.7, 5.6)
SVI ^b	0.53 (0.17)	0.47 (0.17)	7.4 (4.8, 11.4)	6.0 (3.8, 9.9)
Top 25% SVI	66 (11%)	22,872 (6.9%)	1.7 (1.3, 2.2)	1.6 (1.3, 2.1)
Top 50% SVI	288 (47%)	118, 076 (36%)	1.8 (1.5, 2.2)	1.7 (1.5, 2.0)
Housing and Socioeconomic Instability				
Housing Insecurity ^c	64 (10%)	7,003 (2.1%)	5.4 (4.1, 6.9)	5.2 (3.9, 6.7)
≥4 Addresses/1 Year (Residential Mobility)	115 (19%)	27263 (8.2%)	2.6 (2.1, 3.1)	2.21 (1.8, 2.7)
SDoH Diagnosis ^c	82 (13%)	15,956 (4.8%)	3.0 (2.4, 3.8)	2.9 (2.3, 3.7)
Healthcare Utilization				
≥3 ER Encounters ^{a,d}	188 (30%)	41,189 (12%)	3.1 (2.6, 3.7)	2.3 (1.9, 2.8)
≥1 Primary Care Encounter ^{a,d}	166 (27%)	99,193 (30%)	0.87 (0.72, 1.0)	0.76 (0.61, 0.94)
≥1 Women's Clinic Encounter ^{a,d}	102 (17%)	51,321 (15%)	1.1 (0.87, 1.3)	1.0 (0.81, 1.3)
≥1 Primary Care or Women's Clinic Encounter ^{a,d}	226 (37%)	127,389 (38%)	0.93 (0.79, 1.1)	0.85 (0.7, 1.0)
Psychosocial Factors				
Bipolar Affective Disorder ^c	74 (12%)	12,450 (3.7%)	3.5 (2.7, 4.4)	3.4 (2.6, 4.3)
Depression ^c	105 (17%)	36,669 (11%)	1.7 (1.3, 2.0)	1.8 (1.4, 2.1)
Schizophrenia ^c	61 (9.9%)	8,419 (2.5%)	4.2 (3.2, 5.5)	4.1 (3.2, 5.3)
Alcohol Use Disorder ^c	79 (13%)	16,834 (5.1%)	2.8 (2.2, 3.5)	2.8 (2.2, 3.6)
Substance Use Disorder ^c	173 (28%)	30,060 (9.0%)	3.9 (3.3, 4.7)	3.8 (3.1, 4.6)

^aData is in the 2 years prior to HIV diagnosis or last encounter.^bPresented as mean (SD).^cBased on Encounter ICD-10 Data^dBased on completed Encounters

Table 3. Individual-Level HIV Risk Indicators

Variables	n (%)		Odds Ratio (95% CI)	
	Incident HIV (617; 0.20%)	No Incident HIV (332,646; 99.8%)	Unadjusted	Adjusted
STI History				
Bacterial STI ^{a,c}	58 (9.4%)	7,296 (2.2%)	4.6 (3.5, 6.0)	3.1 (2.2, 4.4)
# Bacterial STIs ^{a,b,c}	1.7 (1.1)	1.4 (0.87)	1.7 (1.5, 1.8)	1.4 (1.2, 1.6)
Chlamydia ^{a,c}	32 (5.2%)	5,301 (1.6%)	3.4 (2.3, 4.7)	1.6 (1.0, 2.5)
# Chlamydia Episodes ^{a,b,c}	0.02 (0.26)	0.02 (0.16)	1.7 (1.4, 2.1)	1.1 (0.82, 1.5)
Gonorrhea ^{a,c}	24 (3.9%)	1,804 (0.54%)	7.4 (4.8, 10.9)	3.2 (1.9, 5.1)
# Gonorrhea Episodes ^{a,b,c}	0.05 (0.25)	0.01 (0.09)	3.5 (2.7, 4.5)	2.0 (1.4, 2.8)
New Syphilis ^{a,c}	8 (1.3%)	201 (0.06%)	21.7 (9.8, 41.4)	8.7 (2.9, 25.1)
# New Syphilis Episodes ^{a,b,c}	0.01 (0.11)	0.001 (0.04)	2.8 (1.7, 4.2)	1.6 (0.79, 3.4)
Trichomonas ^{a,c,d}	67 (11%)	12,128 (3.7%)	3.2 (2.5, 4.1)	2.0 (1.5, 2.6)
Hepatitis / HIV Screening				
Positive Hepatitis C Test ^{a,c}	15 (2.4%)	1,508 (0.45%)	5.5 (3.1, 8.8)	5.0 (2.7, 8.7)
# HIV Screening Lab Episodes ^{a,b,c}	0.85 (0.87)	0.36 (0.66)	1.9 (1.8, 2.0)	1.7 (1.5, 1.8)
Receipt of nPEP ^{a,c}	8 (1.3%)	1,363 (0.41%)	3.2 (1.5, 6.0)	2.3 (1.0, 4.3)
Reproductive / Gynecologic				
PID ^{a,d}	5 (0.81%)	925 (0.28%)	2.9 (1.1, 6.4)	1.7 (0.58, 3.6)
Positive Pregnancy Test ^{a,c}	44 (7.1%)	20,239 (6.1%)	1.2 (0.86, 1.6)	0.85 (0.6, 1.2)
Sexual Assault ^{a,d}	4 (0.65%)	537 (0.16%)	4.0 (1.3, 9.5)	3.5 (1.2, 8.3)

^aData is in the 2 years prior to HIV diagnosis or last encounter.^bPresented as mean (SD).^cBased on laboratory data.^dBased on encounter ICD-10 codes. Trichomonas was ascertained from laboratory data and encounter ICD-10 codes, because testing shifted from wet mount to NAAT during the study period.

Note: Abbreviations: Bacterial STI = Bacterial Sexually Transmitted Infection (Gonorrhea, Chlamydia, and Syphilis), # = Number, nPEP = Post-Exposure Prophylaxis Use, PID = Pelvic Inflammatory Disease