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Using molecular epidemiology to trace the history of the injection-related HIV epidemic in New York City, 1985–2019

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Abstract

Objective.—Unintentional drug poisoning and overdose deaths in New York City (NYC) increased 175% between 2010 and 2017, partly due to the transition from non-injectable opioids to heroin injection. This transition has led to concern of a resurgent HIV epidemic among persons who inject drugs (PWID) in NYC. Thus, we sought to characterize HIV transmission dynamics in PWID.

Design.—Genetic network analysis of HIV-1 public health surveillance data.

Methods.—We analyzed HIV diagnoses reported to public health surveillance to determine the trajectory of the HIV epidemic among PWID in NYC, from 1985 through 2019. Genetic distance-based clustering was performed using HIV-TRACE to reconstruct transmission patterns among PWID.

Results.—The majority of the genetic links to PWID diagnosed in the 1980s and 1990s are to other PWID. However, since 2011, there has been a continued decline in new diagnoses among PWID, and genetic links between PWID have become increasingly rare, while links to non-injecting men who have sex with men and other people reporting sexual transmission risk have become increasingly common. However, we also find evidence suggestive of a resurgence of genetic links among PWID in 2018–2019. PWID who reported male-male sexual contact were not preferentially genetically linked to PWID over the surveillance period, emphasizing their distinct risk profile from other PWID.

Conclusions.—These trends suggest a transition from parenteral to sexual transmission among PWID in NYC, suggesting that harm reduction, syringe exchange programs, and legalization of over-the-counter syringe sales in pharmacies have mitigated HIV risk by facilitating safe injection among future new injectors.

Keywords

HIV; molecular epidemiology; injection drugs; heroin; harm reduction; genetic distance; transmission network

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INTRODUCTION

AIDS was first diagnosed in 1981 in a New York City (NYC) person who injected drugs (PWID), six months before the first reports of *pneumocystis* pneumonia and Kaposi's sarcoma in men who have sex with men (MSM) in Los Angeles and NYC were published in June and July of that year [1, 2]. More cases followed in rapid succession, as did perinatal infections transmitted by mothers who were PWID or had PWID as sex partners. New HIV cases among PWID in NYC peaked in 1990 at 6,608, accounting for 46.6% of the 14,170 new diagnoses of HIV/AIDS that year [3]. Dramatic decreases in the number and proportion of diagnoses attributable to injection drug use (IDU) during the 1990s and early 2000s were coterminous with the introduction of needle exchange, harm reduction, and changes in the heroin economy which brought in high-quality, purer, more easily prepared product [4, 5]. In 2000, New York State introduced an expanded syringe access program, which permitted over-the-counter sales of syringes and decriminalized the carriage of ten or fewer syringes [6, 7].

The current surge in opioid overdose deaths in New York City [8] may represent resurgent injection behaviors fueled by the transition from medical and non-medical use of prescription opioids to heroin, and ultimately to heroin injection [9]. However, the causes for this increase in deaths cannot easily be distinguished from concurrent rise in fentanyl related mortality [10]. Nonetheless, this surge has led to concern that a resurgent HIV epidemic among PWID would follow, as has been seen elsewhere in the United States [11]. In our previous investigation into the characteristics of the NYC molecular transmission network [12], we found that recently diagnosed PWID tended to cluster with other HIV-infected persons at relatively low frequencies, suggesting a reduced transmission risk compared with MSM. In this analysis we used molecular epidemiology to trace the history of the injection-related HIV epidemic in NYC, with attention focused on changes over time in the transmission risk of the people whose viral sequences were genetically similar to those of PWID newly diagnosed with HIV.

METHODS

Data Sources.

The principal data source was the NYC HIV surveillance registry. The registry contains every new diagnosis of AIDS (reportable since 1981) and every new diagnosis of HIV (reportable since 2000 and back-dated as appropriate based on laboratory and epidemiologic evidence), including the protease and reverse transcriptase nucleotide sequence from every HIV genotypic resistance test ordered by a NYC provider. Starting in 2005, HIV sequences from drug resistance testing have been reportable to the NYC Department of Health and Mental Hygiene (DOHMH). There are numerous reasons a missing reported genotype, including lack of drug resistance testing by the primary care physician, low viral load at the time of testing, and failure of laboratories to report full sequencing results to DOHMH. Genotyping reporting completeness in NYC is above the national average, even for PWID in NYC [13]. Data represent a cumulative total of 250,614 cases.

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Populations.

The study included in this analysis was all persons diagnosed and reported with HIV in NYC. The molecular analysis was restricted to those diagnoses with a reported HIV partial *pol* sequence (protease and partial-reverse transcriptase genes) through May 2021.

Variable definitions.

Standard surveillance definitions are used to code data in the HIV registry, are used in the NYC Annual HIV Surveillance Reports [3], and were retained for this analysis. Demographic variable and transmission risk definitions followed CDC conventions for surveillance reporting. Birth sex was classified as male or female. Transmission risk was classified hierarchically based on a documented history of IDU, a history of male-to-male sex contact (MSM), a history of sexual contact with a person of the opposite sex and no history of IDU or MSM, birth to a mother diagnosed with HIV, other (e.g., occupational or iatrogenic transmission), or unknown transmission risk. PWID who reported male-male sexual contact were classified as MSM/PWID.

Network construction.

Due to the large size the genetic sequence database, we used HIV-TRACE [12], a genetic distance-based clustering tool, to align protease/reverse transcriptase sequences to the HIV-1 HXB2 reference sequence (positions 2253–3749) and identify pairs of individuals whose viruses were ≤0.015 substitutions/site divergent, using an ambiguity threshold of 0.015 in HIV-TRACE (i.e., resolved distances between sequences with ≤1.5% ambiguities). This level of divergence is consistent with the distribution of genetic distances between named sexual and injection drug using partners in NYC [6]. Therefore, we refer to these people as potential transmission partners. For individuals with more than one reported protease/reverse transcriptase sequence, the earliest reported sequence was used. We did not exclude sites associated with drug resistance mutations, because their inclusion does not bias network construction using HIV-TRACE [14–16]. Two people were determined to be genetically linked if any of their viral sequences was <0.015 substitutions/site divergent. We excluded problematic genetic sequences (≥5% nucleotide ambiguities) as well as potential laboratory contaminants (sequences that were ≤0.015 substitutions/site divergent from the HXB2 reference sequence) [16].

For each person in the genetic network, we counted the number and transmission risk of their potential transmission partners who had been diagnosed previously. When determining the number of links among people of various transmission risk groups, we down-weighted their genetic links to other risk groups by their network degree centrality (i.e., number of links connecting a given node). This approach prevented biasing results towards densely connected risk groups such as MSM [14, 17, 18].

To determine whether PWID and MSM/PWID were preferentially genetically-linked to anyone reporting IDU, we generated 1000 network random permutations in which individual transmission risk was shuffled among persons clustered in the observed network; the connectivity in the network was held static. Using these permutations, we produced null expectations for genetic linkage across risk groups in the network. For each year between

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1985 and 2019, we compared the observed number of links among PWID and MSM/PWID to the null distribution (i.e., permuted networks). As a robustness check to account for the changing prevalence of PWID and MSM/PWID over time, this network permutation was repeated, stratifying the permutations by year of HIV diagnosis.

Subtyping.

HIV-1 subtyping was performed using COMET v2.2 [19]. Viruses were assigned to B or non-B categorization. Subtype B categorization required 100% bootstrap support for assignment.

Statistical analysis.

We computed frequency statistics of the number of diagnoses and the number genotyped among PWID and MSM/PWID. Yearly data from 2006 to 2019 were analyzed and tabulated. These analyses were conducted using SAS version 9.4 (SAS Institute, Cary, NC).

Ethical review.

This was a routine analysis of existing public health surveillance data and thus not subject to Institutional Review Board approval at the New York City DOHMH. The IRB of the University of California at San Diego, which does not conduct public health surveillance, judged the analysis to be minimal risk [research], and “a waiver of individual authorization for the use of Protected Health Information (PHI) was granted as stipulated by the HIPAA Privacy Rule, 45 CFR 164 section 512(I).”

RESULTS

The number of new HIV diagnoses in non-MSM PWID increased from a cumulative total of 39 in the years prior to 1981 to a peak of 6,608 in 1990 (46.6% of all new diagnoses), and subsequently declined to 761 (16.4% of total diagnoses) in 2000. The 1990s saw the introduction of syringe exchange and harm reduction to New York City. Diagnoses declined further to 293 (6.7% of total diagnoses) in 2005 five years after the introduction of New York State’s Expanded Syringe Access Program. HIV diagnoses declined from 247 in 2006 to 35 in 2019, and the proportion of new diagnoses attributed to injection declined from 6.0% to 2.0% ($p = 0.011$; linear regression; Table 1). The years 2006–2019 represent the period of growing concern about opioid overdose deaths in NYC and the related fear that the HIV epidemic among PWID would reverse course (Table 1).

The MSM/PWID epidemic saw 12 cases diagnosed prior to 1981, grew to 496 cases in 1990 (3.5% of total diagnoses), and declined to 127 in 2000, 88 in 2006 and 25 in 2019 (Table 1).

Between 2016 and 2019, the completeness of genotype reporting (percentage of people with an HIV diagnosis who also have a reported HIV sequence) is slightly lower among non-MSM PWID than non-PWID: 63% versus 67% (Table 2). In contrast, MSM/PWID have higher genotype completeness at 73%. There is no temporal trend in these differences in genotype completeness by year that would suggest a source of bias in the genetic linkage analyses among PWID ($p = 0.439$; linear regression), MSM/PWID ($p = 0.782$), or the population as a whole ($p = 0.764$).

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Subtype B virus was more common among PWID than another other transmission risk group, reaching 97.3%, compared with 92.1% subtype B for the rest of the population (Table 3). Subtype B prevalence was slightly lower in MSM and MSM/PWD and substantially lower in people reporting high-risk heterosexual contact.

Molecular Analysis

To better understand the dynamics of transmission involving PWID and MSM/PWID, we inferred a genetic transmission network comprising 4,285 genetic clusters composed of 18,148 individuals (23.1% of all persons with an eligible sequence in the registry). In total, 875 clusters (20.4%) included at least one PWID or MSM/PWID. We found 128 majority (>50%) PWID and/or MSM/PWID clusters comprising 600 individuals (Supplementary Figure 1); only 12% of these cases were diagnosed after 2010. Between 1985 and 2019, just under half (47.3%) of the potential transmission partners of PWID were PWID or MSM/PWID (Figure 1A); in contrast, only 11.8% of potential transmission partners of MSM/PWID were PWID or MSM/PWID (Figure 1B). MSM/PWID tended to be involved in transmission clusters composing other MSM who did not report IDU, indicating that MSM/PWID represents a risk group distinct from PWID.

The pattern of connectivity of PWID and MSM/PWID in the genetic transmission network changed over the course of the epidemic. Between 1985 and 1994, the majority of molecular links to newly diagnosed PWID were other PWID (in some years, 100% of the links, in others 72–81%) (Figure 1A). The proportion of PWID-to-PWID links began to decrease in 1996, dropping to 43% and 38% in 2002 and 2003, respectively, and stayed in this range until 2012, when it dropped again in frequency. We note there have been years of increased connectivity between PWID, notably 2018 and 2019.

Throughout the first three decades of the NYC epidemic, a PWID or MSM/PWID with newly diagnosed HIV was more likely than expected to be genetically linked to a previously diagnosed PWID or MSM/PWID ($P>0.95$) (Figure 2). However, starting in 2011, PWID and MSM/PWID were no longer preferentially genetically linked to other PWID. The molecular data suggest that by 2002, the majority of transmission partners of PWID were no longer PWID themselves, and by 2011, PWID were no longer preferentially genetically linked to other PWID. We note that trend is less prominent if preferential linkage is considered along among people diagnosed with HIV in the same calendar year (Supplementary Figure 2). Regardless, the most recent years of sampling (2018–2019) suggest an increased linkage from PWID to other PWID, accompanied by a slight uptick in proportional HIV diagnoses among PWID.

DISCUSSION

Steady declines in new HIV diagnoses among PWID in NYC suggest that neither the survivors of the first wave of injection-related HIV, nor the later period in which persons with the dual risk of injection and MSM begin to predominate, appears to have intersected with the prescription opioid abuse epidemic that has swept across the United States during the past decade. In contrast, most people in NYC with genetic links to PWID reported a sexual transmission risk. Deaths due to drug overdose rose citywide 175% between

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2010 and 2017 [8, 9]. But HIV diagnoses have not increased, even while more and more prescription opioid users were believed to have turned to heroin as a less expensive, more readily available alternative to the opioids previously prescribed by their medical providers.

Our characterization of the connectivity of PWID and MSM/PWID in a molecular transmission network showed that PWID and MSM/PWID in NYC are now no more likely to be linked to people who reported using injection drugs than to people with any other transmission risk. Further, our analysis of genetic linkage of PWID and MSM/PWID demonstrates that the latter is a distinct transmission risk group, more resembling other groups reporting high risk sexual activity than those reporting IDU. We present some hypotheses as to this shift in genetic linkage between cases. Although the surveillance system does not capture this information directly, our results are consistent with a scenario in which former prescription users who are not MSM and are transitioning to non-prescription and injection opioids may be intersecting with risk networks characterized by low HIV prevalence and/or lower efficiency transmission than parenteral routes. Widespread, low-threshold access to syringes, injection equipment, and additional harm reduction services may play a role in reducing risk of HIV acquisition among new users. However, even a single instance of crossover between low and high-prevalence pools has the potential to spark a resurgent HIV epidemic related to injection [20–22]. The speed of growth of the resulting resurgence would vary by the risk factor distribution within the newly infected network and the relative efficiency of transmission associated with that risk factor. Our MSM/PWID, for example, currently have potential transmission partners that are predominantly non-injecting MSM.

In 2019, the distribution of transmission risk of potential transmission partners of newly diagnosed PWID spans the risk spectrum. Non-injecting MSM begin to appear as significant proportions of PWID partners in 2005. As the HIV epidemic among PWID in NYC has waned, the proportion of genetic links to people with sexual risk factors has increased. Since 2012, potential transmission partners of PWID are no more likely than by chance to report IDU. This trend suggests that sexual transmission routes may have replaced parenteral transmission as the primary source of new HIV diagnoses among PWID and may in part, given its lower efficiency, be responsible for the slower transmission rate among PWID [23]. That said, the slight uptick we observed in PWID genetically linked to other PWID in 2018 and 2019 support the continued focus on this population. A return to parenteral transmission would motivate renewed prevention efforts and messaging focused on a new cohort of PWID.

Is public health surveillance in a position to detect a new PWID outbreak in NYC, and how quickly would it be able to do so? Surveillance receives weekly laboratory reports of new HIV diagnoses, viral loads, CD4+ counts, and viral genotypes, in addition to provider report forms and positive HIV tests on medical examiner cases. All laboratory tests that do not match to an existing case receive a field investigation with medical record review; average time from laboratory report to completion of investigation and entry into the registry is one month. The department’s “Assess. Connect. Engage.” unit offers partner services and linkage to care interview to all newly diagnosed patients; contact efforts begin on or immediately after the day of diagnosis. Routine time-space surge analyses are conducted

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by ZIP code to locate geographic hot spots. Finally, telephone reports are received from astute clinicians on cases of potential public health interest. Detection of outbreaks remains viable during the COVID-19 pandemic. The combined efforts of surveillance, partner services and clinical reporting suggest that any unusual increase in diagnoses would be detected promptly. Detection of outbreaks remains viable during the COVID-19 pandemic, although partner services have scaled back. Moreover, all new cases occur against a high background prevalence (127,287 persons diagnosed and reported in NYC and not known to be deceased at the close of 2018) that is reflected in most city neighborhoods. The time between infection and diagnosis can be lengthy, and only 14% of new diagnoses are detected during the acute stage, making true outbreak detection challenging even in the best of circumstances [24].

Limitations

Despite the stringent criteria used for matching and expert clerical review of uncertain matches in surveillance, errors occur that affect case dispositions and counts. This limitation is inherent in any analysis using population-based surveillance data. Another limitation is that diagnosis and case ascertainment have been based on continuously evolving diagnostic tests, reporting regulations, and surveillance practices. The HIV registry has been maintained since 1981 and contains AIDS diagnoses going back to 1976. HIV antibody testing has undergone four generational changes since 1985, and Western blot confirmatory testing has been supplanted by supplemental testing that distinguishes between antibodies to HIV-1 and HIV-2. Finally, misclassification of transmission risk may result in under- or over-counting persons in various transmission risk categories and subsequent dilution or overestimation of numbers of PWID vs sexual risk partners over time, despite steady improvement in risk factor ascertainment resulting from the initiation in 2006 of partner services interviews offered to all newly diagnosed persons.

The completeness of the molecular transmission network depends on the completeness of sequence reporting (as mandated by NY State law) and the compliance of physicians with DHHS guidelines [25], which since 2007 have recommended that baseline resistance testing be conducted at the first primary HIV care visit for all newly diagnosed persons. Our genotype prevalence pool represents 59% of all persons diagnosed and reported in NYC with HIV and not known to be dead. The molecular transmission network is therefore missing large proportions of unobserved partners, and only 22% of people with an HIV diagnoses in NYC are clustered with someone else in the NYC prevalence pool (at ≤0.015 substitutions/site) although clustering by MSM overall is higher (~50% in recent diagnosis cohorts) than citywide. This percentage has more than doubled for people with a post-2007 diagnosis [16]. Infections that are undiagnosed may also play a role. The majority of our PWID networks are sparsely populated; those that are not contain high proportions of members with older diagnosis dates. This sparsity is likely an artifact of the epidemiology of the epidemic associated with injecting drug use, which peaked in 1990, of mandatory genotype reporting, which began in 2005, and DHHS guidelines, which did not recommend baseline genotyping until 2007. Thus, in order to be included in our network analysis, a PWID from the original wave of injection-related HIV had to have survived to the introduction of highly active combination antiretroviral therapy in 1996, to the widespread

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adoption of resistance testing in the early 2000s, and to the law requiring genotype reporting in 2005. The 2005 law also directly affects our molecular epidemiology, i.e., both PWID and MSM/PWID experience an inflection point in the following year, with the PWID-to-PWID and MSM/PWID-to-MSM/PWID links diminishing and the links with other risk categories, most prominently non-injecting MSM, beginning to increase as genotyping became more widespread, and showing more prominently among non-injecting MSM, who among all transmission risk groups are most likely to be in care and thus to have at least the opportunity to be genotyped.

Conclusion

The opioid epidemic that has engulfed many parts of the United States has not yet intersected with the HIV epidemic among PWID and MSM/PWID in New York City, among whom diagnoses and deaths continue to decline. As of the end of 2019, a cumulative total of 14,351 PWID and 3,285 MSM/PWID had a reported HIV diagnosis in NYC and are presumed to be living. Continuous monitoring by surveillance of new diagnoses and transmission patterns, coupled with real-time partner and linkage to care services for new diagnoses, have the potential to rapidly identify a new surge of injection-related diagnoses. Furthermore, molecular analyses could determine whether any such future surge is related to the original wave of injection-related infections or represents a new, distinct wave of transmission. A potential new wave infection-related infections would require revisiting current HIV prevention policies that previously stanched the spread of HIV via non-sexual routes in NYC.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments.

The study was approved by the University of California (UC) San Diego and NYC DOHMH Institutional Review Boards. All data were collected through standard surveillance protocols and are subject to New York State Redisclosure Law Articles 21 and 27-F. These data cannot be made publicly available and were shared between UC San Diego and NYC DOHMH via a Data Use Agreement.

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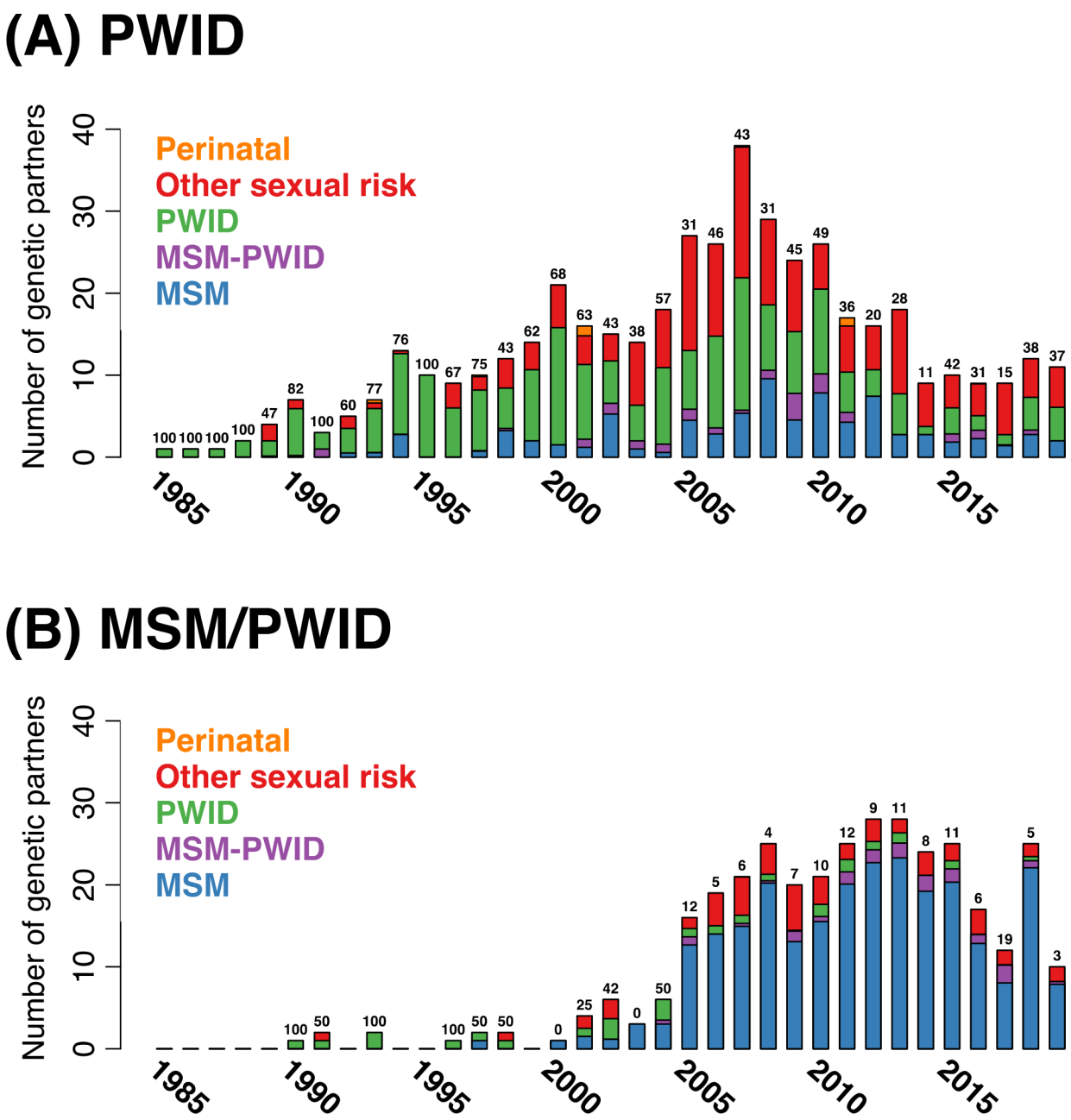

Figure 1. The transmission risk of people connected to PWID in NYC, 1985–2019.

(A) depicts proportion of genetic links from individuals with various transmission risk to newly diagnosed PWID. (B) depicts this linkage to newly diagnosed MSM/PWID. Numbers on bars indicate the percentage of total links that connect PWID and MSM/PWID to other PWID and MSM/PWID. For this display, other sexual risk comprises people who reported high-risk heterosexual contact and people with an unknown transmission risk.

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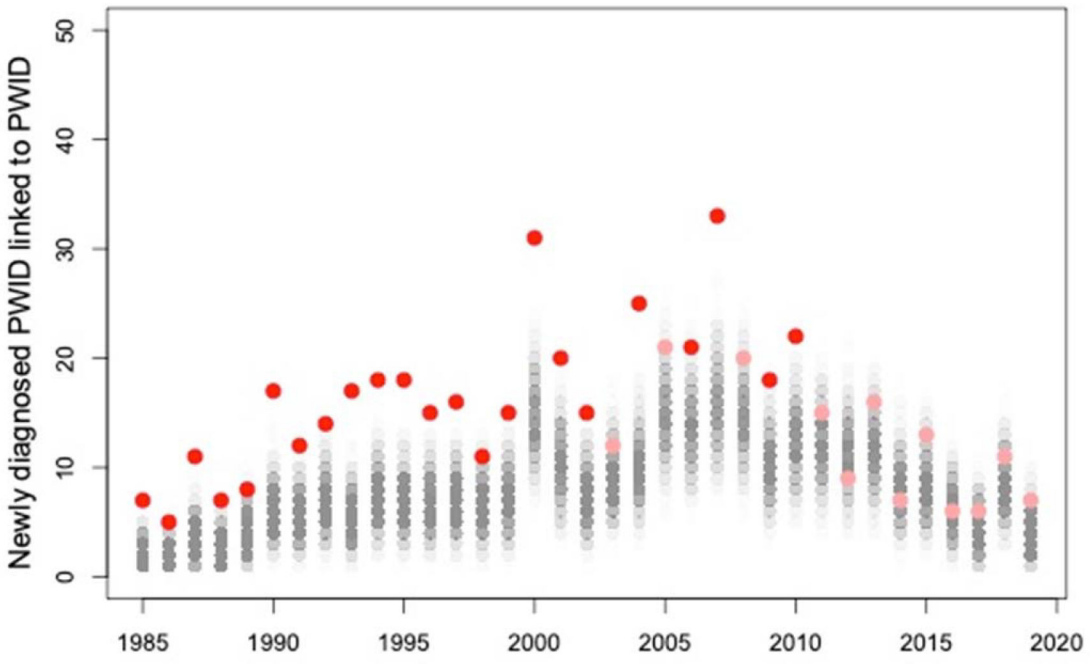

Figure 2. Permutation analysis of genetic linkage between PWID and MSM/PWID. Red circles indicate the number of PWID and MSM/PWID genetically linked to another PWID and/or MSM/PWID diagnosed in a previous year.

Black circles depict the null expectation from 1000 random permutations of the network. Dark red shading indicates an observed value in the network greater than in 95% of the permuted networks. Similar results are seen when permutations are restricted to individuals diagnosed in the same calendar year (Supplementary Figure 2).

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Table 1.

HIV diagnoses among PWID, including MSM/PWID, 2006–2019*, in New York City

Year	Total diagnoses	PWID (non-MSM) diagnoses		MSM/PWID diagnoses	
		N	%	N	%
2006	4148	247	6.0%	88	2.1%
2007	4091	229	5.6%	88	2.2%
2008	3952	205	5.2%	73	1.8%
2009	3584	138	3.9%	60	1.7%
2010	3193	111	3.5%	65	2.0%
2011	3097	99	3.2%	68	2.2%
2012	2898	82	2.8%	64	2.2%
2013	2716	54	2.0%	61	2.2%
2014	2643	56	2.1%	44	1.7%
2015	2390	44	1.8%	48	2.0%
2016	2237	33	1.5%	36	1.6%
2017	2075	33	1.6%	24	1.2%
2018	1904	40	2.1%	47	2.5%
2019	1763	35	2.0%	25	1.4%

*For events reported by May 27, 2021

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Table 2.

HIV genotype completeness among newly diagnosed among PWID, including MSM/PWID, 2006–2019*, in New York City

Year	All diagnoses			PWID (non-MSM)			MSM/PWID		
	Total	Genotyped		Total	Genotyped		Total	Genotyped	
	N	N	%	N	N	%	N	N	%
2006	4148	2595	63%	247	142	57%	88	61	69%
2007	4091	2620	64%	229	141	62%	88	62	70%
2008	3952	2594	66%	205	120	59%	73	54	74%
2009	3584	2450	68%	138	87	63%	60	45	75%
2010	3193	2178	68%	111	78	70%	65	44	68%
2011	3097	2121	68%	99	71	72%	68	52	76%
2012	2898	1992	69%	82	54	66%	64	47	73%
2013	2716	1944	72%	54	41	76%	61	46	75%
2014	2643	1828	69%	56	34	61%	44	33	75%
2015	2390	1651	69%	44	26	59%	48	40	83%
2016	2237	1500	67%	33	24	73%	36	25	69%
2017	2075	1403	68%	33	21	64%	24	18	75%
2018	1904	1183	62%	40	30	75%	47	35	74%
2019	1763	1088	62%	35	20	57%	25	15	60%

* For events reported by May 27, 2021

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Table 3.

HIV-1 subtype B frequency by transmission risk.

Transmission risk	Total	Subtype B	
		N	%
MSM	30491	28,846	94.6
Heterosexual	15981	14,100	88.2
Unknown risk	13990	12,654	90.5
PWID	10169	9,897	97.3
MSM-PWID	2419	2,340	96.7
Perinatal	1666	1,538	92.3
Transgender with sexual risk	1305	1,230	94.3
Other risk	120	115	95.8