**Supplementary Appendix: Expanding Medicaid to Reduce HIV Transmission in Houston, Texas: Insights from a modeling study**

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# A.1. Introduction

This Supplementary Appendix describes additional technical components of the agent-based network model (ABNM) described in the main body of the manuscript, particularly focusing on providing a more detailed explanation of the model processes along with the parameters and data sources. Computer programs and supporting documentation are available in a publicly available GitHub repository (<https://github.com/khanna7/BARS/tree/houston-paper>).

The modeling methods utilized a structure similar to prior ABNMs of HIV transmission[1–5] following the following steps:

* An initial population was generated, as described in Section A.2 below.
* Main and casual partnership networks were simulated on this initial population. The modeling process is described in Section A.3.
* Baseline HIV epidemics, resulting from HIV infections transmitted through networks of main and casual partnerships, were simulated to capture features of the epidemic among young (18-34 years) black men who have with men (YBMSM). The steps mentioned in the main body of the paper to model the HIV epidemics are described in Section A.4.
* Parameters for which the available estimates are variable or might be biased and sensitivity analyses that were conducted to produce reasonable estimates are described in Section A.5.
* Key model outputs were produced when the model was simulated with parameter estimates derived in Section A.6.
* At the conclusion of the baseline phase, interventions were simulated, including all of the key model processes. Additional information on these interventions can be found in Section A.7.

# A.2 Initial Population

The initial population consisted of 10,000 individuals, uniformly distributed between the ages of 18-34 at the start, consistent with empirical data[6]. As time evolved, the rates of departure and entry of individuals were set so that the overall population grew slowly consistent with observed growth rates as per census data**.** The population was randomly seeded with 10% HIV prevalence at the start, sufficient to sustain an epidemic, as has been done in previous agent-ABNM studies to design HIV interventions [2,4,5,7]. The model was simulated over a long period (100 years) to allow epidemic outcomes to become consistent with empirical data; similar “burnin” periods have been instituted in previous ABNM studies [2,4,5,7]. The simulations incorporated a number of demographic, network, biological, and behavioral features mentioned in the Methods section of the main body of the manuscript; these features are described in detail below.

# A.3 Models for Main and Casual Partnership Networks

The theoretical framework for modeling main and casual partnership networks is based upon the exponential random graph models (ERGMs), described elsewhere [8], and implemented in the *statnet* suite of packages [9]in the R programming language.

*Main and Casual Partnerships.* Two different partnership networks were simulated in the model. The log-odds of formation of each partnership type were dependent upon the number and distribution of existing partnerships within the network, and the absolute difference in the relative ages of partners for each partner type. The log-odds of the dissolution of each partnership were derived on estimates of the mean partnership duration.

The log odds of the formation of partnerships in both the main and casual networks was specified as  
where and are two individuals in the network; is a time step (simulated forward in daily units in this model), is the previous time step; is the number of edges; is the number of nodes with degree , specified for degrees 0, 1, and 2 in both the main and casual networks; and is the difference in the absolute values of the ages of individuals individuals who are in main and casual edges multipled by the number of main and casual partnerships The functions corresponding to each of these model terms represent the change in their value corresponding to the “toggle” of one dyad (defined as removing one existing tie, or adding a non-existent one); the change statistic functions are needed to estimate the coefficients , and , corresponding to the edges, degree distribution and age mixing terms respectively. These coefficients are estimated using Markov Chain Monte Carlo techniques [10], as per the algorithmic routines contained in the *statnet* package [11].

For both main and casual networks, the persistence of each partnership was defined as  
where is the coefficient associated with the dissolutoin of one tie. The model simulations, described in Section 4 below, incorporated the formation and dissolution coefficients derived here.

# A.4 Simulating Baseline Epidemics

The estimated models for main and casual partnership networks were simulated forward in daily time steps. These baseline epidemics were simulated for a long burnin period, allowing the model interdependencies sufficient time to equilibrate (100 years in this case). Each step of the simulation included the following processes: (1) entry of individuals into the modeled population; (2) departures of individuals from the modeled population;(3) modeling main and casual sexual networks; (4) HIV testing and diagnosis; (5) temporal evolution of CD4 counts; (6) temporal evolution of HIV RNA (“viral load”); (7) dynamics and effects of antiretroviral (ART) use; (8) dynamics and effects of preexposure prophylaxis (PrEP)use, (9) incidence of external HIV infections; and, (10) transmission of infection within serodiscordant main and casual partnerships. The estimation of the demographic, biological, and treatment parameters required to model these processes is described below.

A.4.1 Arrivals: Individuals aged into the model at 18 years. The arrival of new agents in the model was simulated as a Poisson process, with the mean set to 2.0. Thus, the entry rate was empirically determined, to balance the various departure processes (see Section A.4.2 below), so that the population grew at approximately the same growth rate as the population of interest (see Section A.4.2 below).

A.4.2 Departures and net population growth: Individuals departed from the model on account of the following reasons:

1. Aging out of the model at age >34 years;
2. HIV-uninfected individuals experience mortality, based on daily probabilities estimated from CDC Wonder data [12].
3. Untreated individuals with HIV infection had a maximum lifespan of 4279 days (approximately 11.7 years), estimated by summing the lengths of acute, chronic, and late-stage infection (details in Section A.4.10).
4. HIV-infected individuals on ART experienced an increase in the age-specific mortality rates, based on individual CD4 counts, in accordance with published data [13]. The increase in the daily mortality rates for HIV-infected individuals is below.

|  |  |
| --- | --- |
| **Table A.1. Increased mortality rates for HIV-infected individuals who are not using ART.** | |
| CD4 count (cells/µl) | % increase in age-specific mortality rates |
| < 50 | 51% |
| 50 – 99 | 37% |
| 100 – 199 | 26% |
| 200+ | 0% |

A.4.3 Sexual network structure: Two different sexual networks within this population were modeled, based on two types of partnerships: “main” and “casual”. The formation and dissolution processes of both partnership types were set such that their cross-sectional structure remained consistent with empirical data. The key parameters for main and casual partnerships, estimated for a given day, were: the mean number and distribution of partnerships, the mean partnership duration, and the mean of the absolute difference in ages for partners.

Sexual networks were dichotomized as “main” and “casual”. The underlying cohort data [14,15] used to parameterize these networks also contained information on “exchange” partners; these partnerships were modeled within the “casual” partnership typology. The same cohort data were used to estimate other parameters used to model the sexual networks, namely the mean number of cross-sectional main and casual partnerships, the distribution of the number of main and casual partnerships (0, 1, 2), and age-mixing. The degree distributions were estimated by computing overlaps in the dates of first and last sex between the study respondent and each of their partners (each respondent reported on up to 5 partners in the last six months). Because the age ranges of agents in our model spanned a relatively narrow range (18 – 34 years), the age-mixing parameter was estimated as the mean of the absolute values of the difference in the ages of the partners, in contrast to other models that were developed for broader age ranges and used the difference in the square-roots of partner ages [1,16]. The durations of mean and casual partnerships were estimated using the last partner reports from NHBS data [17].

A.4.4 Temporal evolution of CD4 counts: The CD4 count of uninfected men was assumed to be constant at 916 cells/µl [18]. Upon HIV seroconversion, an individual’s CD4 count declined piecewise linearly, using a deterministic model where CD4 count was dependent on age at seroconversion, sex, and time since seroconversion, as described by Pantazis et al [19]:  
where was the CD4 count at years after seroconversion, was 23.53; was an indicator of African descent (set equal to 1 here), with a coefficient estimated at -0.76; was an indicator for female (set equal to 0 here), estimated at -1.49 and estimated at 0.34; was the age at seroconversion, withcoefficient estimated at: 0 for , -0.1 for

A.4.5 Temporal evolution of HIV RNA (“viral load”): The viral load trajectory was modeled deterministically. For each infected, untreated individual, viral load was expressed as a six-parameter curve with a steep increase from 0 to peak viremia at 6.17 on the log10 scale over the first 45 days of infection, followed by a decline to the viral set point of 4.2 log10 over the next 45 days [20]. This viral set-point is maintained for the next 3550 days [21]. There is a final steady increase in viral load during late stage infection, where the viral load rises to 5.05 log10[22], over the course of 728 days [21]. The viral load of individuals who initiated ART decreased until treatment is interrupted, at which time the viral load increased again, as explained above, as long as treatment remained interrupted.

A.4.6 HIV testing and diagnosis: A heterogeneity of testing behaviors were modeled. Consistent with population-based cohort data, 7.8% of individuals <26 years and about 2.3% of individuals >=26 years of age were designated as never testing [23]. The remaining individuals were classified into categories defined by the frequency of testing. An individual was diagnosed if, at the time of testing,they had been infected for longer than the detection window of the test (i.e. 22 days[24]). The distribution of the number of HIV tests in the last two years is given in Table A.3 below. Each individual was assigned to one of these categories, and a daily probability of testing for them was computed based on a number of tests parameter that was sampled from the discrete number of tests belonging to that interval.

A.4.7 Dynamics of ART use: At the time of infection, each individual who was eligible for treatment was assigned to one of four states of ART adherence: adherence levels at the two extremes, almost always adherent (A), almost never adherent (N), and two categories of partial (P) adherence: usually(P+) and sometimes (P-). The distribution of ART adherence was estimated from longitudinal cohort data in the uConnect study [14,15]. Of the HIV-positive individuals completing all three visits (n=93), 32% were virally suppressed (<200 copies log viral load RNA) at all three visits (classified as category A defined above), 28% were suppressed at two visits (classified as category P+ above), 30% were suppressed at one visit (classified as category P- defined above), and 10% were never suppressed (classified as category N, defined above).

After 30 days, each person’s adherence for the next 30-day window was assessed, consistent with these four possible adherence states: 0.95 for A, 0.67 for P+, 0.33 for P-, and 0.05 for N. This cycle was repeated every 30 days given typical medication prescription patterns. (Typical medication prescription patterns for ART include a 30-day supply and 2-3 refills depending upon the client’s needs. This assumption was made in conjunction with our panel of HIV providers who care for YBMSM as well as the DHHS guidelines for ART treatment.)The distribution of times between diagnosis and ART initiation were empirically estimated from cohort data [14,15]and are given in Table 1 in the main body of the manuscript.

A.4.8 Dynamics of PrEP use: To model PrEP use, individuals were classified into four categories of adherence as per published data: 21.1% of men took 0 pills/week (non-adherent), 7.0% took <2 pills/week (low adherence), 10.0% took 2–3 pills/week (moderate adherence), and 61.9% took 4+ pills/week (higher adherence)[26]. PrEP use is assumed to reduce HIV infection probability in these adherence groups by 0%, 31%, 81%, and 95%, for non, low, moderate, and high adherence, respectively, in accordance with previous modeling work [27]. On average, PrEP uptake (i.e., the proportion of HIV-negative individuals using PrEP at any given time) was about 13.7% [14,15]. To model this, consider probability of stopping PrEP on any given day. If is the number of HIV-negatives and is the proportion of HIV-negative individuals using PrEP, then on any given day is the number of users who stop PrEP. From the above definitions, it also follows that the number of HIV-negatives who are not using PrEP is . If we define as the probability that any non-user initiates PrEP on a given day,

to maintain the same number of users at any given step, implying that A selection probability of as defined above was set for HIV-negative individuals to initiate PrEP, enabling the model to maintain a specified proportion of HIV-negative individuals on PrEP.

For the interventions that prioritized serodiscordant couples and network position, the selection procedure for new PrEP initiators was implemented in two steps. In the first step, the process to maintain baseline PrEP rates was operationalized by assigning a selection probability for new PrEP initiators, as described above. In the second step, additional individuals were sampled from the pool of the target intervention group (serodiscordant couples or individuals in the highest scoring network positions) to make up the difference in the baseline and target levels of PrEP uptake. Selection from the target intervention group was also implemented probabilistically; the numerator of this probability was computed by considering the number of individuals required to make up the difference in the baseline and target levels of PrEP uptake, and the denominator was the total number of HIV-negative individuals.

To estimate PrEP retention, data reported in a study from two Southern US cities was used [27]. This study found that approximately 60% of the population was retained on PrEP at 6 months. Since the geometric distribution is often used to model waiting times, we applied it to model PrEP retention periods. We found that a geometric distribution with a mean of 200 days (approximately 6.6 months) produces about 60% retention after 6 months. Thus, the PrEP retention period was modeled as a geometric distribution with a mean of 200 days.

A.4.9. Incidence of external HIV Infections: To model HIV infections from individuals not in the defined population (i.e. “external” infections), the following parameters were considered: (1) the overall incidence rate among YBMSM, estimated at 5-7 per 100 person years (py) from two different population-based Houston cohorts of YBMSM [16,29,30]; (2) the proportion of this overall HIV incidence that consists of new HIV infections transmitted from older BMSM to YBMSM and vice versa, estimated at 28% [31]; the proportion of infections that are transmitted to YBMSM from older BMSM, assumed to be between 50% and 80%. Thus, the lower bound for a total number of infections incident externally among YBMSM in the model was 28%×50%×5 per 100 py = 0.70 per 100 py, and the upper bound was 28%×80%×7 = 1.56per 100 py. The daily probability for each HIV-negative person to get externally infected thus ranged between and Thus, this probability was used to conduct a Bernoulli trial for simulating an externally incident infection for each HIV-negative person at each time step. It was also assumed that the risk of getting externally infected was uniformly distributed with respect to age because this assumption produced simulated outcomes that were most consistent with empirical data.

Based on sensitivity analyses, we assumed that the risk of getting externally infected was uniformly distributed with respect to age because it produced the most consistent outcomes with the empirical data. External infections from women [32]and non-Black MSM [31] were not included due to evidence that very few infections among YBMSM are linked to either of these populations[32].

A.4.10. Transmission of HIV infection: Transmission of HIV infections through anal intercourse between HIV-infected and HIV-uninfected individuals within main and casual partnerships was modeled at each time step of the simulation. The probability of transmission during each sex act was derived using a number of different attributes, including stage of infection (determined by time since seroconversion) and viral load (in turn determined by the ART status of the infected partner) of the infected individuals, PrEP use by the uninfected partner, condom use at the time of intercourse (which reduced probability of transmission by 80%), and circumcision status of the uninfected partner.

Three stages of HIV infection were considered: acute, defined as lasting for 90 days from the onset of infection[20]; chronic infection that lasts for 3460 days [21]; and late-stage infection for 728 days [21]. Sensitivity analysis based on estimates from empirical data were previously used to derive another key parameter, the relationship between chronic stage and infectivity [32] (see Table A.5 for further details).

# A.5 Model Input Parameters

Primary parameters for model inputs are listed in Table A.2 below.

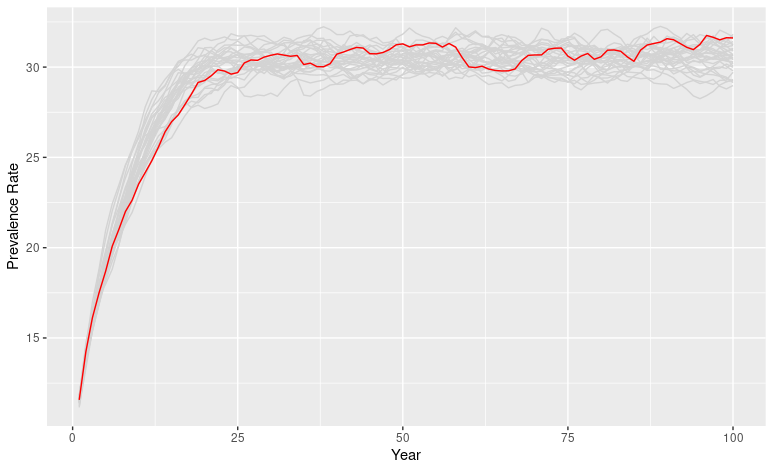
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| --- | --- | --- |
| **Table A.2: Parameters to Model HIV Transmission among young Black men who have sex with men (YBMSM), Houston, TX** | | |
| **Demography** | | |
| **Parameter** | **Estimate** | **Source** |
| Age range | 18 – 34 years | Defined population of interest |
| HIV prevalence among 18-year old persons at entry into the model | 1% | [33] |
| Mortality for uninfected individuals | Age-specific mortality rates for Houston; achieving 35 years in age | CDC[34] |
| **Sexual Behavior** |  |  |
| Mean partnership duration (days) | Main: 970 Casual: 388 | NHBS[35] |
| Mean number of partnerships (per person) on any given day. | Main: 0.42  Casual: 0.46 | YMAP[36] |
| Frequency of sex (per partnership per day) | Main: 0.189  Casual: 0.053 | Per previous analysis[32] |
| Proportion of acts where condoms are used in serodiscordant partnerships | Main:  never: 0%; rarely: 2.9%; sometimes: 17.6%;  usually: 20.9%; always: 58.6%.   Casual:  never: 10.3%; rarely:6.9%; sometimes: 10.3%; usually: 13.7%; always: 58.4% | YMAP[36] |
| Distribution of number of sexual partnerships on any given day | Main: 0 (63.7%), 1 (34.0%), 2 (1.9%) Casual: 0 (59%), 1 (26%), 2 (10.4%) | YMAP[36] |
| **Testing and Diagnosis** | | |
| Detection window of test | 22 days | [37] |
| Testing among HIV-negative/undiagnosed individuals | Persons <26 years:5.4% never test.  Persons ≥ 26 years: 2.7% never test. | NHBS[35] |
| HIV testing frequency (number of tests in the last two years) | |  |  | | --- | --- | | 1-2 tests: | 0.427 | | 3-4 tests: | 0.294 | | 5-6 tests: | 0.144 |   6+ tests: 0.134 | YMAP [36] |
| **Linkage to Care and ART** | | |
| Time between HIV diagnosis and ART initiation | 0-1week: 3.8%  1week-1month: 10.03%  1-3months: 21.1%  3-6 months: 22.9%  6months-1year: 24.5%  1-2years: 14.5%  2-5years:3.1% | NHBS[35] |
| CD4 evolution for ART initiated | CD4 count recovers by 15 cells/µl every month until pre-infection level or for 3 years, whichever is first | [38,39] |
| Viral load evolution for ART initiated | Declines to 200 copies/ml in 30 days | [40] |
| Distribution of ART adherence | Never: 3%  Sometimes: 12%  Usually: 25%  Always: 60% | YMAP[36] |
| **PrEP Use** |  |  |
| Number of PrEP users | 20% of HIV-negative persons at any given time | NHBS[35] |
| Time that a PrEP user is retained on PrEP | 200 days (6.5 months) | [27]and see Appendix Section 4.8. |
| Adherence to PrEP, for those who have initiated | None (0 pills/week): 21.1%  Low (<2 pills/week): 7.0%  Moderate (2–3 pills/week): 10.0%  High (4+ pills/week): 61.9% | [41] |
| Reduction in transmission associated with levels of PrEP adherence | Non-adherence: 0%; low: 31%; moderate: 81%; high: 95% | [42] |
| **Biology** |  |  |
| CD4 in uninfected men | 916 cells/µl | [43] |
| CD4 decline in individuals with untreated HIV | As per a linear model | [44] |
| Acute stage duration | Infection to peak viremia = 45 days; peak viremia to viral set point = 45 days | [20] |
| Chronic stage duration | 3550 days | [45] |
| Late stage (AIDS) duration | 728 days | [45] |
| Level of peak viremia | 6.17 log | [22] |
| Viral set point | 4.2 log | [22] |
| Max. late-stage viremia | 5.05 log | [22] |
| **Infection Transmission** | | |
| Per condomless sex act (chronic stage) | 0.00092 | [32] |
| Increase in infectivity corresponding to one unit increase in log viral RNA | 2.89 | [46] |
| Infectivity multiplier for acute infection | 5 | [32] |
| Infectivity multiplier for late-stage infection | 1 | [32] |

# A.6 Sensitivity Analysis of Key Model Parameters

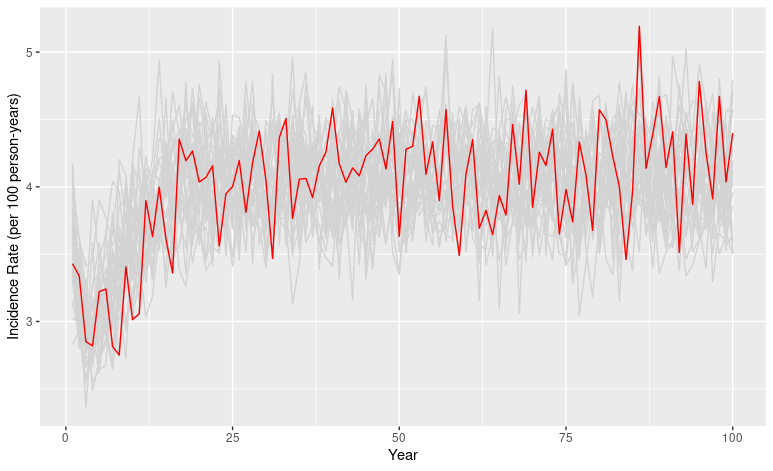
Figure A.1 shows the prevalence and incidence trajectories of 30 stochastic model runs with the parameter estimates provided in Table A.3 above. To validate the simulation model, a number of outputs were compared to empirically derived target values, as described in Table A.4 below. HIV prevalence and incidence outcomes for each of the 30 burnin runs, averaged over the last year, are shown in Table A.5.

**Figure A.1: Prevalence (A) and incidence (B) trajectories over thirty baseline model runs with the parameters listed in Table 1.\***

**(A) Prevalence trajectories**



**(B) Incidence trajectories**



\* The red curve denotes the one instance that was selected for the intervention analyses presented in the main text; the gray lines represent the other 29 instances. The y-axis shows HIV prevalence (Figure A) and incidence (Figure B) at a given time, and the x-axis shows the given year.

|  |  |  |  |
| --- | --- | --- | --- |
| **Table A.3: Comparison of simulated and target values for key model inputs.** | | | |
| **Parameter** | **Simulated statistics** | **Target statistics** | **References (for target statistics)** |
| Total number of main and casual partnerships per person | 0.81 | 0.87 | NHBS[35] |
| Population Growth Rate | 1.1% per year | 1.1 – 1.2% per year | World Population Review[47] |
| Mean duration of main partnerships | 894 days | 970 days | NHBS[35] |
| Mean duration of casual partnerships | 378 days | 388 days | NHBS[35] |
| HIV Testing Frequency (number of tests in past 2 years) | |  |  | | --- | --- | | 1-2 tests: | 0.427 | | 3-4 tests: | 0.294 | | 5-6 tests: | 0.144 |   6+ tests: 0.134 | |  |  | | --- | --- | | 1-2 tests: | 0.457 | | 3-4 tests | 0.299 | | 5-6 tests: | 0.109 |   6+ tests: 0.134 | YMAP [36] |
| ART uptake | 63% of HIV-positive YBMSM | 67% of YBMSM living with HIV | [48] |
| PrEP retention | 6.5 months | 6.7 months | [27]and see Appendix Section 4.8. |
| Per cent HIV-negatives on PrEP | 19.6% | 20% | [14,15] |

|  |  |  |
| --- | --- | --- |
| **Table A.4: Key model outputs produced with baseline model run for 100 years.** | | |
| **Simulation number** | **Overall prevalence (%)** | **Overall incidence rate (per 100 py)** |
| 1 | 30.69 | 3.51 |
| 2 | 30.51 | 4.29 |
| 3 | 30.68 | 4.71 |
| 4 | 30.98 | 4.36 |
| 5 | 30.40 | 3.96 |
| 6 | 30.04 | 3.50 |
| 7 | 31.67 | 4.15 |
| 8 | 28.98 | 4.20 |
| 9 | 29.22 | 3.71 |
| 10 | 31.24 | 3.59 |
| 11 | 30.63 | 4.30 |
| 12 | 31.24 | 3.96 |
| 13 | 30.27 | 4.71 |
| 14 | 29.79 | 3.64 |
| 15 | 29.27 | 3.60 |
| 16 | 29.31 | 4.12 |
| 17 | 30.28 | 4.01 |
| 18 | 31.81 | 4.80 |
| 19 | 31.61 | 4.40 |
| 20 | 30.89 | 4.15 |
| 21 | 29.99 | 3.96 |
| 22 | 30.53 | 4.00 |
| 23 | 30.51 | 4.11 |
| 24 | 29.71 | 4.36 |
| 25 | 30.24 | 4.24 |
| 26 | 31.41 | 4.09 |
| 27 | 29.14 | 3.86 |
| 28 | 30.70 | 4.57 |
| 29 | 31.19 | 3.73 |
| 30 | 31.05 | 4.57 |

Since all of the above instances showed a reasonable match with our target statistics, one instance was randomly chosen (simulation number 19 from Table A.4) for further intervention analysis.

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