Test and Treat DC: Forecasting the Impact of a Comprehensive HIV Strategy in Washington DC

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Background. The United States and international agencies have signaled their commitment to containing the human immunodeficiency virus (HIV) epidemic via early case identification and linkage to antiretroviral therapy (ART) immediately at diagnosis. We forecast outcomes of this approach if implemented in Washington DC.

Methods. Using a mathematical model of HIV case detection and treatment, we evaluated combinations of HIV screening and ART initiation strategies. We define current practice as no regular screening program and ART at CD4 counts $\leqslant$350 cells/μL, and we define test and treat as annual screening and administration of ART at diagnosis. Outcomes include life expectancy of HIV-infected persons and changes in the population time with transmissible HIV RNA levels. Data, largely from Washington DC, include undiagnosed HIV prevalence of 0.6%, annual incidence of 0.13%, 31% rate of test offer, 60% rate of acceptance, and 50% linkage to care. Input parameters, including optimized ART efficacy, are varied in sensitivity analyses.

Results. Projected life expectancies, from an initial mean age of 41 years, are 23.9, 25.0, and 25.6 years for current practice, test and treat, and test and treat with optimized ART, respectively. Compared with current practice, test and treat leads to a 14.7% reduction in time spent with transmissible HIV RNA level in the next 5 years; test and treat with optimized ART results in a 27.3% reduction.

Conclusions. An expanded HIV test and treat program in Washington DC will increase life expectancy of HIV-infected patients but will have a modest impact on HIV transmission over the next 5 years and is unlikely to halt the HIV epidemic.

Recent reports from the District of Columbia Department of Health describe an human immunodeficiency virus (HIV) epidemic in Washington DC comparable to that in East Africa [1, 2]. Three percent of adults in the United States capital are known to be living with HIV; many more remain undiagnosed and unable to obtain either lifesaving care or counseling to reduce the spread of infection [1].

In response to the continuing spread of HIV in the United States and internationally, both the United States National Institutes of Health and the World Health Organization have committed to implementing and evaluating universal testing and treatment for prevention of HIV infection [3, 4]. This comprehensive approach aims to benefit infected individuals through early detection while decreasing their subsequent HIV transmission by lowering community levels of HIV RNA [5]. The approach is motivated by the persistent evidence that HIV prevention efforts to date have not controlled HIV transmission, with the view that combinations of interventions will be required to contain the epidemic [6]. A recent joint announcement from the Washington DC Department of Health and the National Institutes of Health highlights a piloting of a test-and-treat strategy as part of a new, $26.4 million, 2-year partnership [7].

This article aims to assist in the evaluation of a test

Received 12 January 2010; accepted 18 April 2010; electronically published 9 July 2010.

Presented in part: 5th International AIDS Society Conference on HIV Pathogenesis, Treatment, and Prevention. Cape Town, South Africa. 19–22 July 2009. Abstract LBPEC04.

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Clinical Infectious Diseases 2010; 51(4):000–000

1058-4838/2010/5104-00XX$15.00
DOI: 10.1086/655130

and treat strategy. We use a widely published computer model of HIV infection to project how program performance, including overall rates of test offer, acceptance, and linkage to care, will affect both individual patient and population-wide outcomes in Washington DC.

METHODS

Analytic overview. We use the Cost-effectiveness of Preventing AIDS Complications (CEPAC) Model of HIV screening and treatment to evaluate the impact of alternative HIV screening and treatment strategies. We examine 8 strategies, including current screening practice without regular screening and without antiretroviral therapy (ART) (for comparison), no regular screening with ART initiation at CD4 count ⩽350 cells/μL, and all 6 combinations of the following: (1) routine HIV screening once, every 3 years, or annually and (2) ART starting at CD4 counts ⩽350 cells/μL or immediately at diagnosis. We define current practice as no regular screening combined with ART at CD4 counts ⩽350 cells/μL; we define the test and treat strategy as annual screening with ART initiation at HIV diagnosis. In a final, ninth strategy, we examine the impact of test and treat with optimized ART regimens, with improved adherence and suppression efficacies higher than those reported in clinical trials.

For each strategy, we examine several scenarios of screening performance, characterized by different probabilities of test offer, test acceptance, and linkage to care for newly identified HIV cases. Model simulations result in projections of life expectancy for HIV-infected individuals as well as their mean CD4 count at detection. We also report the population impact of each strategy, measured over a 5-year horizon, by the amount of HIV-infected population time spent with a transmissible HIV RNA level (>500 copies/mL).

The CEPAC model. The CEPAC model is a mathematical simulation model that projects the clinical course of HIV disease and the epidemiological trajectory of infection on the basis of alternative assumptions about the time of HIV detection and treatment initiation [8–11]. The model, composed of screening and disease modules, is used to consider how a program of accelerated detection and immediate ART might suppress HIV RNA levels among infected patients, thereby slowing the progress of their own HIV disease and decreasing its transmission to others.

The screening module. In the CEPAC model, HIV infection may be detected or undetected. However, HIV-infected patients are only eligible for HIV-related care and ART on successful disease detection and linkage to care. Without detection and linkage, infected patients progress with untreated HIV disease. The screening module is used to determine when and how HIV-infected patients receive a diagnosis, via 1 of the following 3 mechanisms: (1) "background" screening, which currently occurs in a variety of settings in the United States; (2) development of an AIDS-related opportunistic infection; or (3) a routine HIV screening program. Current practice (without regular HIV screening) is defined as detection via mechanisms 1 and 2 but not 3. We assume that detection via mechanisms 1 and 2 leads invariably to HIV care, whereas there may be imperfect linkage and loss to follow-up among patients detected via mechanism 3. This leads to conservative estimates with respect to the value of a regular screening program. To describe the characteristics required for completion of a routine screening program, we define the performance index for a screening program as that program's joint probability of test offer, test acceptance, and linkage to care for each encounter.

Among those with HIV infection, demographic and clinical characteristics, including age, sex, CD4 count, and HIV RNA level, are defined to be representative of the population with prevalent HIV infection reported from Washington DC. The CEPAC model determines if and when new cases of HIV infection occur on the basis of user-specified incidence rates and the demographic characteristics of the Washington DC population. Further details of the screening module have been published elsewhere [11–13].

The disease module. In the disease module, HIV-infected patients are characterized by health states defined by current CD4 count and HIV RNA level; transition between health states occurs in monthly cycles. In the absence of HIV case detection and treatment, HIV-infected patients follow a trajectory of HIV RNA–dependent monthly CD4 count decrease, resulting in an increased risk of opportunistic infections and HIV-related mortality [8, 10]. Patients also face risk of death due to age-, sex-, and race-adjusted background mortality [1, 14, 15].

Patients identified with HIV infection and successfully linked to care are eligible to start ART and opportunistic infection prophylaxis if initiation criteria are met [16, 17]. We considered the following 2 ART initiation strategies: current practice with ART initiation at CD4 counts ⩽350 cells/μL; and ART at diagnosis, regardless of CD4 count. Successful ART leads to HIV RNA suppression (HIV RNA levels, ⩽500 copies/mL) and a concomitant CD4 increase at rates reported in clinical studies [18–24]. With treatment failure and HIV RNA rebound, a subsequent ART regimen is initiated. The model specifies 4 highly efficacious ART regimens followed by 2 late salvage regimens with poorer suppressive efficacies [18–24].

Calculating community viral load and transmissible HIV RNA level. The model records each patient's total HIV uninfected life-months as well as time spent in each HIV RNA level stratum when infected (HIV RNA level strata were >100,000 copies/mL, 30,001–100,000 copies/mL, 10,001–30,000 copies/mL, 3,001–10,000 copies/mL, 501–3,000 copies/mL, and 0–500 copies/mL). These values are aggregated over large numbers of individual patient simulations to project expected time

Table 1. Model Input Parameters to Examine a Human Immunodeficiency Virus (HIV) Test and Treat Strategy in Washington DC

Variable	Base case value	Range examined	Reference
Baseline cohort characteristics			
Undiagnosed HIV prevalence[a]			
Total	0.6	0.2–3.0	[1, 33]
Asymptomatic, chronic HIV infection	0.49	…	[11, 27]
Symptomatic, chronic HIV infection	0.1	…	[11, 27]
Acute, primary HIV infection	0.008	…	[11, 27]
Annual HIV incidence	0.13	0.04–0.13	Estimated [32]
Age, mean years ± SD	41 ± 10.3	…	[1]
Male sex	46.6	…	[1]
Race/ethnicity			[1]
White	36.0	…	
Black	55.6	…	
Hispanic	8.4	…	
Distribution of initial CD4 count			
Acute, primary HIV infection,[b] mean cells/μL ± SD	534 ± 164	…	[31]
Chronic HIV infection,[c] mean cells/μL ± SD	262 ± 70	183–332	[1]
Baseline cohort characteristics			
HIV RNA distribution in chronic HIV infection			[35]
>100,000 copies/mL	0.0	…	
30,001–100,000 copies/mL	25.7	…	
10,001–30,000 copies/mL	25.0	…	
3001–10,000 copies/mL	25.2	…	
501–3000 copies/mL	16.3	…	
<500 copies/mL	7.7	…	
HIV testing protocols			
Average background HIV test frequency	Every 5 years	…	[11]
Sensitivity[d]	99.6	…	[12]
Specificity[d]	97.5	…	[12]
Test offer probability	31	30–100	[29]
Test acceptance probability	60	30–100	[29]
Probability of HIV-infected return for test results and linkage to care	50	50–100	[29]

NOTE. Data are percentage of cases, unless otherwise indicated. SD, standard deviation

[a] Undiagnosed HIV infection is calculated from the reported HIV prevalence in Washington DC (3%) and multiplied by the Centers for Disease Control and Prevention reported estimate of undiagnosed cases to diagnosed cases (21%) [1, 28, 33, 40]. Relative frequencies of acute, asymptomatic, and symptomatic cases are calculated on a 12-year natural history timeline, assuming 2 months are spent with acute infection, 2 years with symptomatic disease (AIDS), and the remaining time with asymptomatic, chronic disease [11, 27].

[b] Starting CD4 cell count for incident cases.

[c] Starting mean CD4 cell count for prevalent cases.

[d] Sensitivity and specificity refer to the characteristics of a single rapid test, not the confirmatory process; test sensitivity is assumed to be 0 during the acute infection window period (~2 months).

spent with transmissible HIV RNA levels (ie, >500 copies/mL or >3000 copies/mL) over a defined time horizon (eg, 5 years) [25]. This distribution of total time in each HIV RNA stratum is defined as the "community viral load" [26].

Input parameters. Population characteristics of the HIV-infected cohort are derived from the District of Columbia, HIV/AIDS Epidemiology Update 2008 [1] (Table 1). We use the reported 3% diagnosed HIV prevalence and apply recent Centers for Disease Control and Prevention estimates, which indicate that 21% of all HIV-infected cases in the United States are undiagnosed, to obtain an undiagnosed HIV prevalence of 0.6% [33]. We rely on traditional models of infectious disease dynamics to estimate an HIV annual incidence of 0.13% [32]. The Washington DC population mean age is 41 years, 46.6% are male, and 55.6% are black; at simulation initiation, the prevalent HIV-infected undiagnosed population has a mean CD4 count of 262 cells/μL. Background mortality rates reflect the demographic characteristics of the Washington DC population [1, 14, 15].

We assume that screening programs use rapid HIV testing (sensitivity, 99.6%; specificity, 97.5%), and reactive tests are followed by Western blot confirmation [12, 34]. In the base case,

Table 2. Base Case Results for Screening and Treatment Strategies in Washington DC

Performance index,[a] strategy	ART initiation, CD4 count	CD4 at detection of prevalent cases, cells/μL	CD4 at detection of incident cases, cells/μL	HIV-infected life expectancy, years)	Total HIV-infected follow-up time at 5 years,[b] years	Follow-up time with HIV RNA level >500 copies/mL (5 years), years (%)	Reduction in time with transmissible viral load (5 years),[c] %
Performance index 9.4%, test frequency							
No regular screen	No ART	162	352	15.8	8850	8150 (92.1)	+25.6
No regular screen[d]	⩽350 cells/μL	162	352	23.9	10,100	6500 (64.3)	…
Once	⩽350 cells/μL	171	352	24.0	10,140	6370 (62.8)	−1.9
Every 3 years	⩽350 cells/μL	173	366	24.2	10,140	6320 (62.4)	−2.5
Annually	⩽350 cells/μL	180	388	24.7	10,180	6170 (60.6)	−4.9
Once	At diagnosis	171	352	24.3	10,150	5860 (57.8)	−9.6
Every 3 years	At diagnosis	173	365	24.6	10,160	5780 (56.8)	−11.0
Annually[d]	At diagnosis	180	388	25.0	10,210	5,530 (54.2)	−14.7
Annually	Optimized ART at diagnosis[e]	180	388	25.6	10,280	4720 (46.0)	−27.3
Performance index 38%, test frequency							
No regular screen	No ART	162	352	15.8	8870	8160 (92.1)	+25.8
No regular screen[d]	⩽350 cells/μL	162	352	23.9	10,090	6490 (64.3)	…
Once	⩽350 cells/μL	201	352	24.3	10,270	5960 (58.1)	−8.1
Every 3 years	⩽350 cells/μL	204	401	25.1	10,290	5820 (56.6)	−10.3
Annually	⩽350 cells/μL	220	449	25.8	10,400	5450 (52.4)	−16.0
Once	At diagnosis	201	352	24.6	10,300	5420 (52.7)	−16.4
Every 3 years	At diagnosis	204	401	25.4	10,310	5110 (49.6)	−21.2
Annually[d]	At diagnosis	220	449	26.1	10,410	4440 (42.6)	−31.7
Annually	Optimized ART at diagnosis[e]	220	449	29.2	10,490	3320 (31.7)	−48.8
Performance index 73%, test frequency							
No regular screen	No ART	162	352	15.8	8850	8150 (92.0)	+25.5
No regular screen[d]	⩽350 cells/μL	162	352	23.9	10,100	6500 (64.4)	…
Once	⩽350 cells/μL	234	352	24.6	10,450	5490 (52.6)	−15.4
Every 3 years	⩽350 cells/μL	237	434	25.8	10,470	5280 (50.4)	−18.7
Annually	⩽350 cells/μL	247	483	26.2	10,540	4960 (47.1)	−23.5
Once	At diagnosis	234	352	24.9	10,460	4920 (47.0)	−24.2
Every 3 years	At diagnosis	237	435	26.1	10,480	4380 (41.8)	−32.6
Annually[d]	At diagnosis	247	483	26.6	10,560	3640 (34.5)	−43.9
Annually	Optimized ART at diagnosis[e]	247	483	27.2	10,660	2270 (21.3)	−65.1

NOTE. ART, antiretroviral therapy; HIV, human immunodeficiency virus.

[a] Performance index is the per encounter joint probability of test offer, test acceptance, and linkage to care. The performance index of 9.4% is derived from a test offer rate of 31%, test acceptance of 60%, and linkage to care of 50% [29]. The performance index of 38% is derived from test offer rate of 80%, test acceptance of 60%, and linkage to care of 80%. The performance index of 73% is derived from a test offer rate of 90%, test acceptance of 90%, and linkage to care of 90%.

[b] Follow-up time includes the entire HIV-infected population, both diagnosed and undiagnosed.

[c] The percent reduction in time spent with HIV RNA level >500 copies/mL is computed in comparison to current practice (ie, the no regular screening and ART initiation at CD4 cell counts ⩽350 cells/μL strategy), as reported in the second row of the table.

[d] We assume that the no regular screening and ART initiation at CD4 cell counts ⩽350 cells/μL strategy represents current practice. The test and treat strategy is represented by annual screening and ART administration at diagnosis.

[e] ART efficacy under optimized ART increases, for all 6 ART regimens, virologic suppression by 15% above the base case.

we use reported data from a successful Washington DC emergency department routine HIV screening experience, where per encounter probabilities included a test offer rate of 31%, 60% acceptance, and 50% linkage to care, resulting in a program-based performance index of 9.4% [29]. In a sensitivity analysis, we also examine a hypothetical optimistic scenario of intensified efforts to scale up screening participation with an 80% offer rate, 60% acceptance, and 80% linkage to care for those identified, resulting in a performance index of 38%. For comparative purposes, we additionally consider an idealized program with a 90% offer rate, 90% acceptance, and 90% linkage to care (performance index, 73%).

Data on the natural history of disease, including CD4 count decrease by HIV RNA stratum, and CD4-defined risks of opportunistic infections have been described elsewhere [8, 9, 30, 35]. ART-eligible patients initiate a treatment regimen with efficacies representative of those reported in the literature [18–24]. Four sequential regimens, ranging in rates of virologic suppression at 24 weeks of 60%–86%, are available, each resulting in a 100–190 CD4 cells/μL immunologic benefit at 48 weeks [18–20, 23, 24]. On exhaustion of these 4 highly effective regimens, 2 late salvage regimens are also available [21, 22]. Because a test and treat strategy may focus attention on adherence to improve ART efficacy, we also examined an optimized ART strategy by increasing virologic suppression rates of each regimen by 15% [36].

RESULTS

Clinical impact of test and treat on HIV-infected individuals. Among prevalent HIV-infected cases, the CD4 counts at detection range from 162 cells/μL with current practice to 180 cells/μL with annual screening (Table 2). Among those with incident infections, more frequent screening increases mean CD4 count at detection from 352 cells/μL (current practice) to 388 cells/μL (annual screening). In an HIV-infected population with a mean age of 41 years, per person life expectancy ranges increase from 23.9 years under current practice (no additional screen and ART at CD4 counts $\leqslant$350 cells/μL) to 25.0 years with the test and treat strategy (annual testing and ART at diagnosis). With an ART starting criterion of CD4 count $\leqslant$350 cells/μL, increasing screening frequency from once to every 3 years to annually improves life expectancy in those HIV-infected individuals by 0.08, 0.3, and 0.8 years, respectively, compared with no regular screening. With identical screening frequencies, per person life expectancy increases are 0.3–0.4 years for ART at diagnosis versus ART at CD4 counts $\leqslant$350 cells/μL.

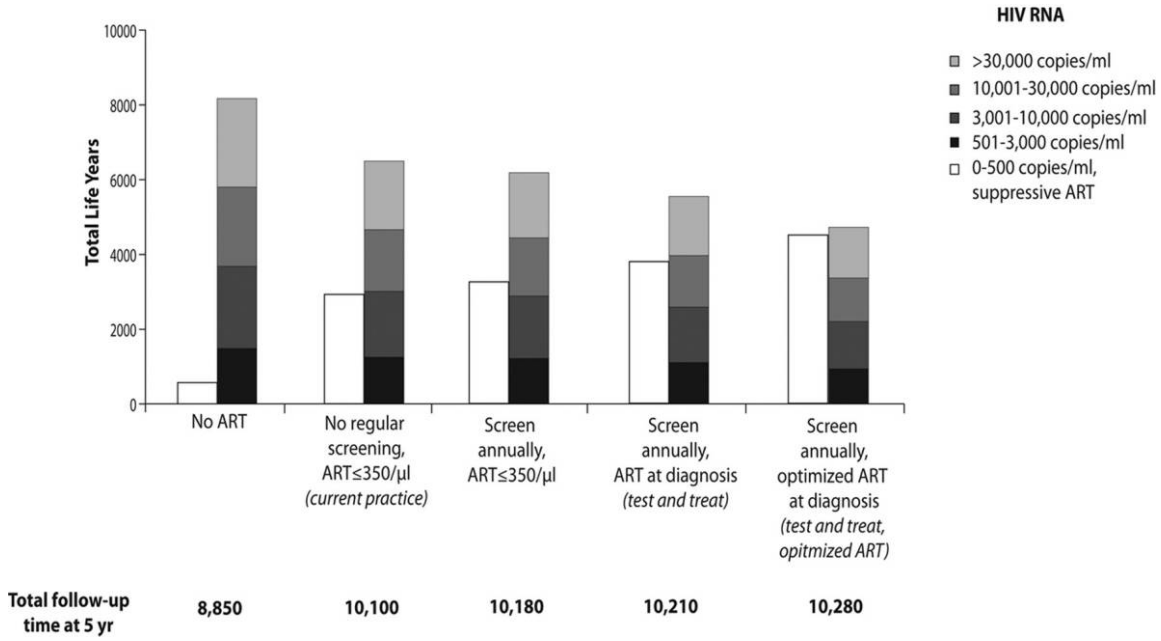

Figure 1. Over a 5-year time horizon in the base case, cumulative person-years spent at each viral load stratum (*vertical axis*) under the following 5 representative strategies (*horizontal axis*): no regular screening, no antiretroviral therapy (ART) strategy; current practice; annual testing, ART administration at CD4 cell count $\leqslant$350 cells/μL; test and treat; and test and treat with optimized ART. For each strategy, all person-years spent with a human immunodeficiency virus (HIV) RNA level >500 copies/mL are added to create the *colored bar*. Within the *colored bar,* different shades represent total time spent within alternative HIV RNA strata. Total time spent receiving fully suppressive ART is indicated by the *white bar.* The sum of the *colored* and *white bars* yields the total HIV-infected life-years, reported at the bottom of each set.

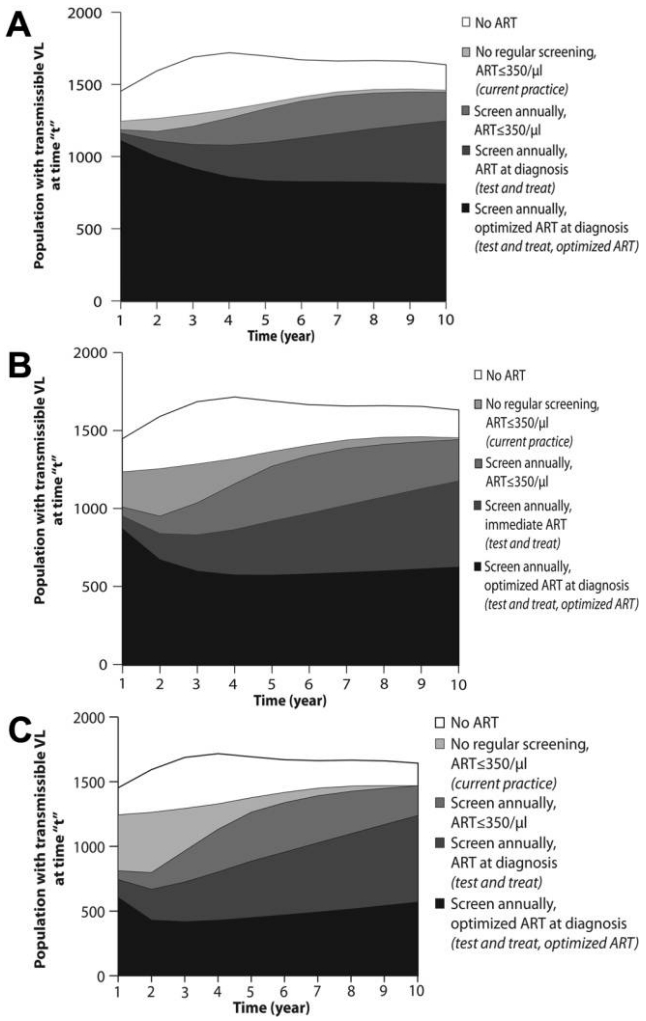

Figure 2. Rate of accrual of life years in the human immunodeficiency virus (HIV)-infected population (*vertical axis*) with a transmissible HIV RNA level (>500 copies/mL) over a 10-year time horizon (*horizontal axis*). The height of the curve denotes the number of people with transmissible viral load living in the population at any moment in time. The area under each curve represents the community viral load burden. The following 5 representative strategies are shown: no regular screening, no antiretroviral therapy (ART) strategy (*clear*); current practice (*light gray*); annual testing, ART administration at CD4 cell count ⩽350 cells/μL (*medium gray*); test and treat (*dark gray*); and test and treat with optimized ART (*black*). *Panels A–C* represent each of these strategies under the following alternative assumptions about the participation index: base case, 9.4% (*A*); optimistic, 38% (*B*); and idealized, 73% (*C*).

Community viral load and transmissible HIV RNA. For the estimated 6410 prevalent and incident cases expected over the next 5 years in Washington DC, the no regular screening and no ART strategy results in 8150 life-years in the community with a transmissible viral load (HIV RNA level, >500 copies/mL; Table 2 and Figure 1). Current practice and test and treat lead to 6500 and 5530 life-years of transmissible viral load. Compared with current practice, test and treat decreases the proportion of time with transmissible viral load over a 5-year time horizon from 64.3% to 54.2% (Table 2) and decreases the proportion of time with viral load over >3000 copies/mL from 51.9% to 43.5% (data not shown). Over a 5-year horizon, the test and treat strategy, compared with current practice, offers a 14.7% reduction in overall population time spent with transmissible HIV RNA levels (Table 2).

Test and treat with optimized ART. Test and treat with optimized ART increases projected life expectancy to 25.6 years, which is 0.6 years more than test and treat alone. Compared with current practice, test and treat with optimized ART leads to a 27.3% reduction in population time spent with transmissible infection over 5 years (Table 2 and Figure 1).

Performance index. Projected life expectancy and community viral load in the current practice strategy are unchanged as performance index varies. With 38% program performance, benefits to test and treat increase compared with the base case; projected life expectancy is 26.1 years, and 4440 life-years (42.6% of time) are spent with transmissible HIV RNA levels (Table 2). With 73% program performance, clinical and population benefits from test and treat improve; projected life expectancy is 26.6 years, and compared with current practice, there is a 43.9% reduction in overall population time spent with transmissible infection (Table 2). Test and treat with optimized ART in this idealized scenario reduces time spent with transmissible HIV RNA by 65.1%, compared with current practice.

Alternative testing strategies, performance scenarios, and ART efficacies change the rate at which total HIV transmissible life-years are lived (Figure 2). The area under each curve represents the cumulative time spent with transmissible viral load (>500 copies/mL), which is a proxy for overall transmission of infection in the population. More frequent testing with ART at diagnosis decreases life-years spent with HIV RNA levels >500 copies/mL; improvements in program performance and optimized ART also result in fewer total life-years with transmissible HIV RNA levels (Figure 2). With 73% program performance (Figure 2*C*), the area under the test and treat with optimized ART curve represents a substantial reduction in the number of life-years lived within the cohort with potential for active viral transmission, a 65.4% reduction from current practice over 10 years.

Other sensitivity analyses. In sensitivity analyses examining a less severe HIV epidemic (undiagnosed HIV prevalence

The figure is available in its entirety in the online edition of *Clinical Infectious Diseases*.

Figure 3. Rate of accrual of life years in the human immunodeficiency virus (HIV)-infected population (0.21% prevalence and 0.04% incidence) with a transmissible HIV RNA level (>500 copies/mL) over a 10-year time horizon.

The figure is available in its entirety in the online edition of *Clinical Infectious Diseases*.

Figure 4. Rate of accrual of life years in the human immunodeficiency virus (HIV)-infected population (mean CD4 count at time of diagnosis, 332 cells/μL) with a transmissible HIV RNA level (>500 copies/mL) over a 10-year time horizon.

0.21% and 0.04% annual incidence), similar to those in Miami and Philadelphia, the test and treat strategy offers a 14.7% reduction in time spent with transmissible HIV RNA levels over 5 years (Figure 3*A*–3*C*) [37–39]. Analogous results are also achieved when the estimate of undiagnosed HIV infection in Washington DC is doubled (undiagnosed prevalence, 1.2%; annual incidence, 0.16%) [28, 33, 40]. In sensitivity analyses varying the mean CD4 count of the undiagnosed HIV-infected population from 183 cells/μL to 332 cells/μL (Washington DC, 2002 and 2007), test and treat results in a 14.0% and 17.2% reduction in population time spent with transmissible infection, compared with current practice (Figure 4*A*–4*C*) [1]. Near elimination of the HIV-infected population with transmissible viral load is achieved with monthly HIV screening, 100% program participation and linkage to care, and perfectly suppressive and durable ART efficacy (Figure 5).

DISCUSSION

Using data from Washington DC, one of the epicenters of the United States HIV epidemic, we demonstrate that an intensive test and treat strategy can yield substantial benefits to individuals, improving HIV-infected life expectancy by up to 1.1 years, compared with the current standard of care. Although most of the increase in life expectancy is achieved with improvements in testing frequency and coverage, an additional 4 months are likely added by ART initiation immediately at diagnosis. And, compared with test and treat alone, further improvements in ART regimen efficacy may contribute an additional 7 months of life expectancy.

Beyond the clinical benefits to infected individuals, we find that test and treat may have quantifiable population benefits. A test and treat strategy may reduce overall life-years spent with transmissible HIV infection over the next 5 years by 15%. Any prevention intervention with the potential to decrease transmission by 15% and to produce substantial increases in individual HIV-infected life expectancy warrants further investigation. However, suggestions that a test and treat strategy might be sufficient to eradicate the HIV epidemic create public expectations that cannot be realized [5]. The transmission effect is the indirect result of viral suppression benefits that accrue most directly to the infected person. Therefore, prevention benefits result largely from earlier treatment initiation; providing ART at diagnosis increases the number of transmissions averted over 5 years, compared with frequent testing and guideline-concordant ART alone. Overall life-years with transmissible HIV RNA levels, and likely overall transmissions, may be reduced by almost one-quarter if test and treat could be combined with major efforts to improve ART adherence and rates of virologic suppression.

This analysis highlights the interplay of the components of test and treat and their impact on HIV-infected individuals and the population. It also underscores that the success of any test and treat strategy hinges on the process of successfully making HIV test offers, completing tests, linking infected patients to care, and maximizing the effects of ART [41]. Numerous published programs that exemplify extraordinary efforts, testing large numbers of patients, and identifying many new cases of HIV, even beyond Washington DC, have overall process success rates (from offer to acceptance to linkage to care) of only ~10% [29, 42]. These very low levels of participation will provide individual benefits to those identified but will be inadequate to have a meaningful impact on the population. Although programs with extensive breadth (80% of the population offered) and depth (annual or more frequent testing), as illustrated by the optimistic scenario, may be challenging to achieve, such efforts could have a larger impact on population outcomes. Furthermore, improved ART efficacy, likely attainable with currently available potent regimens and beyond that even reported in trials, is critical to effectively decrease transmission.

Like all model-based studies, this analysis is limited by the input data available. We derived input parameters from published sources, incorporating data from the Washington DC 2008 report whenever possible [1]. In the optimized and idealized scenarios, we intended to portray a very high level of program and ART performance; the results demonstrate, even under such optimism, the anticipated magnitude of population benefit achievable from a test and treat approach. To assess such population benefits, we report percent reduction in transmissible HIV RNA levels. The higher the initial HIV prevalence/incidence, the more the percent reduction translates into increased infections averted in absolute terms. The analysis of community viral load is restricted to prevalent and incident cases; to the extent that second- and third-generation HIV infections substantially contribute to community viral burden

The figure is available in its entirety in the online edition of *Clinical Infectious Diseases*.

Figure 5. Rate of accrual of life years in the human immunodeficiency virus (HIV)-infected population with a transmissible HIV RNA level (>500 copies/mL) over a 10-year time horizon under the following hypothetical scenario: monthly HIV test offered, test acceptance 100%, rate of linkage to HIV care among those identified 100%, monthly clinic visits, 100% viral suppression while receiving antiretroviral therapy and perfect adherence.

over a 5-year horizon, the benefits of test and treat may have been underestimated. Data continue to emerge on the benefits and risks of ART at CD4 counts >350 cells/μL. Although this model excludes the benefits (and/or risks) of early ART on non–AIDS-related morbidities, such as cardiovascular and renal disease, the input parameters reflect the toxicity profiles of current treatment and, therefore, likely underestimate the benefits from earlier ART [43]. Natural history data used for this analysis may underestimate the proportion of hepatitis C–coinfected patients in the urban Washington DC population and, therefore, may overestimate the life expectancy of HIV-infected persons, in general [44]. Finally, we have excluded costs from this analysis. Cost-effectiveness results, to be methodologically sound, must be reported on a population-wide scale. As such, these results are more speculative with regard to future transmissions and detract from the prevention message (rather than the economic one) that lies at the heart of current debate over test and treat.

We find that dedicated efforts to address the HIV epidemic in Washington DC and in other heavily affected United States cities will substantially affect the survival of HIV-infected patients identified, averting many missed diagnoses and new AIDS cases. Moreover, earlier detection, linkage, and treatment of infected persons is likely to have a dramatic impact on secondary HIV transmission, reducing the number of new infections by as much as 15%. However, the success of such interventions hinges on careful attention to process and implementation by making frequent offers, securing high levels of consent, linking all detected cases to care, and initiating ART immediately. Even if future implementations greatly exceed the performance observed in recent, highly organized, well-financed programs, it is very unlikely that a test and treat strategy will stop the epidemic in Washington DC.

THE CEPAC UNITED STATES INVESTIGATORS

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Acknowledgments

We thank Bernard M. Branson, MD, for his critical review of the manuscript.

Financial support. National Institute of Allergy and Infectious Diseases (R37 AI042006, K24 AI062476, and P30 AI060354), the National Institute of Mental Health (R01 MH065869 and R01 MH073445), and the Doris Duke Charitable Foundation (Clinical Scientist Development Award).

Potential conflicts of interest. All authors: no conflicts.

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