Supplemental Digital Content 1:

Cost-Effectiveness of the National HIV/AIDS Strategy (NHAS) Goal of Increasing Linkage to Care for HIV-Infected Persons

The Progression and Transmission of HIV/AIDS (PATH) is an individual Monte Carlo simulation health state transition model that tracks individuals (index patients) with human immunodeficiency virus (HIV) infection and their infected partners from the time of infection until death. [1] We used PATH to estimate the costs and life-measures for analyzing the National HIV/AIDS Strategy on increasing linkage to care of HIV infected individuals. Details of the PATH model have been described by Prabhu et. al., PLoS One, 2011 [1] and here we only give a brief description of the model. Since its original publication, the PATH model has been updated to include recent changes in HIV treatment, costs, and the transmission analysis. Therefore, we also discuss these updates to the model.

Brief description of PATH

The model initially simulates a first generation of HIV-infected individuals (index persons) from the time of infection through the following disease stages: acute infection, asymptomatic infection, symptomatic infection/AIDS (acquired immune deficiency syndrome), and death. We assumed individuals entered the model, i.e. were infected, at age 35 years and that HIV was transmitted through injection drug use (IDU) for 12.9% of the individuals and through sexual transmission for all others.[2] We did not model behavioral differences in risk

groups (men who have sex with men (MSM), high-risk heterosexual or IDU), and we did not stratify by gender or race. We present a schematic flowchart of disease progression for HIV-infected individuals in the PATH model in Figure S1. We ran the model with both point estimates and distributions of values around the point estimates for key variables.

The acute phase of infection is characterized by high HIV viral load in the initial weeks of infection. We modeled the acute phase to last for one calendar-year quarter, which is the time unit in our model, and to have a viral load of 5.3 $\log_{10}$ copies/ml.[3,4] We assumed the CD4 count at infection was 900 cells/μL.[5]

During the asymptomatic infection phase we assumed there were no AIDS-related symptoms, but the stage was marked by a steady viral load (the HIV viral load set point of 4.5 $\log_{10}$ copies/ml)[6] and declining CD4 count in the absence of treatment. We used estimates of the rate of CD4 count decline for different ranges of HIV viral load reported by Rodriguez et al. (2006).[7]

Symptomatic HIV infection or AIDS was characterized by the occurrence of an opportunistic infection (OI), determined by different probabilities, or a drop in CD4 count to below 200 cells/μL. We assumed the probability of having an OI increased with a decline in CD4 count, and we modeled six categories of such illnesses.[8,9]

We assumed HIV-infected individuals in the model could die from causes either related to HIV/AIDS or other factors. For individuals who were not yet on treatment, we used different quarterly probabilities of death for persons infected through sexual transmission[10] or through IDU transmission.[11] These

probabilities increased as an individual's CD4 count declined. The maximum number of years of life remaining for an individual infected with HIV in the PATH model was limited by life expectancy at the age of infection based on U.S. life tables.

We assumed individuals could be tested at any time after the acute phase of infection with the exact time determined by the setting under review. For example, in the linkage to care setting in the main paper, CD4 count was used to determine the time of diagnosis. We assumed that HIV testing was conducted using a rapid test followed by a confirmatory Western blot. Linkage to care and treatment initiation occurred at some time after testing, the criteria for which were described in the main paper.

We assumed the natural progression of HIV described above was altered upon initiation of treatment with antiretroviral therapy (ART), which is associated with suppressed viral load, higher CD4 cell counts, improved life expectancy, and improved quality of life.[12,13] We modeled a maximum of four treatment regimens followed by one non-suppressive or salvage regimen.[14] Once suppressed, HIV viral load was assumed to remain constant at 1.3 $\log_{10}$ copies/ml as long as the regimen was effective.[15] When a particular treatment regimen ceased to be effective, we assumed that the HIV viral load rebounded to 3.7 $\log_{10}$ copies/ml.[16] Regimen durations were based on the literature.[16-18] We assumed that CD4 counts increased when ART regimens were effective. The maximum CD4 count that could be achieved during sustained HIV viral load suppression depended upon the CD4 count at initiation of the first ART

regimen.[19] Probabilities of death for patients on ART varied by CD4 count at initiation of ART, age, mode of transmission (sexual or IDU), and whether patients had an OI. [12,13]

To estimate quality-adjusted life years (QALYs), we assigned utility weights for each quarter that were based on Tengs and Lin (2002)[20] and that varied by CD4 count and presence of an OI during that quarter. Once we aggregated QALYs over the life of an individual, we subtracted the sum from the QALYs associated with an HIV-uninfected person and estimated QALYs lost due to HIV infection. Measuring QALYs lost from infection resulted in consistent quality of life estimates when transmissions to partners were included in the model.

The PATH model also included disease progression in a second generation of infected individuals arising from transmissions from the index persons. The number of transmissions in each quarter was determined by probabilities defined by the phase of infection and treatment status of the index person. During the course of the simulation, the model tracked and collected cost and life expectancy measures for index patients and their infected partners. A summary of the input parameters including updated values is provided in Table S1.

Updates to the PATH Model

The PATH model has been updated with recent estimates of HIV-related measures as described below.

Treatment regimen:

Depending on the linkage to care assumption, an individual initiated treatment at a CD4 count of either 350, 200, or 40 cells/μL starting with the first line of regimen. In accordance with expert opinion and recent clinical trials,[21-25] we assumed the probability of initial viral load suppression depended on the CD4 count at start of treatment.

Cost components:

Costs were incurred at different stages of infection and components included costs for non-HIV medication, HIV diagnosis, treatment, follow-up CD4 cell count and RNA tests, health care utilization, and opportunistic infection treatment. Costs, originally derived from Schackman et al. (2006),[26] were updated using estimates from Gebo et al., (2010)[27] and adjusted to $US 2009. We retained the ART regimens and treatment costs developed by Schackman et al. (2010).[26]

Updated estimates of HIV transmission to sexual and needle sharing partners:

An infected index person can transmit HIV infection through sexual contact or injection drug use (IDU) at a rate dependent on the infection phase. Pinkerton et. al. (2007) and Prabhu et. al. (2009) [28,29] estimated sexual transmission rates as a ratio of incidence to prevalence in each of three transmittable populations: acute unaware, non-acute unaware, and non-acute aware. In our model we subdivided the aware population further to differentiate between persons on and not on ART and the success of viral load suppression from treatment. Modifying the model in Pinkerton et. al. (2007)[28],

I = total number of sexual transmissions on any given day,

γ_k = daily transmission rate in group k,

N_k = number of people living with HIV/AIDS (PLWH) in group k, and

I_k = proportion of new infections transmitted by group k, where,

$k = \{1,2,3,4,5\}$ constitute the HIV transmittable populations grouped according to their stage of infection, i.e., 1= acute unaware; 2= non-acute unaware; 3= non-acute aware not on treatment; 4= non-acute aware on treatment with viral load not suppressed; 5 = non-acute aware on treatment with suppressed viral load. Then,

$$\mathrm{I} = \sum_{k=1}^{5} \gamma_k \mathrm{N}_k$$

$$I_1 = \frac{\gamma_1 N_1}{I} = \frac{\frac{\gamma_1}{\gamma_2} N_1}{\frac{\sum_{k=1}^{5} \gamma_k N_k}{\gamma_2}} = \frac{\mu_{12}\mu_{23}N_1}{\mu_{12}\mu_{23}N_1 + \mu_{23}N_2 + N_3 + \frac{1}{\mu_{34}}N_4 + \frac{1}{\mu_{35}}N_5}$$, where,

$\mu_{12} = \frac{\gamma_1}{\gamma_2}$; $\mu_{23} = \frac{\gamma_2}{\gamma_3}$; $\mu_{34} = \frac{\gamma_3}{\gamma_4}$; $\mu_{35} = \frac{\gamma_3}{\gamma_5}$; and $\mu_{12}\mu_{23} = \frac{\gamma_1}{\gamma_3}$.

In general, μ_{ij} is the ratio of daily transmission rate in group i (i.e., γ_i) to the daily transmission rate in group j (i.e., γ_j). For example, $\mu_{12} = \frac{\gamma_1}{\gamma_2}$ = the daily transmission rate in acute unaware group divided by the daily transmission rate in non-acute unaware group.

Denoting the denominator as $D = \mu_{12}\mu_{23}N_1 + \mu_{23}N_2 + N_3 + \frac{1}{\mu_{34}}N_4 + \frac{1}{\mu_{35}}N_5$, we can write: $I_2 = \frac{\mu_{23}N_2}{D}$; $I_3 = \frac{N_3}{D}$; $I_4 = \frac{\frac{1}{\mu_{34}}N_4}{D}$; $I_5 = \frac{\frac{1}{\mu_{35}}N_5}{D}$. Let n_k be the total number of unprotected sex acts across all partners, and α_k be the average per-act transmission probability for individuals in group k. Pinkerton et.al. (2007) [28]

estimated $\mu_{12} = 8.1$ and, under the assumption that the risk of transmission from condom-protected acts is zero, estimated $\gamma_2 \sim n_2\alpha_2$ since $\alpha_2$is a relatively small value. Similarly, we use $\gamma_k \sim n_k\alpha_k \text{for } k = \{3,4,5\}$, $\mu_{ij} = \frac{n_i\alpha_i}{n_j\alpha_j} \text{ for } ij = \{23, 34, 35\}$, and estimate values of μ_{ij} by assuming a 57% [range 52-59%] reduction in the prevalence of UAV (unprotected anal or vaginal intercourse) with at-risk partners in the aware group compared to unaware group,[30,31] no change in behavior across groups 3, 4, and 5 (i.e., $n_3 = n_4 = n_5$), no change in virology across groups 2, 3, and 4 ($\text{i.e.}, \alpha_2 = \alpha_3 = \alpha_4$), and a 90% [range 80-99%] reduction in infectivity due to viral load suppression from treatment.[32] Therefore, we can write $\mu_{23} = \frac{1}{1-0.57} = 2.33$, $\mu_{34} = 1, \text{and } \mu_{35} = \frac{1}{1-0.9} = 10$. Note that, though we assumed equal transmission rate between groups 3 and 4 (i.e., $\mu_{34} = 1$), we separated the groups to allow for future consideration of partial reduction in transmission arising from treatment even when viral load is not completely suppressed.

As in Prabhu et al. (2009),[29] values of N_k were estimated using the 2006 U.S. estimates of incidence and prevalence, i.e., the total number of PLWH was 1,106,300,[33] incidence of HIV infections was 56,300, of which 47,207 incidences were from sexual transmission,[2] the number of undiagnosed HIV cases was 232,700,[33] and the number of diagnosed cases of HIV was 873,600. N_1 was estimated by assuming that the acute phase of infection lasts for 49 days,[28] and during this phase, the person was assumed to remain serostatus-unaware. N_3, N_4, and N_5 were estimated by assuming that 65% of the aware population were linked to care of which 80% were on ART, and 80% of people

on ART had a suppressed viral load [expert opinion].[34] Values for I_k were estimated by applying the estimates of N_k and μ_{ij} in the formulation for I_k presented earlier. The number of new infections from group k per year was estimated as I_k X 47,207 and the annual transmission rates as incidence divided by prevalence, i.e., $\frac{I_k X\ 47{,}207}{N_k}$.

IDUs were assumed to contribute 12.9% of new HIV infections and the transmission rate from the non-acute unaware population was assumed to be 0.165 per year.[35,36] IDU transmission rates for all other groups were estimated by using the proportion of sexual transmission rates across the groups.

Figure S1. Schematic flowchart of PATH model overview

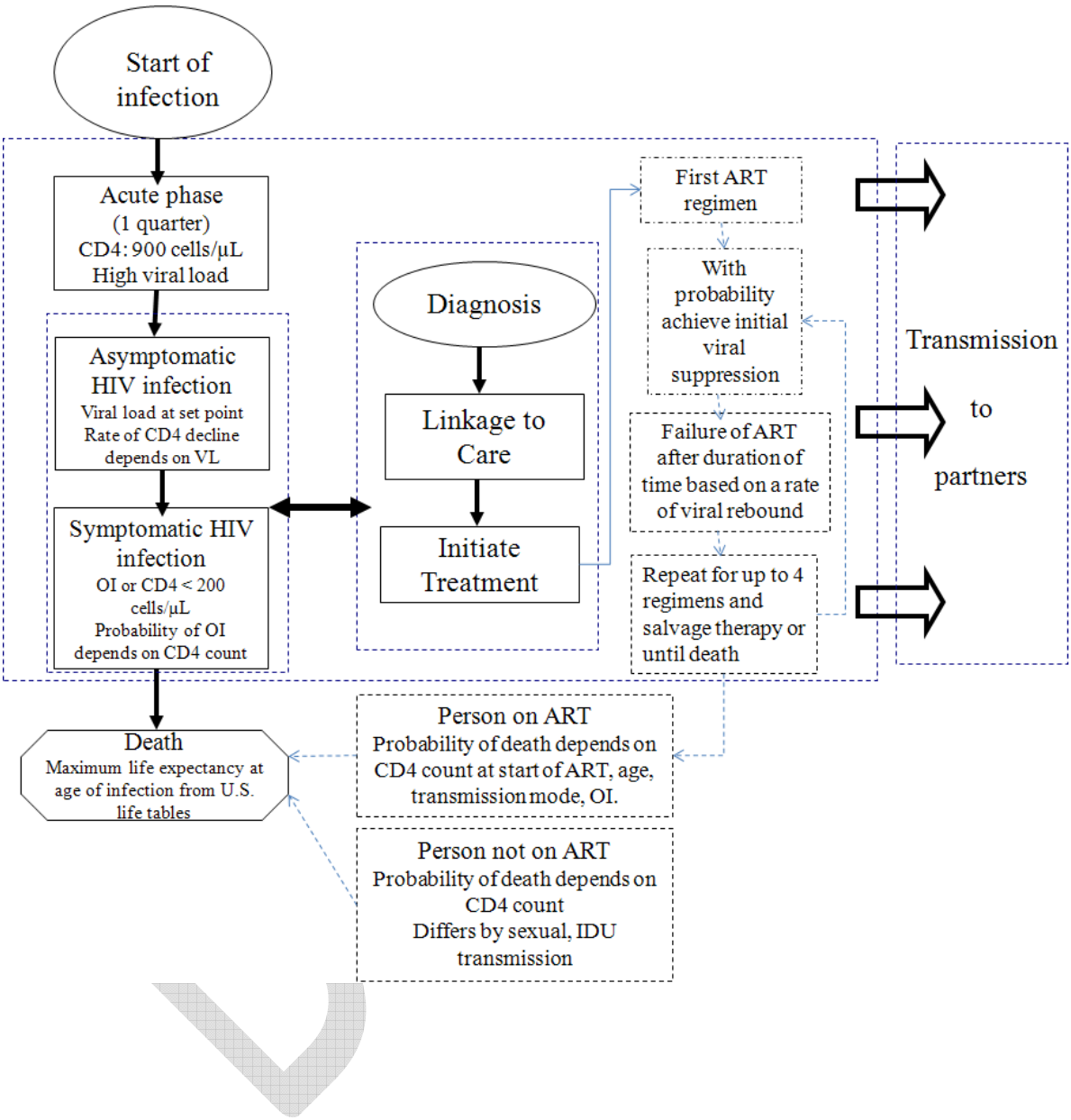

Table S1: Complete List of Input Parameters

Variable	Mean Value	Range	Source
Age at infection (years)	35	30 - 40	[a]
Discount rate for costs and quality-adjusted life years (QALYs)	3%		37
Natural Disease Progression			
CD4 cell count when infected (cells/μL)	870	750 - 900	5
Acute phase HIV viral load (log_{10} copies/ml)	5.3	4.4 – 6.2	3,4
HIV viral load set point (log_{10} copies/ml)	4.5	4.0 – 5.0	6,17
Natural rate of CD4 cell count decline (cells/μL/quarter) as a function of HIV viral load stratum (log_{10} copies/ml)			7
≤ 2.7	5.1	2.4 – 7.8	
2.7 – 3.3	9.9	7.2 – 12.3	
3.3 – 4.0	12	9.9 – 13.8	
4.0 – 4.6	14.1	11.7 – 16.2	
≥ 4.6	19.5	17.1 – 21.9	
Quarterly Probability of Developing an Opportunistic Infection (OI) (%)			12,13
Pneumocystis pneumonia (PCP)	0.1 – 10.7[b]		
Mycobacterium avium complex	0.0 – 3.6		
Toxoplasmosis	0.0 – 0.8		
Cytomegalovirus infection	0.0 – 5.5		
Fungal infection	0.0 – 3.3		
Other	0.1 – 11.4		

Cumulative probability for all OIs	0.3 – 35.3		
Quarterly Probability of Death for Antiretroviral Therapy (ART)-Naïve Individuals (%)			
Sexual transmission: CD4 cell count (cells/μL)			[10]
≥ 650	0.043		
500 – 649	0.05		
350 – 499	0.08		
200 – 349	0.145		
50 – 199	0.767		
< 50	4.9		
Injection drug use (IDU) transmission: CD4 cell count (cells/μL)			[11]
≥ 350	1.069		
200 – 349	1.486		
< 200	4.068		
ART Regimens			
Minimum CD4 cell count to initiate ART (cells/μL)	350		[38]
Suppressed HIV viral load level (log_{10} copies/ml)	1.3	1.0 – 2.7	[15]
Rebound HIV viral load level (log_{10} copies/ml)	3.7	3.1 – 4.5	[16]
Maximum number of ART regimens	4		[c]
Probability of virologic suppression in ART regimens 1 – 4: CD4 cell count (cells/μL)			[21,22]
>200	0.84		
50 - 200	0.79		
< 50	0.774		

Rate of HIV viral load rebound (% experiencing rebound after one year, first regimen)	7.4		[16,17]
Percent increase in rate of HIV viral load rebound for each successive regimen compared to its previous regimen	18		[18]
HIV viral load above set-point during salvage therapy (log_{10} copies/ml)	0.8	0.0 – 1.5	[35]
HIV viral load above set-point during salvage therapy after onset of AIDS (log_{10} copies/ml)	1	0.0 – 2.0	[35]
Quarterly increase in CD4 cell count during HIV viral load suppression (cells/μL/quarter)			[19]
Quarters 1 – 2	68		
Quarters 3 – 12	40		
Quarters 12+	0		
Maximum CD4 cell count achieved based on CD4 cell count at initiation of ART (cells/μL)			[19]
< 50	410		
50 – 200	548		
201 – 350	660		
351 - 500	780		
> 500	870		
Quarterly Probability of Death After Initiation of ART (%)			[12,13]
Sexual transmission			
No AIDS symptoms			
Age 16 – 29 years	0.09 – 0.26[d]		

Age 30 – 39 years	0.12 – 0.32		
Age 40 – 49 years	0.15 – 0.43		
Age ≥ 50 years	0.29 – 0.81		
Clinical symptoms of AIDS			
Age 16 – 29 years	0.19 – 0.53		
Age 30 – 39 years	0.25 – 0.69		
Age 40 – 49 years	0.32 – 0.93		
Age ≥ 50 years	0.64 – 1.77		
Injection drug use transmission			
No AIDS symptoms			
Age 16 – 29 years	0.27 – 0.75		
Age 30 – 39 years	0.35 – 0.99		
Age 40 – 49 years	0.46 – 1.30		
Age ≥ 50 years	0.87 – 2.44		
Clinical symptoms of AIDS			
Age 16 – 29 years	0.58 – 1.63		
Age 30 – 39 years	0.75 – 2.06		
Age 40 – 49 years	0.99 – 2.77		
Age ≥ 50 years	1.84 – 5.11		
Utility Weights to Estimate Quality Adjusted Life Years (QALYs)			[20]
OI or CD4 count < 200 cells/μL	0.702		
CD4 cell count ≥ 200, < 350 cells/μL	0.818		
CD4 cell count > 350 cells/μL	0.935		
Quarterly Costs (2009 $)			
Individuals in care: cost components include inpatient, outpatient, and emergency department resource utilization, and non-HIV medication	1221- 6135 [e]		[27]

Individuals not in care: non-HIV medication costs only	542 [f]		[27]
Additional costs of opportunistic infections (each occurrence)	3,492 – 20,542[g]		[26]
Cost of ART (each quarter)			[26]
Regimen 1	4,143		
Regimen 2	4,542		
Regimen 3	6,686		
Regimen 4	13,699		
Salvage Therapy	7,159		
CD4 cell count test (one each quarter)	42		[27]
HIV viral load test (one each quarter)	101		[27]
HIV genotype test (beginning of each regimen)	425		[27]
Annual Rates of Transmission (# events per year per person)			
Sexual transmission			Derived from [28,29]
Acute	0.733		
Non-acute unaware	0.091		
Non-acute aware, not on ART	0.039		
Non-acute aware, on ART but viral load not suppressed	0.039		
Non-acute aware, on ART with suppressed viral load	0.004		
IDU transmission			Derived from [28,29,35,36]
Acute	1.337		
Non-acute unaware	0.165		

Non-acute aware, not on ART	0.071		
Non-acute aware, on ART but viral load not suppressed	0.071		
Non-acute aware, on ART with suppressed viral load	0.007		

[a]Writen communication, R. Song, Centers for Disease Control and Prevention, June, 2008.
[b]The lower and upper bounds for various types of OIs reflect probabilities for CD4 cell counts of > 500 cells/μL and 0 – 50 cells/μL respectively. Probabilities of an OI at intermediate CD4 cell counts lie within these bounds.
[c]Expert opinion (2009)
[d]The lower and upper bounds reflect the probability of death for CD4 cell counts ≥ 350 cells/μL and < 25 cells/μL, respectively. Probabilities of death at intermediate CD4 cell counts lie within these bounds.
[e]The lower and upper bounds for costs reflect costs for CD4 cell counts > 500 cells/μL and ≤ 50 cells/μL, respectively. Costs for intermediate CD4 cell counts lie within these bounds.
[f]Reflects average cost over all CD4 cell count ranges.
[g]Numbers represent costs for different opportunistic illnesses.

1. Prabhu VS, Farnham PG, Hutchinson AB, et al. Cost-Effectiveness of HIV Screening in STD Clinics, Emergency Departments, and Inpatient Units: A Model-Based Analysis. *PLoS One.* 2011;6(5):e19936.
2. Hall HI, Song R, Rhodes P, et al. Estimation of HIV incidence in the United States. *JAMA.* Aug 6 2008;300(5):520-529.
3. Schacker TW, Hughes JP, Shea T, et al. Biological and Virologic Characteristics of Primary HIV Infection. *Ann Intern Med.* 1998;128(8):613-620.
4. Schacker TW, Collier AC, Hughes JP, et al. Clinical and epidemiologic features of primary HIV infection. *Ann Intern Med.* 1996;125(4):257-264 (erratum: Ann Intern Med 1997;1126:1174).
5. Turner BJ, Hecht FM, Ismail RB. CD4+ T-lymphocyte measures in the treatment of individuals infected with human immuno deficiency virus type 1: a review for clinical practitioners. *Arch Intern Med.* 1994;154:1561-1573.
6. Herbeck JT, Gottlieb GS, Li X, et al. Lack of Evidence for Changing Virulence of HIV-1 in North America. *PLOS One.* 2008;2(1-8).
7. Rodriguez B, Sethi AK, Cheruvu VK, et al. Predictive value of plasma HIV RNA level on rate of CD4 T-cell decline in untreated HIV infection. *Jama-J Am Med Assoc.* Sep 27 2006;296(12):1498-1506.
8. Paltiel AD, Weinstein MC, Kimmel AD, et al. Expanded Screening for HIV in the United States — An Analysis of Cost-Effectiveness. *New Engl J Med.* 2005;352:586-595.

9. Weinstein MC, Goldie SJ, Losina E, et al. Use of genotypic resistance testing to guide HIV therapy: Clinical impact and cost-effectiveness. *Ann Intern Med.* Mar 20 2001;134(6):440-450.
10. The United Kingdom Collaborative HIV Cohort (CHIC) Study. Rate of AIDS diseases or death in HIV-infected antiretroviral therapy-naive individuals with high CD4 cell count. *AIDS.* 2007;21:1717-1721.
11. Wang C, Vlahov D, Galai N, et al. Mortality in HIV-Seropositive versus -Seronegative Persons in the Era of Highly Active Antiretroviral Therapy: Implications for When to Initiate Therapy. *The Journal of Infectious Diseases.* 2004;190:1046-1054.
12. Antiretroviral Therapy Cohort Collaboration. Importance of baseline prognostic factors with increasing time since initiation of highly active antiretroviral therapy: collaborative analysis of cohorts of HIV-1-infected patients. *Journal of acquired immune deficiency syndromes.* 2007;46(5):607-615.
13. Antiretroviral Therapy Cohort Collaboration. Prognosis of HIV-1-infected patients up to 5 years after initiation of HAART: collaborative analysis of prospective studies. *AIDS.* 2007;21(9):1185-1197.
14. Dybul M, Fauci AS, Bartlett JG, et al. Guidelines for using antiretroviral agents among HIV-infected adults and adolescents. *Ann Intern Med.* 2002;137((5 Pt 2)):381-433.
15. Raboud JM, Montaner JSG, Conway B, et al. Suppression of plasma viral load below 20 copies/ml is required to achieve a long-term response to therapy. *AIDS.* 1998;12(13):1619-1624.
16. The United Kingdom Collaborative HIV Cohort (CHIC) Study. Treatment switches after viral rebound in HIV-infected adults starting antiretroviral therapy: multicentre cohort study. *AIDS.* 2008;22:1943-1950.
17. Vo TTN, Ledergerber B, Keiser O, et al. Durability and Outcome of Initial Antiretroviral Treatments Received during 2000-2005 by Patients in the Swiss HIV Cohort Study. *JID.* 2008;197:1685-1694.
18. Smith CJ, Phillips AN, Dauer B, et al. Factors associated with viral rebound among highly treatment-experienced HIV-positive patients who have achieved viral suppression. *Hiv Med.* Jan 2009;10(1):19-27.
19. Gras L, Kesselring AM, Griffin JT, et al. CD4 cell counts of 800 cells/mm3 or greater after 7 years of highly active antiretroviral therapy are feasible in most patients starting with 350 cells/mm3 or greater. *J Acquir Immune Defic Syndr.* Jun 1 2007;45(2):183-192.
20. Tengs TO, Lin TH. A meta-analysis of utility estimates for HIV/AIDS. *Medical Decision Making.* Nov-Dec 2002;22(6):475-481.
21. Lennox JL, Dejesus E, Berger DS, et al. Raltegravir versus Efavirenz regimens in treatment-naive HIV-1-infected patients: 96-week efficacy, durability, subgroup, safety, and metabolic analyses. *J Acquir Immune Defic Syndr.* Sep 1 2010;55(1):39-48.
22. Lennox JL, DeJesus E, Lazzarin A, et al. Safety and efficacy of raltegravir-based versus efavirenz-based combination therapy in

treatment-naive patients with HIV-1 infection: a multicentre, double-blind randomised controlled trial. *Lancet.* Sep 5 2009;374(9692):796-806.

23. Molina JM, Andrade-Villanueva J, Echevarria J, et al. Once-daily atazanavir/ritonavir versus twice-daily lopinavir/ritonavir, each in combination with tenofovir and erntricitabine, for management of antiretroviral-naive HIV-1-infected patients: 48 week efficacy and safety results of the CASTLE study. *Lancet.* Aug 23 2008;372(9639):646-655.

24. Molina JM, Andrade-Villanueva J, Echevarria J, et al. Once-Daily Atazanavir/Ritonavir Compared With Twice-Daily Lopinavir/Ritonavir, Each in Combination With Tenofovir and Emtricitabine, for Management of Antiretroviral-Naive HIV-1-Infected Patients: 96-Week Efficacy and Safety Results of the CASTLE Study. *Jaids-J Acq Imm Def.* Mar 1 2010;53(3):323-332.

25. Mills AM, Nelson M, Jayaweera D, et al. Once-daily darunavir/ritonavir vs. lopinavir/ritonavir in treatment-naive, HIV-1-infected patients: 96-week analysis. *AIDS.* Aug 24 2009;23(13):1679-1688.

26. Schackman BR, Gebo KA, Walensky RP, et al. The lifetime cost of current human immunodeficiency virus care in the United States. *Med Care.* Nov 2006;44(11):990-997.

27. Gebo KA, Fleishman JA, Conviser R, et al. Contemporary costs of HIV healthcare in the HAART era. *AIDS.* Nov 13 2010;24(17):2705-2715.

28. Pinkerton SD. How many sexually-acquired HIV infections in the USA are due to acute-phase HIV transmission? *AIDS.* Jul 31 2007;21(12):1625-1629.

29. Prabhu VS, Hutchinson AB, Farnham PG, et al. Sexually acquired HIV infections in the United States due to acute-phase HIV transmission: an update. *AIDS.* Aug 24 2009;23(13):1792-1794.

30. Marks G, Crepaz N, Janssen RS. Estimating sexual transmission of HIV from persons aware and unaware that they are infected with the virus in the USA. *AIDS.* Jun 26 2006;20(10):1447-1450.

31. Marks G, Crepaz N, Senterfitt JW, et al. Meta-analysis of high-risk sexual behavior in persons aware and unaware they are infected with HIV in the United States: implications for HIV prevention programs. *J Acquir Immune Defic Syndr.* Aug 1 2005;39(4):446-453.

32. Attia S, Egger M, Muller M, et al. Sexual transmission of HIV according to viral load and antiretroviral therapy: systematic review and meta-analysis. *AIDS.* Jul 17 2009;23(11):1397-1404.

33. HIV prevalence estimates--United States, 2006. *MMWR Morb Mortal Wkly Rep.* Oct 3 2008;57(39):1073-1076.

34. Mocroft A, Phillips AN, Ledergerber B, et al. Estimated average annual rate of change of CD4(+) T-cell counts in patients on combination antiretroviral therapy. *Antivir Ther.* 2010;15(4):563-570.

35. Sanders GD, Bayoumi AM, Sundaram V, et al. Cost-effectiveness of screening for HIV in the era of highly active antiretroviral therapy. *N Engl J Med.* Feb 10 2005;352(6):570-585.

36. Zaric GS, Barnett PG, Brandeau ML. HIV transmission and the cost-effectiveness of methadone maintenance. *Am J Public Health.* Jul 2000;90(7):1100-1111.
37. Gold MR, Siegel JE, Russell LB, et al., eds. *Cost-Effectiveness in Health and Medicine*. New York: Oxford University Press; 1996.
38. Panel on Antiretroviral Guidelines for Adults and Adolescents. Guidelines for the use of antiretroviral agents in HIV-1-infected adults and adolescents. In: Department of Health and Human Services, edDecember 1, 2009:1-161.