Expanded Access to Highly Active Antiretroviral Therapy: A Potentially Powerful Strategy to Curb the Growth of the HIV Epidemic

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We developed a mathematical model using a multiple source of infection framework to assess the potential effect of the expansion of highly active antiretroviral therapy (HAART) coverage among those in medical need on the number of individuals testing newly positive for human immunodeficiency virus (HIV) and on related costs in British Columbia, Canada, over the next 25 years. The model was calibrated using retrospective data describing antiretroviral therapy utilization and individuals testing newly positive for HIV in the province. Different scenarios were investigated on the basis of varying assumptions regarding drug resistance, adherence to HAART, therapeutic guidelines, degree of HAART coverage, and the timing of HAART uptake. Expansion of HAART lead to substantial reductions in the growth of the HIV epidemic and related costs. These results provide powerful additional motivation to accelerate the roll out of HAART programs aggressively targeting those in medical need, both for their own benefit and as a means of decreasing new HIV infections.

The continued growth of the HIV epidemic poses a formidable challenge, even in the developed world [1]. The incidence of HIV infection remains unacceptably high [2]. Despite their potential efficacy and obvious appeal, HIV prevention strategies have been only partially successful [3]. Unfortunately, neither a cure nor a highly effective preventive vaccine are anticipated within the foreseeable future [4, 5]. At the rate of growth of the epidemic, in addition to the increasing human toll, the costs attributable to care and management of this growing and aging population may become unsustainable, in part because of the success of therapy in keeping infected patients alive for long periods [6–8].

Highly active antiretroviral therapy (HAART) has led to a dramatic decrease in morbidity and mortality among individuals infected with HIV, and international guidelines widely recommend that HAART be used before overt immune deficiency is apparent [9, 10]. The main goal of HAART is to attain durable suppression of viral replication (i.e., plasma HIV-1 RNA levels <50 copies/mL) [9, 10]. In recent years, provision of HAART to those in need has become an increasingly important and feasible global priority [11]. The current strategy is focused on the roll out of HAART programs on the basis of strict medical need [12, 13]. Priority is therefore being given to the treatment of those at the highest risk of disease progression or death. However, HAART coverage remains suboptimal, even in resource-rich areas of the world [11]. For example, despite universal access to health services and antiretroviral therapy in the province of British Columbia (BC), Canada, it is estimated that only 50% of those medically eligible to receive HAART are currently taking therapy [6]. Logistical and funding limitations have typically hampered the ability of therapeutic programs to expand to hard-to-reach popula-
tions or to those at earlier stages of HIV infection, leading to avoidable HIV/AIDS-related morbidity, mortality, and associated resource utilization.

In adherent patients, HAART predictably decreases plasma HIV-1 RNA levels to below the levels of detection of currently available assays [14]. As a result, HAART leads to an immunological benefit characterized by an increase in CD4 cell count, reducing, hence, the risk of AIDS-related disease progression and death [15]. Of note, HAART-induced decreases in plasma HIV-1 RNA levels have been shown to be associated with similarly marked reductions in HIV-1 RNA levels in genital secretions in men and women [16, 17] and with a substantial reduction in the risk of mother-to-child HIV transmission when HAART is given to HIV-infected pregnant women [18–20]. More recently, HAART has been shown to be associated with a decrease in HIV transmission between serodiscordant heterosexual couples, despite continued exposure [21–23]. It is therefore reasonable to speculate that antiretroviral therapy could have an additional beneficial impact by reducing the spread of HIV [24].

Several early mathematical modeling studies raised the concern that any possible benefit of antiretroviral therapy on the spread of HIV could be readily offset by even modest increases in HIV risk behavior [25–30]. More recently, however, Abbas et al. [28] have revisited this issue, proposing a mathematical model that predicts that antiretroviral therapy should have both individual and public health benefits that increase with time and, importantly, with the proportion of infected persons treated.

We therefore conducted the present study to assess the potential effect of expanding HAART coverage on the number of individuals testing newly positive for HIV and on related direct treatment costs in British Columbia over the next 25 years, using varying scenarios regarding rates of emergence of HIV drug resistance, degree of adherence to HAART, shift in therapeutic guidelines, and degree of HAART coverage.

**METHODS**

**The British Columbia Centre for Excellence in HIV/AIDS (BC-CfE) transmission model.** The BC-CfE semideterministic dynamic transmission model (figure 1) was built on the basis of the different stages of the natural history of HIV infection, whereby lower CD4 cell counts and higher plasma HIV-1 RNA levels are independently associated with a higher chance of HIV transmission [31–33]. The model also takes into consideration different predominant sources of HIV transmission in British Columbia. This includes transmission from men who have sex with men (MSM), injection drug users (IDUs), and MSM/IDUs, allowing the evaluation of the individual and overall contribution of these sources to the epidemic. The model characterizes the natural history of HIV infection on the basis of 4 infectivity periods: susceptible, primary infection (≤12 weeks since HIV infection), symptomatic phase defined by 4 plasma HIV-1 RNA strata (<3, ≥3 and <4, ≥4 and <5, and ≥5 log_{10} copies/mL), and late stage (individuals experiencing opportunistic diseases) [31]. During any of these infectivity periods we considered death as a possible outcome. The model considers a CD4 cell count ≤200 cells/mm^3 to be the key eligibility criterion for starting therapy.

In this model, plasma HIV-1 RNA levels and CD4 cell counts are updated every 3 months until death, and patient trajectories follow 3 main pathways. In the first, patients never receive treatment, and the plasma HIV-1 RNA and CD4 cell count trajectories are assumed to follow the natural history of HIV infection until death [31]. The second represents patients receiving HAART (i.e., started therapy with a CD4 cell count ≤200 cells/mm^3), and CD4 cell counts and plasma HIV-1 RNA levels are modeled using populational data collected through the BC-CfE Drug Treatment Program (BC-CfE DTP). The third pathway represents patients waiting to start treatment once their CD4 cell count reaches ≤200 cells/mm^3. Antiretroviral agents are distributed at no cost to all eligible HIV-infected British Columbia residents through the DTP under the auspices of the BC-CfE [14, 34]. Antiretroviral therapy guidelines are regularly updated by the center and remain consistent with those of the International AIDS Society–USA [9].

We modeled the epidemic in 3 major high-risk populations: MSM, IDUs, and MSM/IDUs. Appendix A, which appears only in the electronic edition of the Journal presents all of the details for the building of the BC-CfE transmission model. The transmission parameters for each risk category are described at the beginning of appendix A. The assumptions for these parameters were based on an extensive review of the literature and on consultations with experts in the field. These assumptions were calibrated and validated using 2 sources of information: the number of individuals testing newly positive for HIV obtained from the British Columbia Centre for Disease Control (BC-CDC) [35, 36], and the past and current active number of patients enrolled in the BC-CfE DTP. Figure 2A and 2B demonstrates that the parametric assumptions are adequate to fit the empirical data (P > .5 for statistical difference).

**Estimates of direct treatment costs.** Cost estimates associated with HAART expansion were based on the BC-CfE DTP and on the current recommendations for treating a treatment-naive patient with HIV/AIDS [9]. In 2006, the direct treatment cost to treat each person with first-line therapy was estimated to be Can$17,288.22 (source, BC-CfE DTP). To calculate the treatment cost in the years 2007–2030, we assumed a discounting of the future health rate equal to 3%, after considering what value of discounting is conventionally used in the literature [37]. To obtain the lifetime cost for treating each individual testing newly positive for HIV, we based our estimate on a life expectancy of 22.9 years at the age of 30 years [34].
Structure of the BC-CfE model. Appendix A presents the BC-CfE transmission model in detail. Briefly, the BC-CfE model is composed by 1 deterministic and 2 random components. The deterministic component estimates the number of individuals testing newly positive for HIV generated each time the model is updated, defined by the set of differential equations for each risk category. The 2 random components consist of 2 statistical modeling techniques to predict CD4 cell count and plasma HIV-1 RNA level trajectories and the probability of the emergence of HIV drug resistance. We used generalized additive mixed models (GAMMs) to predict CD4 cell count and plasma HIV-1 RNA level trajectories [38]. GAMMs are an extension of generalized linear models, which allow for fitting smooth nonparametric curves describing the association between outcomes and predictor variables.

The following predictors were investigated: age, sex, CD4 cell count, plasma HIV-1 RNA level (log_{10} transformed), presenting date, previous AIDS diagnosis, history of injection drug use, first HAART date, first HAART regimen, adherence, drug resistance, and death. The presenting date was defined as the date of the first (ever) plasma HIV-1 RNA level and CD4 cell count measurement before therapy initiation. Estimates of adherence to antiretroviral therapy were based on dispensed medications, also known as refill compliance. For this study, we limited our measurement of adherence to the first year of therapy, estimated by dividing the number of months of medications dispensed by the number of months of follow-up. This measure of adherence has been found to be independently associated with HIV suppression and survival among HIV-infected persons enrolled in the DTP [34, 39]. HIV drug-resistance genotyping data were based on assays attempted on all plasma samples collected during the first 30 months after the initiation of HAART for which the plasma HIV-1 RNA level was ≥1000 copies/mL or by physician request. Samples were assigned to 1 of 4 resistance categories (nucleoside reverse-transcriptase inhibitor [NRTI], protease inhibitor [PI], or nonnucleoside reverse-transcriptase inhibitor [NNRTI], or lamivudine/emtricitabine resistance [3TC]) on the basis of a modification of the International AIDS Society–USA table, as detailed in D’Aquila et al. [40].
RESULTS

Predictors of population plasma HIV-1 RNA level and drug resistance. The main predictors of current plasma HIV-1 RNA level were adherence and previous plasma HIV-1 RNA level, whereas the main predictors of the emergence of drug resistance were plasma HIV-1 RNA level, CD4 cell count, and adherence. The estimates of resistance by adherence level were derived from previously published data [41]. Of note, the current level of adherence in the BC-CfE DTP is estimated to be 78.5% [41]. In brief, individuals with adherence levels in the stratum of 80% to <95% were more likely to have any drug resistance (median probability, 0.44; interquantile range [IQR], 0.32–0.56), followed by 95% to 100% (median probability, 0.40; IQR, 0.27–0.51), 40% to <80% (median probability, 0.23; IQR, 0.16–0.31), and 0% to <40% (median probability, 0.19; IQR, 0.11–0.28) (figure 3). These predicted probabilities of resistance were further stratified by plasma HIV-1 RNA level, CD4 cell count, and exposure category (MSM, IDUs, and MSM/IDUs) and incorporated into the BC-CfE model.

Impact of the expansion of HAART coverage. The BC-CfE model was used to estimate the potential impact of the progressive expansion of HAART coverage for those who need it on the number of individuals testing newly positive for HIV. Current HAART coverage in the province of British Columbia is estimated to be 50% of the individuals in immediate medical need (namely, those with symptomatic disease or a CD4 cell count ≤200 cells/mm³) [6]. Additional HAART coverage rates modeled include 75%, 90%, and 100%. Our model suggests that, at the present level of coverage of 50% and with a stable level of adherence (presently estimated to be 78.5% on the basis of provincial refill compliance data [41]), we can expect to see an increase in the annual number of individuals testing newly positive for HIV from 421 to 462 between 2006 and 2030 (figure 4 and figure 5 [4 panels on the left-hand side]). Our model further suggests that an increase in HAART coverage to 75%, 90%, and 100% of those in medical need (with no change in the criterion used for HAART eligibility or in adherence level) would lead to a decrease in the annual number of individuals testing newly positive for HIV in the province of British Columbia of 37%, 54%, and 62%, respectively. Furthermore, if adherence were to decrease to a level below 40% (in the 50% coverage scenario), the number of individuals testing newly positive for HIV would be predicted to increase by 10% per annum. In contrast, if adherence were to increase to levels above 80% (in the 50% coverage scenario), the number of individuals testing newly positive for HIV would be expected to decrease by 0.3% per annum. As shown in the figure, the magnitude of this change would increase substantially with enhanced coverage rates.

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

Figure 2. A, Comparison of empirical (source: British Columbia Centre for Excellence in HIV/AIDS drug treatment program [DTP]) and estimated no. of individuals receiving antiretroviral treatment from 1993 through 2005. B, Comparison of empirical (source: British Columbia Centre for Disease Control [BC-CDC]) and estimated no. of individuals testing newly positive for HIV from 1993 through 2005.

Figure 3. Estimated probability of emergence of HIV drug resistance, stratified by adherence level (0% to <40%, 40% to <80%, 80% to <95%, and 95% to 100%).
Effect of changes in HAART initiation on HIV transmission.
We further explored the potential effect of a relatively modest change in the initiation of HAART from a CD4 cell count ≤200 cells/mm³ to a CD4 cell count ≤350 cells/mm³, which is still within the range of current guidelines [9]. The change is dependent on the degree of coverage and the level of adherence. For example, initiating HAART at 350 CD4 cells/mm³ with a stable coverage rate of 50% and an adherence rate of 78.5% would be expected to be associated with an increase in the annual number of individuals testing newly positive for HIV from 419 to 432 between 2006 and 2030 (figure 4B and figure 5 [4 panels on the right-hand side]). In contrast, if the same change in therapy initiation is associated with an increase in HAART coverage to 75%, 90%, and 100%, the model predicts that the annual number of individuals testing newly positive for HIV will decrease by 40%, 58%, and 67%, respectively. Also, if adherence in the population were to increase to levels between 80% and 100% in the 50% coverage scenario, the number of individuals testing newly positive for HIV would decrease by 40%, 58%, and 67%, respectively.

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

Figure 4. Results from the British Columbia Centre for Excellence in HIV/AIDS transmission model of the effectiveness of highly active antiretroviral therapy (HAART) uptake scenarios and coverage rates on the no. of individuals testing newly positive for HIV from 2006 through 2030. In panels A and B, we considered the effect of immediate (1-year) HAART uptake by varying coverage rates (from 50% to 75%, 90%, or 100%) and the impact of drug resistance measured indirectly through adherence (78.5% [current level]), given current guidelines (CD4 cell count ≤200 cells/mm³) (A) and new guidelines (CD4 cell count ≤350 cells/mm³) (B). In panel C, we considered the effect of HAART uptake given current guidelines (CD4 cell count ≤200 cells/mm³) and current adherence level (78.5%) and varying coverage rates from 50% to 75% and uptake scenarios (1, 3, or 6 years).

Figure 5. Results from the British Columbia Centre for Excellence in HIV/AIDS transmission model on the effectiveness of highly active antiretroviral therapy (HAART) on the no. of individuals testing newly positive for HIV.
HIV would be predicted to decrease by 41% to 67%, depending directly on the higher coverage rate.

The effect of changing therapy initiation and coverage rates on the cumulative averted number of individuals testing newly positive for HIV is shown in table 1. After 25 years, at the current adherence level a total of 3108 (29% of total new positive test results), 4776 (44%), and 5701 (53%) new positive HIV test results could be averted if the coverage rate were to increase from 50% to 75%, 95%, and 100%, respectively.

**Impact of varying rates of HAART uptake.** We further explored the impact of varying rates of expanded HAART uptake using our model. Maintaining the current guidelines (CD4 cell count ≤200 cells/mm³) and adherence level (78.5%), we considered 3 uptake scenarios: 1 year (immediate), 3 years (mid-term), and 6 years (long term). Figure 3C (as well as figure 6) shows the model predictions for the number of individuals testing newly positive for HIV in each of the scenarios. These scenarios produce a consistent pattern whereby faster expansion of HAART uptake is associated with a faster decrease in new positive HIV test results. For example, it is anticipated that it would take an additional 2 to 4 and 13 to 14 years for the 3- and 6-year uptake scenarios, respectively, to catch up with the 1-year uptake scenario with regard to the averted number of individuals testing newly positive for HIV.

**Results for direct treatment costs associated with HAART expansion.** Next we present the predictions of the model for the excess direct costs for treating all HIV-positive individuals in British Columbia who were infected because of delaying HAART expansion. In general, immediate HAART uptake is costly in the short-term; however, it is also associated with decreased number of individuals testing newly positive for HIV. Table 2 shows the total averted number of individuals testing newly positive for HIV and direct treatment saving costs associated with treating more individuals. As noted in this table, the longer it takes to expand HAART coverage, the more new people testing positive for HIV are observed (from 34 to 414, depending on the scenario). We also observed that if HAART expansion were done

### Table 1. Total no. of individuals testing newly positive for HIV and averted no. of individuals testing newly positive for HIV from 2006 through 2030.

<table>
<thead>
<tr>
<th>Therapy initiation, treatment coverage rate</th>
<th>Adherence level</th>
<th>Total no. testing newly positive</th>
<th>Averted no. testing newly positive</th>
</tr>
</thead>
<tbody>
<tr>
<td>HAART at ≤200 cells/mm³</td>
<td>Actual (78.5%)</td>
<td>0% to &lt;40%</td>
<td>80% to &lt;95%</td>
</tr>
<tr>
<td>50% (actual)</td>
<td>10,771</td>
<td>11,291</td>
<td>10,759</td>
</tr>
<tr>
<td>75%</td>
<td>7663</td>
<td>8333</td>
<td>7649</td>
</tr>
<tr>
<td>90%</td>
<td>5995</td>
<td>6714</td>
<td>5981</td>
</tr>
<tr>
<td>100%</td>
<td>5070</td>
<td>5803</td>
<td>5057</td>
</tr>
<tr>
<td>HAART at ≤250 cells/mm³</td>
<td>50% (actual)</td>
<td>10,564</td>
<td>11,265</td>
</tr>
<tr>
<td>75%</td>
<td>7144</td>
<td>8049</td>
<td>7112</td>
</tr>
<tr>
<td>90%</td>
<td>5328</td>
<td>6300</td>
<td>5295</td>
</tr>
<tr>
<td>100%</td>
<td>4327</td>
<td>5320</td>
<td>4296</td>
</tr>
</tbody>
</table>
| NOTE. HAART, highly active antiretroviral therapy; –, indicates that 50% coverage was the reference group.
immediately, it would generate total and per capita lifetime treatment cost savings starting at Can$95 million and Can$368 thousand.

**DISCUSSION**

In the present study, we investigated the potential impact of scaling up HAART, as a strategy to decrease HIV load at the population level, on the spread of HIV. We considered different scenarios by varying guidelines, coverage rates, HAART coverage, and adherence. Our model consistently predicted that enrolling at least 75% of individuals clinically eligible for treatment would be associated with a substantial decrease in new HIV infections. Furthermore, we showed that there is a complex interaction between guidelines for the initiation of therapy (i.e., the size of target population), HAART coverage, and adherence as well as the emergence of resistance with respect to the relationship between HAART coverage and HIV incidence. In summary, our model shows that the impact of HAART expansion on HIV transmission is optimized by rapid entry and a high rate of coverage of medically eligible HIV-infected individuals and by higher adherence.

Previous studies have produced conflicting evidence regarding the impact of HAART on the spread of HIV [25–28, 30, 42–45]. These studies include mathematical simulation models [25–28, 42–45] and ecological studies [30]. Although most studies support the notion that increased use of HAART could lead to a decrease in HIV incidence, the magnitude of the effect remains poorly characterized. Furthermore, some of these studies have suggested that any potential gain derived from the expansion of HAART coverage on HIV transmission could be easily overwhelmed by even modest increases in high-risk behavior or decreased adherence to HAART [28, 42–44]. Our results suggest that these concerns, although still valid, may have been overestimated, largely because previous models tended to underestimate the effect of HAART on HIV transmission. Of note, in British Columbia there has been a substantial growth in the incidence of sexually transmitted diseases, including syphilis (from 0.5 to 6.8/100,000 population), gonorrhea (from 13.0 to 28.0/100,000 population from 1996 to 2005), and chlamydia (from 123.4 to 213.3/100,000 population from 1996 to 2005) with no overall impact on new HIV infections per year [32, 33, 46].

Our model and those already published in the literature are different in several ways [25–28, 30, 42–50]. The BC-CfE transmission model has a random structure that is comprised of advanced statistical methods using GAMMs, which allow the transmission probabilities (which highly depend on plasma HIV-1 RNA level and other clinical and behavioral parameters) to change over time. The structure of this transmission model allowed us to take into account the complexity of the process underlying the HIV epidemic without requiring elaborate assumptions about the distribution of plasma HIV-1 RNA level, CD4 cell count, and other clinical parameters over time. The model also considered different assumptions for the level of therapy adherence in a population, given that scaling up treatment can potentially increase the likelihood of suboptimal adherence, virologic failure, mortality, and drug resistance. In addition, the dynamic aspect of the BC-CfE model allowed us to adjust our estimates for changes in the prevalence of individuals carrying drug-resistant HIV variants in the population. Differently from other models, the BC-CfE model incorporated changes over time in important behavior parameters that are influential in the HIV epidemic of major risk groups in British Columbia (MSM, IDUs, MSM/IDUs), via the calibration of parameters on the basis of the data from BC-CDC and the BC-CfE DTP. Finally, this model is flexible and can produce short- and long-term predictions of the effect of expanded HAART coverage on the HIV epidemic.

Several unique features of the model have been described here. First, our initial model represents a theoretical exercise aimed to relate the impact of lowering HIV load at the population level, via improvements in HAART coverage, on HIV transmission. In a second stage, the model was optimized by adjusting infectivity parameters to match empirical data collected pro-

<table>
<thead>
<tr>
<th>HAART coverage</th>
<th>Averted no. of individuals testing newly positive</th>
<th>Direct treatment cost savings for 1-year uptake, Can$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1-year uptake</td>
<td>3-year uptake</td>
</tr>
<tr>
<td>50% to 75%</td>
<td>3202</td>
<td>3169</td>
</tr>
<tr>
<td>75% to 90%</td>
<td>1724</td>
<td>1700</td>
</tr>
<tr>
<td>90% to 100%</td>
<td>959</td>
<td>982</td>
</tr>
</tbody>
</table>

**NOTE.** On the basis of data from the British Columbia Centre for Excellence in HIV/AIDS Drug Treatment Program, the estimated direct treatment cost for a person starting first-line therapy was Can$17,288.22 in 2006, and a discount rate of 3% was used for adjustment of cost during 2007–2030. The life expectancy used to calculate the lifetime cost savings was 22.9 years for a person infected at the age of 20 years who started treatment at a median of 10 years after infection.
spective in the past between 1993 and 2005. Then, we used semideterministic mathematical modeling to predict the effect of HAART expansion over time, which allowed us to work with a small number of simple differential equations. Our model was built on the principle that expanded access to HAART will be used as a supplement, rather than replacement, of current HIV control programs, such as education about and promotion of safer sex, condom use, microbicides, needle exchanges, and methadone maintenance, to cite a few. Therefore, a major feature of this mathematical model is that the risk behavior–specific transmission parameters are not detrimentally influenced by the expansion of HAART coverage over the next 25 years. We also should emphasize that, for expansion of HAART coverage to be a successful complementary strategy to limit the HIV epidemic, it is necessary that HIV screening programs also be expanded in order to discover patients without a diagnosis who are unaware of their HIV status. Finally, our model is flexible enough to easily incorporate and access different treatment coverage rates, different adherence scenarios, and assumptions regarding any of the transmission parameters. Of note, the number of individuals testing newly positive for HIV was used as a surrogate for HIV incidence in our study, because the latter is not routinely determined by the provincial health authorities [35, 36]. Future work should be aimed to carefully characterize the relationship between expansion of HAART coverage, HIV load, and HIV incidence prospectively.

In conclusion, we have successfully simulated and validated the dynamics of the HIV epidemic in British Columbia, particularly as it relates to the impact of expanding HAART coverage and the emergence of individuals testing newly positive for HIV. Our results indicate that a higher HAART coverage consistently leads to a decrease in the number of individuals testing newly positive for HIV. Although increased use of HAART is associated with an increase in the prevalence of individuals carrying drug-resistant virus, as a result of varying degrees of incomplete adherence and, consequently, high HIV-1 RNA plasma viral load, this appears to be relatively small and has a limited impact on the overall strategy. We therefore conclude that expansion of HAART coverage should lead to a substantial reduction of the growth of the HIV epidemic and related direct treatment costs. Our model supports a powerful and as-of-yet little appreciated additive preventive value of expanding HAART coverage.

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References


