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Impacts of HIV and Population-Wide Treatment on the Elderly in a Simulated African Population

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Abstract

The HIV/AIDS pandemic is reshaping the populations of sub-Saharan Africa through increasing mortality and decreasing fertility. A two-sex, stochastic microsimulator is used to describe some of the HIV-related impacts on the structure of infected populations similar to those living in Southern Africa with an emphasis on the consequences for the elderly and the effects of population-wide treatment. After 40 years of epidemic the growth rate is reduced to zero or below; the age structure converges on a younger, two-tiered shape with fewer young children and adults; the age-specific sex ratio attains a high of roughly 1.5 males per female in the 45-49 age range and a low of 0.5 males per female at ages older than 70; the dependency ratio falls substantially; the number of orphans rises to 37 percent (maternal), 30 percent (paternal) and 22 percent (dual) of all children alive below age 15; and the percentage of adults aged 50 and older who have surviving grandchildren but no surviving children rises to 23 percent (females) and 13 percent (males). The impacts of preventative and antiretroviral treatment programs initiated in year 31 of the 40 year simulations are described and compared. The significant fertility reduction that accompanies the preventative program has strong undesired effects while the antiretroviral programs are generally less disruptive to population dynamics. A combination of the two approaches is clearly the most beneficial and usually less disruptive than either applied separately.

(235 words)

Key Words

HIV, AIDS, Simulation, Africa, Elderly, Orphans, Growth Rate, Mortality, Fertility, Individual-level, Model.

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1 Introduction

The high levels and continued increase in HIV prevalence in parts of sub-Saharan Africa are bringing with them substantial increases in adult mortality (Dorrington et al., 2001; Timaeus, 1998; UNAIDS, 2002) and decreases in fertility(Gregson, 1994; Gregson et al., 1999; Zaba, 1998). These together change the composition of infected populations significantly with important consequences for the elderly. Added to this is the potential for widespread prevention and treatment programs that will have substantial impacts of their own.

One of the most important and widely recognized consequences for the elderly is the creation of a large number of "AIDS orphans" – children who lose one or both of their parents as a result of HIV-related mortality. The "Children on the Brink: 2002" report indicates that there were 38 million orphans in Africa in 2001 with 11 million of those attributable to AIDS mortality (UNAIDS UNICEF USAID, 2002). The same report predicts that there will be 42 million orphans in total in Africa in 2010 with 20 million of those the result of AIDS mortality, and others make even larger estimates of the number of orphans by 2010 (Monk, 2002). Bicego and colleagues (Bicego et al., 2003) observe a strong correlation between the percent children who are orphans and national HIV prevalence for several African countries during the 1990s, lending weight to the conclusion that HIV is related to the rise in the number of orphans.

The sheer number of orphans being created is unusual in human history, as is the fact that many are dual orphans who have lost both parents and must be cared for by someone else. Compounding the problem for the elderly is the fact that their own numbers and the proportion of the population they comprise are also being affected by the epidemic, and all of the changes wrought by an HIV epidemic evolve as the epidemic grows and stabilizes. Accordingly, to adequately understand the total impact of the epidemic on the number and proportion of children who are orphans and on the number of grandparents who will be alive to care for them, one must employ a "whole population" model that takes into account all of the major avenues through which HIV impacts a population and how these interact with one another. Gregson and colleagues constructed one such model in the mid 1990s (Gregson et al., 1994) and used it to predict the major increase in the number and proportion of children who are orphaned as a result of HIV. Many of their theoretical findings are being validated now as the epidemic unfolds and large numbers of orphans begin to appear in the worst effected African countries (UNAIDS UNICEF USAID, 2002).

Gregson and colleagues also examined more general demographic impacts of an HIV epidemic and demonstrated significant changes in the age-specific sex ratio, population age structure and overall growth rates – all of which affect the elderly as the underlying structure and size of the population changes. Results presented here largely corroborate their findings while adding some additional nuances.

The work presented here employs a different modeling strategy from that used by Gregson and colleagues to explore many of the same questions. The individual-level microsimulator employed here is capable of modeling marriage, sex and the biological and behavioral impacts of HIV. The individual-level nature of the model allows it to track the links between parents and children and grandparents and grandchildren. In

comparison to the deterministic, compartmental model used by Gregson and colleagues, this model provides a direct means through which to measure the number of orphans and the number of grandparents living with orphaned grandchildren. Additionally, it is able to realistically model two different types of intervention; a preventative largely behaviorally-mediated intervention, and an antiretroviral treatment program that reduces viral load and increases the time between infection and death without having other specific preventative effects. Treatment programs of these two types are simulated in the late phase of an HIV epidemic to ascertain their overall effects and how these impact on the situation of the elderly.

Below we discuss the specific questions to which the model is addressed and provide some context for each. Following that is a brief discussion of the simulator itself. A full description of the model is outside of the scope of this work but can be found in detail elsewhere (Clark, 2001a). Finally there is a detailed presentation of the results from fifteen 40-year simulations, three in each of five scenarios: 1) a stable population with no HIV, 2) a population with untreated HIV, 3) a population with HIV treated with a preventative program, 4) a population with HIV treated with antiretrovirals and 5) a population with HIV treated with both preventative and antiretroviral treatment programs.

2 Questions

As a heterosexually transmitted HIV epidemic grows and a substantial fraction of the general adult population is infected, the structure of the population gradually changes resulting from the influences of HIV – often mediated through behavioral mechanisms – on mortality, and perhaps more important, on fertility. These structural changes have significant implications for older people, and it is specifically these impacts that are the focus of the questions posed here.

In addition to the structural changes resulting from the disease directly, prevention and treatment programs have significantly different structural effects that arise from their varied effects on transmission and progression of HIV infections. Taking this into account each of the questions posed below is examined in five different treatment scenarios to ascertain the impacts of both the HIV epidemic itself and various treatment programs.

2.1 Population Growth Rates

The overall population growth rate is a gross measure of the stability of a population. Sharp changes in the population growth rate usually reflect significant transient and permanent changes in the age structure of the population and lead to populations of different overall sizes. Transitions of this type change the overall milieu in which older people live, the size of the support system that nurtures them, and potentially the size of the subgroups of the population that need their support. Questions relating to the impact of HIV on the population growth rate include:

1. What is the magnitude, direction and timescale of the effect of a realistic HIV epidemic on the population growth rate?
2. Is there any difference in the effects of a realistic HIV epidemic effect on the male

and female population growth rates?

3. How does the effect of a realistic HIV epidemic on the population growth rate change with the overall prevalence of HIV in the population?
4. What impact do preventative (behavioral) and antiretroviral treatment programs have on the population growth rate after an HIV epidemic has stabilized, is there a sex differential in these impacts?

2.2 Population Age Structure

Changes in the age structure of the population are of significant importance to the elderly because they reflect changes in the proportion of the population living at each age and hence in the ratios of people living at each age compared to other ages. Because it kills a large number of middle-aged people and prevents the birth of a large number of infants, an HIV epidemic causes very substantial changes in the population age structure. These changes are dynamic and evolve as the epidemic grows and matures. At each stage the implications for the elderly are different, but at all stages substantial. Questions relating to the impact of HIV on the population age structure include:

1. How do the proportions of children, young adults, adults and elderly change as a realistic HIV epidemic grows and matures?
2. Is there a sex differential in these changes?
3. What impact do preventative (behavioral) and antiretroviral treatment programs have on the population age structure after an HIV epidemic has stabilized, is there a sex differential in these impacts?

2.3 Age-Specific Sex Ratios

Heterosexually transmitted HIV epidemics infect females and males at different ages resulting in different age-specific prevalence patterns for females and males, and as a result the age distribution of HIV-related deaths is different for females and males. These age differences result in the potential for an impact on the age-specific sex ratio of the population with the possibility that the elderly population will be composed of substantially more of one or the other sex. Questions relating to the impact of HIV on the age-specific sex ratio include:

1. What is the impact of a realistic HIV epidemic on the age-specific sex ratio?
2. How does this impact change as the epidemic grows and matures?
3. What impact do preventative (behavioral) and antiretroviral treatment programs have on the age-specific sex ratio after an HIV epidemic has stabilized?
4. What implications do changes in the age-specific sex ratio have for the elderly?

2.4 The Dependency Ratio

The dependency ratio summarizes the population age distribution as the ratio of the number of people aged 0-14 and 65+ divided by the number of people aged 15-64. This standard indicator reflects the ratio of the number of "dependent" people to the number of productive people in the population. Through its impact on the population

age structure an HIV epidemic will change the dependency ratio in important ways. Questions relating to the impact of HIV on the dependency ratio include:

1. What is the impact of a realistic HIV epidemic on the dependency ratio?
2. Are there sex differentials in this impact?
3. How does the impact change as a realistic HIV epidemic grows and stabilizes?
4. What impact do preventative (behavioral) and antiretroviral treatment programs have on the dependency ratio after an HIV epidemic has stabilized?
5. What changes in the population age structure explain changes in the dependency ratio as a realistic HIV epidemic grows and stabilizes?

2.5 Number of Orphans

One of the most discussed impacts of heterosexually-transmitted HIV epidemics is the excess number of orphans they can generate, and the potential for those orphans to place additional burdens on the elderly. Establishing the relationship between the population prevalence of HIV and the number of additional orphans generated is complicated by the facts that 1) HIV-related mortality of females and their children is strongly correlated leading to significant excess HIV-related mortality of orphans who result from HIV-related mortality of women and 2) the magnitude of non-HIV-related excess mortality suffered by orphans as a result of simply being orphans is poorly measured. This is mainly because orphans have historically been relatively rare, are often difficult to identify if they have been fostered, are less likely than non-orphans to be covered by many data collection systems, and finally because the excess mortality seems to persist for only a short period immediately after the death of the parent(s) it is less likely that dead children who have very recently been orphaned are properly categorized as orphans at the time of their death.

Given that accurate, reliable, empirical data describing orphans are unusual, we *must* turn to modeling to gain a better understanding of the relationship between overall indicators of an HIV epidemic and the number and sex-age distribution of orphans. To accurately model the creation of orphans, the link between *individual* parents and their *individual* children must be represented – something that is only possible in an individual-level modeling framework like the one employed here. In the context of HIV/AIDS maternal, paternal and dual orphans are of interest which requires the model to represent and account for marriage so that children who lose their father can be identified. However simply accounting for the links between parents and children is insufficient because the overall number of orphans of a given age is a function of fertility, adult mortality, child mortality and the size of the base population of adults and orphans to which those rates are applied. Consequently to realistically model orphanhood it is necessary to accurately model an entire two-sex population at the individual level with realistic pairing dynamics.

The model utilized for this work is of this type and is suitable to accurately model orphanhood dynamics in the context of a whole population. Because mother-to-child transmission of HIV is modeled and infection with the HI virus has realistic duration-since-infection impacts on the mortality of both adults and children, the correlation between the mortality of mothers and children resulting from HIV infection is correctly modeled. The model *does not* account for the additional non-HIV-related excess

mortality associated with being an orphan; empirical work in progress by the author suggests that there is very substantial excess mortality associated with becoming an orphan – during the first year after losing their mother for young children. Taking this into account, the mortality of orphans is underrepresented in the model and consequently the model produces a conservative (slight over) estimate of the number of orphans.

Specific questions relating to the number and fraction of all children who are maternal, paternal and dual orphans as an HIV epidemic grows and stabilizes include:

1. How many and what percent of children are maternal, paternal and dual orphans in a rapidly growing population with relatively high mortality but no HIV?
2. How many and what percent of children who are maternal, paternal and dual orphans in the same population infected with HIV, and how do these indicators change as the HIV epidemic grows and stabilizes?
3. What impact do preventative (behavioral) and antiretroviral treatment programs have on the number and percent of children who are maternal, paternal and dual orphans after an HIV epidemic has stabilized?

2.6 Numbers of Children and Grandchildren

The important intergenerational consequence (alluded to above) of a significant increase in the number of orphans is that after children lose both parents grandparents often must take responsibility for them. If large numbers of maternal and dual orphans are produced, these orphaned grandchildren will become a significant challenge for grandparents, and in particular grandmothers, who must care for them.

To adequately describe and investigate this issue, all links between parents and children must be known so that links between grandparents and grandchildren can be reconstructed. Empirical studies of this issue must both record these links and have good data to describe orphans – something that very few empirical studies have. This strongly motivates a modeling approach to gain insight into these issues. The model employed here produces a realistic base population effected by HIV, including the number and age distribution of children, orphans, adults and adults aged 50 and over. Combined with the links between parents and children, it is possible to examine changes in the average number of orphaned grandchildren per adult aged 50 and over, taking into account sex differentials brought about by the sex differentials in HIV-related mortality and consequent distortions in the age-specific sex ratio.

Specific questions relating to the number of orphaned grandchildren per adult aged 50 and older as an HIV epidemic grows and stabilizes include:

1. How many and what percent of adults aged 50 and older have surviving orphaned grandchildren but no surviving children in a rapidly growing population with relatively high mortality but no HIV?
2. How many and what percent of adults aged 50 and older have surviving orphaned grandchildren but no surviving children in the same population infected with HIV, and how do these indicators change as the HIV epidemic grows and stabilizes?
3. Are there sex differentials in the number and percent of adults aged 50 and older who have surviving orphaned grandchildren but no surviving children as an HIV epidemic grows; what are they?

4. How are the number and percent of adults aged 50 and older who have surviving orphaned grandchildren but no surviving children related to HIV prevalence of the population and of the population 50 and older as an HIV epidemic grows and stabilizes?
5. What impact do preventative (behavioral) and antiretroviral treatment programs have on the number and percent of adults aged 50 and older who have surviving orphaned grandchildren but no surviving children after an HIV epidemic has stabilized?

3 Methods

The questions posed above are generally not amenable to empirical investigation *in their entirety* because that would require observation of whole populations over the entire period during which HIV epidemics subjected to various forms of treatment grow and stabilize. Additionally the level of detailed information necessary to investigate the intergenerational questions is typically not available from empirical studies. And finally, ethical concerns relating to measuring the incidence and prevalence of HIV would make empirical studies of this type prohibitively expensive and in most cases impossible – through the influence of the investigation on the process.

In contrast, modeling is comparatively *very* cheap, flexible, fast and ethically neutral. The primary disadvantage is that models are not the real thing nor will they even reflect the real thing very well; so whatever insights we gain through modeling are transmitted through a comparatively blurry, low resolution lens. Additionally, the closer a model is to representing the complexity of reality, the more difficult it is to parameterize with empirically observed parameters. So we live with relatively simple models that average over the internal complexity of reality and reflect the aggregate reality well or more complex models that begin to adequately reflect the internal complexity of reality but do not reflect aggregate reality well because complete sets of empirically observed parameter values are not available. Models in the first group are useful to project or predict into the future with reasonable accuracy, while models in the second group are useful to probe the inner workings of a system and understand how the components of a system are related to each other in a dynamic sense over time and as the system is perturbed.

Because we are interested in understanding how heterosexually transmitted HIV effects populations, we want to understand how it effects various components of a population and how these effects in turn effect other components of the population and interact synergistically to produce the population-level impacts that we observe. Investigations of this type are best pursued by using the second type of model that offers an ability to manipulate and observe the inner workings of a process in detail.

The questions posed here are investigated using a two-sex, individual-level stochastic microsimulator (Clark, 2001d) parameterized with empirically observed mortality, fertility and nuptiality parameters describing a rural population living in Southern Zambia between 1950 and 1995 (Clark, 2001c) so that the populations it simulates closely resemble rapidly growing, high fertility, high mortality, polygynous populations similar to those now living in Southern Africa. The simulator models polygynous

marital and both polygynous and polyandrous non-marital pairings of men and women, and within all these types of union, individual sexual intercourse events. The sexual intercourse events lead to both conceptions and the transmission of the HI virus, thereby tightly and accurately coupling reproduction of the population and transmission of the HI virus. Once infected with the HI virus an individual gradually develops AIDS and eventually dies. During this progression the disease impacts on both their behavior and biology causing them to be, among many things, less likely to enter into unions of all types, less likely to have sex, more likely to be divorced, increasingly more likely to die and if female less likely to conceive and more likely to miscarry.

3.1 Simulator

Following is a very brief description of the simulator containing just enough detail to understand what it is, how it works and what it produces. For a detailed description of the simulator please see Clark (Clark, 2001d), and for a complete exposition of the demographic parameters and how they were calculated please see Clark (Clark, 2001c). Both these references can be downloaded from (Clark, 2001b)

The simulator contains entities corresponding to *individual people*, *individual unions* (both marital and extra-marital) between men and women, *fertility histories* for women, and *pregnancies* for women. Together with the union-mediated *links* between spouses or partners and between parents and children, this is sufficient to model all the important dynamics of a whole population.

Simulated time is incremented in units of one month, and during each month, every entity is exposed to the risk of the events for which it is eligible. Event hazards governing the monthly probability of occurrence of each event are compared to random numbers to decide which events occur during a given month. These occurrences and their repercussions are recorded – often changing the eligibility of the effected entities for future events – and the process is repeated until the desired number of months have been simulated.

The simulator utilizes a relational database to store the long event histories generated by this process. The Structured Query Language (SQL) is used to manipulate the database – both to conduct the simulation and to extract data for analysis. The additional logic necessary to run the simulator is written in a standard programming language that makes changes to the database by executing SQL. A significant benefit of this architecture is that the entire history of every modeled entity is recorded permanently, which allows unlimited *ad hoc* analysis of the resulting simulated population using the same tools one would employ to analyze similar data describing a real population. Another significant benefit is that any simulation can be stopped and restarted without having to employ any special logic or procedures. Additional benefits include the ability to easily utilize both time invariant and time varying parameters, and through the straightforward use of SQL, to employ dynamically scaled parameters that are functions of aggregate indicators that are calculated on the fly as the simulation progresses.

3.1.1 Simulator Components

The simulator is organized around modules that manage events relating to 1)

mortality, 2) fertility, 3) nuptiality, 4) extra-marital unions, or affairs, and 5) sexual intercourse – which occurs with differing frequency in the context of the two types of unions between men and women. An additional module manages the effects of infection with HIV on the event probabilities that are managed by the other five modules. Please see Clark (Clark, 2001d) for a detailed description of these components and how they interact.

The set of parameters used to fully parameterize the simulator is too large to fully describe here; see Clark (Clark, 2001d) for details. Stable populations unaffected by HIV that result from running the simulator for many years with the parameter set used for this work have: 1) mortality levels associated with expectations of life at birth of about 50 years for males and about 52 years for females, 2) Fertility levels associated with a total fertility rate of about 6.2, 3) and annual proportionate growth rates of about 4%.

3.1.2 Effects of HIV

An HIV disease progression (DP) indicator is used to govern the progress of an infected individual's HIV infection. The DP indicator consists of a time series of values that correspond loosely to an infected individual's HIV viral load as the disease progresses. The shape of this indicator with time is different for children and adults, reflecting the different pace of the disease in children and adults. The shape of the DP indicator with time is that of a lop-sided "U" with the initial value being small but rapidly decreasing to a very small value that persists for some time and then increases very slowly. For children the rapid increase begins at about 18 months after infection, and for adults the DP begins to increase steadily from about 80 months reaching substantial levels at about 120 months.

HIV treatment programs that utilize antiretroviral pharmaceuticals to suppress viral load and extend the life of infected individuals are modeled by changing the shape and time scale of the DP indicator. For adults the whole curve is stretched by about ten years and the intermediate values are diminished to approximately zero – mimicking the effects of properly administered antiretroviral drugs. For children the curve is stretched by about three years.

The DP indictors translate into average times between death and infection of about ten years for adults and two years for children with no antiretroviral treatment, and twenty years for adults and five years for children with antiretroviral treatment.

Transmission of HIV between adults occurs only through heterosexual sexual intercourse from an infected individual to their partner with an average per intercourse probability of transmission of roughly 10^{-3} over the course of an HIV infected individual's disease. Individuals can be infected more than once, but their DP indicator starts at the date of their first infection. The actual transmission probability applied to each intercourse event is scaled by the DP indicator of the infected individual allowing the transmission probability to change as the disease progresses and roughly reflect the infected individual's viral load, and hence their potential to transmit.

Infected mothers transmit the HI virus to their newborns at birth with an average transmission probability of about 0.3 over the course of an infected woman's disease. Again, the specific transmission probability applied to a given birth is scaled by the mother's DP indicator allowing her transmission probability to track the progress of her disease and accurately reflect her viral load, and hence her potential to transmit at

each time following her own infection.

The transmission probability utilized in both the horizontal and vertical modes of transmission is governed by the infected individual's DP indicator allowing changes in the DP indicator – such as those that implement the virtual antiretroviral treatment program – to be reflected in the transmission probabilities.

Infection with HIV has a number of other effects whose details will not be discussed here beyond mentioning that they are implemented; see Clark (Clark, 2001d) for details on these effects. Being HIV positive effects:

- the probability that a conception will lead to a miscarriage,
- the fecundity of an infected female,
- the daily hazard of intercourse between a male and female if one or both are infected,
- the probability of transmitting the HI virus from an infected to an uninfected individual through sexual intercourse,
- the probability of transmitting the HI virus from an infected woman to her newborn child through the birth process,
- the probability that a possible couple with one or both possible partners infected will form a marital union,
- the probability that a marital union will dissolve if one or both of the partners is(are) infected, and
- the probability that an infected individual dies.

3.2 Indicators

Indicators presented to explore the questions posed above include measures of the composition of the population through time and the prevalence of HIV in various sex-age groups. The majority of these are familiar, are calculated in the standard way and should be self-explanatory.

The orphanhood indicators relate to maternal (mother dead), paternal (father dead) and dual (both parents dead) orphans. Raw numbers and percent of children who are orphans of each type are presented as a function of time. The percent children who are orphans is calculated as the ratio of the number of children who are orphaned (by type) to the total number of children, including the orphans.

The grandchildren indicators relate the total number of adults aged 50 and over (by sex) to the number of their surviving children and grandchildren over time. The indicators presented here are the sex-specific: 1) total number of adults aged 50 and older, 2) total number of adults aged 50 and older who have *no* surviving children but at least one surviving grandchild under the age of fifteen and 3) percent of total adults aged 50 and older who have *no* surviving children but at least one surviving grandchild under the age of fifteen.

3.2.1 HIV Prevalence

Sex-age-specific HIV prevalence is calculated as the ratio of the number of person years lived infected with HIV to the total number of person years lived. Age-specific HIV prevalence values are weighted by the age structure of wider age groups to

aggregate HIV prevalence across those wider age groups (i.e. 15-64).

3.3 Running the Simulator: Five Scenarios

To start the analysis presented here, a stable population consisting of 1,271 females and 1,230 males was created by letting the simulator run for 150 years from an initial population consisting of fifteen females and fifteen males of young reproductive age. This stable population served as the starting point for all of the simulations presented here and is referred to as **P0** below.

The choice was made to begin "treatment" in the treated simulations in year 31 of the 40 years that are simulated for each of the scenarios described below. This is to roughly mimic the sequence of events in Southern African HIV epidemics. HIV has been affecting Central Africa for 30 to 40 years and Southern Africa for 15 to 30 years. Widespread treatment programs are in the near future for South Africa and perhaps for other nations in the region, so it seems appropriate to let the HIV epidemics grow for three decades before instituting treatment programs in order to roughly mirror the timescale of the real epidemics and their potential treatment in Southern Africa. Future work will address the impact of treatment programs earlier and later in the life of an epidemic.

3.3.1 Population without HIV

Three simulations were run from P0 for 40 years without any HIV infection, resulting in final population sizes of roughly 12,000 individuals. These thee simulations serve as the healthy "control" to which the HIV-effected simulations can be compared. They also provide some feel for the level of stochastic variability that one may expect to see in the various indicators unaffected by HIV, but unfortunately it is not possible to explore this topic here. Averages of these three simulations are presented as the "No HIV" results below.

3.3.2 Population with Untreated HIV

Three simulations were run from P0 for 40 years with HIV. Throughout these simulations there was a *very* low hazard equal to 15 per 100,000 per month of "external" infection for adults aged fifteen to 49. Because the simulated population is a closed population the epidemic has to be seeded in some reasonably realistic way, and without modeling special high risk groups and there subdynamics within the population, this seems like the most reasonable approach. Over the course of a typical 40 year "HIV" simulation, about 2-3 percent of the infections of adults fifteen to 49 resulted from "random external" transmission – a value that does not seem unreasonable. Averages of these three are presented as the "HIV" results below.

3.3.3 Population with HIV Treated with Behavioral Preventative Programs

A behaviorally-mediated HIV prevention program was simulated using the three HIV simulations. Starting in year 31 and running for ten years through year 40, each of the three HIV populations were subjected to a complete cessation of the formation of extramarital relationships. This had the immediate effect of reducing women's exposure to intercourse and completely curtailed the high levels of sexual mixing across age groups that results from the formation of and dissolution of extramarital

relationships. The immediate consequences are a sharp reduction in fertility and a concurrent sharp reduction in the transmission (incidence) of HIV. Although this is a drastic and ultimately "unreal" treatment strategy, the results clearly demonstrate what the overall effect of such a program would be; realistic implementations could attain at best substantially less impact than that demonstrated here. Averages of these three simulations are presented as the "HIV with 'Behavioral' Treatment Starting in Year 31" results below.

3.3.4 Population with HIV Treated with Pharmacological Antiretroviral Programs

A pharmacologically-mediated HIV treatment program was simulated using the three HIV simulations. Starting in year 31 and running for ten years through year 40, in each of the three HIV populations the untreated DP indicator was removed and replaced with the lengthened and reduced DP indicator described above in "Effects of HIV" that reflects the impact of antiretroviral drugs on the progression of HIV disease and a treated infected individual's viral load. This had the effect of substantially reducing the transmission of HIV from mothers to children and between adults and of dramatically reducing the other impacts of HIV, including its fertility-reducing and mortality-elevating effects. This virtual treatment was applied to *all* HIV-infected individuals, and so like its behavioral corollary does not reflect a real-world treatment program that would undoubtedly only reach a limited fraction of HIV-infected individuals. That understood, it reveals the maximum effect that such a treatment program could have and a typical time course over which that effect might be observed. Averages of these three simulations are presented as the "HIV with Antiretroviral Treatment Starting in Year 31" results below.

3.3.5 Population with HIV Treated with Both Preventative and Antiretroviral Programs

The last set of three simulations apply both types of treatment using the three HIV simulations starting in year 31. Averages of these three simulations are presented as the "HIV with Both 'Behavioral' and Antiretroviral Treatment Starting in Year 31" results below.

4 Findings

4.1 Population Growth Rates

The simulated control population not effected by HIV has a stable overall population growth rate of four percent per annum for both sexes, panel 1 of Figure 1. When HIV is introduced into this population, panel 2 of Figure 1, it takes about ten years for all-age prevalence to rise more than a few percent, and at about this time the overall population growth rate for both sexes begins to decline. There is a steady decline that is similar for both sexes between years ten and 30 of the epidemic as all-age prevalence is rising steadily, but after year 30 when all-age prevalence begins to stabilize, at about 27 percent for females and 20 percent for males, the female growth rate declines faster than the male and drops slightly below zero. The male growth rate follows dropping below zero in about year 35 of the epidemic, and then they both

stabilize at about -0.5 percent. A realistic heterosexually transmitted HIV epidemic reduces a robust stable population growth rate of four percent to roughly zero over the course of roughly 20 years while the all-age prevalence of the HI virus is increasing from negligible levels to steady-state levels with about one quarter of the population infected, but with a significant sex differential leaving many more females than males infected.

Instituting a behavioral prevention program that eliminates all extramarital relationships between females and males in year 31 of the epidemic, panel 3 of Figure 1, has a very dramatic impact on the population growth rate immediately causing it to decline sharply over the course of three years from roughly zero percent for both sexes to roughly -2.5 percent for both sexes. Immediately after reaching its minimum it begins to recover to end the 40 year period of the simulation at about zero percent for both sexes. Over this same period, year 31 to year 40 of the epidemic, the all-age prevalence falls in a nearly linear trajectory from about 27 percent for females and 20 percent for males to roughly 5 percent for both sexes. The sharp reduction in the growth rates caused by this type of prevention program results from removing a significant exposure to sexual intercourse – and hence conception – for females. The steady recovery of the growth rate back to about zero percent between years 33 and 40 is the result of a change in the population prevalence structure toward a population with fewer HIV positive people. As the HIV negative population increases in size relative to the HIV positive population the fertility-inhibiting impacts of the disease have progressively less impact on the overall population fertility rate, and the overall population growth rate recovers. The sharp reductions in all-age prevalence result from the immediate curtailment (but not elimination) of incidence combined with ongoing HIV-related mortality of the HIV positive population – effectively reducing the "birth rate" of new infections to nearly zero without a concomitant change in the mortality of existing infections. A final note; this impact of this "prevention" program is exactly the same as the impact of an effective condom distribution and education program that results in condom usage within all extramarital relationships. The simulator has the ability to model condom usage, and future work will combine the behavioral intervention (reduction in extramarital relationships) with condom usage in both marital and extramarital relationships. The most important message from this result is that preventing transmission of the HI virus through abstinence (of one sort or another) or condom usage has a very substantial impact on fertility that in turn, over time, has a very substantial impact on the overall size and composition of the population.

Instituting an antiretroviral treatment program in year 31 produces radically different results, panel 4 of Figure 1. In this case the population growth rate immediately *increases* to nearly its pre-epidemic level of four percent. It then begins a very gradual decline to about 2.5 percent in year 40. At the same time all-age prevalence actually increases slightly above what it is in the untreated simulation – to levels of about 28 percent for females and 22 percent for males. This increase in prevalence has the same impact as the change in prevalence observed in the behavioral treatment program, except in the other direction and with much smaller magnitude. The increased prevalence increases the fraction of the population that is HIV-positive thereby giving the fertility-inhibiting effects of the disease more weight at the population level. Counteracting this is the significantly reduced viral load of the HIV

positive population that is brought about by the treatment; resulting in an overall muted impact on the population growth rate. The reason that there is an impact at all is because HIV positive individuals' viral load does eventually begin to increase and they do eventually die – both contributing to lower fertility when women are impacted.

Panel 5 of Figure 1 displays what happens when both types of program are combined. The result appears to be a straightforward combination of the effects of the programs on their own. The overall growth rate for both sexes stabilizes at about 1.5 percent after a sharp increase and then decrease immediately following initiation of the programs, and the all-age prevalence gradually falls to about 23 percent for females and 17 percent for males. These results are the most desirable in all respects. They do not involve very sharp, disruptive changes in the size and composition of the population, the resulting growth rate is acceptable, and the all-age prevalence is coming steadily down. Moreover it is the most humane; some would say ethical, approach because it prevents new HIV infections while giving existing infections the best chance possible of living a full life and fulfilling their obligations.

4.2 Population Age Structure

Figure 2 and Figure 3 present the male and female proportionate age structures by age and years since the HI virus was introduced into the population for the five scenarios described above in Methods. The simulated population without HIV, panel 1 in both figures, has a very stable age distribution for both sexes with a gently curved shape and broad base with about 22 percent of the population in the zero to four age group – indicating a young age distribution that is in line with the relatively high growth rate of four percent per annum experienced by this population. There are no distortions with either age or time in either the female or male age structures.

Panel 2 of both figures displays the age structure of the untreated HIV-infected population over the 40 years of the simulated epidemic. Three significant distortions begin to develop in years eleven through fifteen and progress at a rapid rate through roughly year 35. One, the proportion of the population in the zero to nine age range begins to decline rapidly and eventually stabilize at fifteen to seventeen percent for both sexes. Two, the proportion of the population ten to 29 begins to rise significantly and is still rising at year 40, with typical increases of two to four percent! Three, the proportion of the population 30 to 50 falls substantially with typical decreases of one to two percent. Additionally, the adult-aged distortions are more pronounced for females and begin at slightly younger ages. The end result after forty years of the epidemic, and after the epidemic has largely stabilized, is a "stepped" age structure with a relatively constant *proportion* of people zero to roughly 14, a rapid decline in the *proportion* of people 15 to roughly 39, and then a relatively constant proportion of people 40 to 64 or so, followed by the normal decline as old age mortality begins to take its toll. The reduction in the proportion of very young people is brought about through the fertility-inhibiting effects of HIV infection, while the reduction in the proportion of people at middle and older adult ages is the result of HIV-related mortality. The increase in the proportion of young adults is a simple byproduct of the reductions in numbers and proportions at ages younger and older and the fact that this young adult age group has not yet experienced increases in mortality associated with HIV because they have not yet lived long enough to reach end-stage disease.

Panel 3 of Figure 2 and Figure 3 contains the male and female age structures of the simulated populations infected with HIV and subjected to the behavioral preventative treatment program. They are very similar to the untreated age distributions with one significant difference. The proportion of the population in the zero to nine age range falls precipitously immediately following onset of the treatment program to levels of about ten to 15 percent. The age structure at other ages remains largely as it is in the untreated epidemic with perhaps slightly less vigorous hollowing out of the older adult ages.

Panel 4 shows the effects of the antiretroviral treatment program, and again they are very different. Because there is no fertility inhibiting impact, the proportion zero to nine increases, most dramatically in the zero to four age group – because the treatment is only applied for ten years and does not have time to dramatically impact the five to nine age group. Interestingly the proportion five to 24 actually falls quite sharply. This is because these ages are stuck in limbo between what is happening at younger and older ages. While the number and proportion at younger ages is increasing strongly because fertility is recovering, the number at older ages is at least not decreasing and sometimes slowly increasing due to the deferment of HIV-related mortality at those ages. Throughout the number of individuals living between five and 24 remains roughly constant, especially toward the younger end of that age range, and consequently the proportion in that age group decreases as the number at younger ages increases.

Finally panel 5 displays the effect of combining the two types of treatment program, and as with growth rates, the combined effect is a clear combination of the effects of the two treatment programs applied separately. However in this case the fertility reduction associated with the behavioral preventative treatment program does produce a sufficiently large reduction in the numbers of people zero to nine to substantially reduce the proportion of the population living at those ages, but not nearly as much as is the case in the absence of the antiretroviral treatment program. In addition to partially ameliorating the fertility impact of the behavioral program, the antiretroviral program also preserves middle-age adults and attenuates the hollowing out of that age range.

In light of the larger questions we want to answer relating to the impact of an HIV epidemic on the elderly, the age structure changes displayed here are central. They indicate that the impact of HIV on a population is more subtle than has been recognized, and in particular that the impact on the age structure is perhaps not what has been often mentioned; namely that the dependency ratio will increase dramatically. These results point to the opposite result – that the dependency ratio may actually *decrease* (after possibly increasing transiently) to a stable level (after the epidemic has stabilized) below its pre-HIV level, mainly as a result of the significant reductions in fertility that accompany the epidemic and the fact that the young adult age group will not change much in relative size compared to the size of the whole population. These questions will be examined in some detail below in The Dependency Ratio section.

4.3 Age-Specific Population Sex Ratios

Figure 4 displays the age-specific sex ratio of the simulated populations by the number

of years since HIV was introduced. Panel 1 shows a reasonable age-specific sex ratio for a stable population unaffected by HIV; the sex ratio at very young ages is slightly above 100 and then drops very slowly with age until in the 50 to 54 age group it is between 90 and 100 and then continues to fall slowly. This change with age is brought about by the initial sex ratio at birth having been set at 105 to reflect the typical sex ratio at birth of black populations and then the sex differentials in mortality that progressively remove more males from the population as it ages. The plot in panel 1 reveals a significant amount of random variation in the age-specific sex ratio that reflects the stochastic processes responsible for creating it.

Panel 2 of Figure 4 reveals the effects of HIV on the age-specific sex ratio as an HIV epidemic grows and stabilizes. The most prominent feature is the increase in the sex ratio between ages twenty and 54 that builds steadily starting about twenty years into the epidemic. By year 40 the sex ratio between ages 30 and 49 is between 140 and 150 indicating a surplus of males in that age group of nearly 50 percent! In year 40 the sex ratio at ages 55 to 59 is back down to 100, but then continues to fall to reach levels of roughly 50 by age 65-69, now indicating a deficit of males of roughly 50 percent. These changes are brought about by the strong sex differentials in the age pattern of HIV-related mortality – not shown here. Because the age-specific prevalence for females is significantly younger, and becomes progressively more so as the epidemic stabilizes, females begin dying of HIV-related causes much earlier than males, and consequently compose less and less of the population during middle age. The situation reverses at older ages when HIV-related mortality begins taking a heavy toll on males in addition to the normally higher mortality of males. By these ages, the excess HIV-related mortality of females has largely played out and the number of females decreases at the normal rate with age, while the number of males is decreasing much more quickly than it would in the absence of HIV. This substantial distortion in the age-specific sex ratio has big consequences for the elderly segment of the population – the most obvious being that they will be mostly female. Additionally these surviving females will lose their husbands and male companions in a relatively short age range forcing them to go through major transitions over a short period of time.

Panel 3 contains the effects of the behavioral preventative treatment program starting in year 31 – the lines labeled "31-35" and "36-40" in the plot. As expected, it does not have any particular impact at young ages because there is really no sex differential in HIV-related mortality at those ages, and the impact of this program does not involve mortality anyway. At adult ages, the impact is mild and subtle, mainly a slight attenuation of the increase in the sex ratio across all effected ages. This is the result of fewer infections, but persisting HIV-related mortality which is the main driver of this phenomenon.

Panel 4 shows the effects of the antiretroviral treatment program, and these are more substantial. Because the antiretroviral program has a strong impact on HIV-related mortality (deferring it by about ten years) there is a significant effect on the age-specific sex ratios. Older HIV-infected people have been living with the infection for some time when the treatment campaign starts and consequently their mortality is not deferred as long as younger HIV-infected individual's is. For this reason, there is more impact at younger ages than older ages, and the impact is primarily to delay (with age) the familiar HIV-related changes in the age structure – by about ten years which

is in line with the average delay in HIV-related mortality associated with the simulated antiretroviral treatment program.

Panel 5 displays the effects of the combined treatment program. There is no surprise here – an effect that appears to be about equal parts of the effects of the treatment programs separately. This is slightly disheartening because it suggests there is no real way to avoid or "treat" the HIV-related distortions in the age-specific sex ratio. At the same time it is not surprising because this distortion is primarily caused by the sex-age differentials in incidence of HIV that result in females dying of HIV-related causes at younger ages than males.

4.4 The Dependency Ratio

Results describing changes in the dependency ratio resulting from HIV and the various treatment programs are displayed in three figures: Figure 5, Figure 6 and Figure 7. Figure 5 displays trends in the dependency ratio for both sexes combined along with the proportion (as a percent of total population) of individuals ages zero to fourteen, fifteen to 64 and 65 and older. Figure 6 displays the same trends by sex along with trends in the sex-specific HIV prevalence. Figure 7 displays trends in the sex-specific dependency ratio along with trends in the sex-specific proportion of the population zero to fourteen and fifteen to 64.

Panel 1 of Figure 5 displays trends in the dependency ratio and population composition that impacts on the dependency ratio for the stable population unaffected by HIV. Apart from a small wiggle in the first five years, all of these indices remain constant with a two-sex dependency ratio of about 1.15.

Introducing HIV into this population results in a steady, nearly linear *decrease* in the dependency ratio starting between years ten and fifteen after HIV is introduced, panel 2 of Figure 5. By year 40 after the HIV epidemic has stabilized, the two-sex dependency ratio has fallen to about 0.95 and does not appear to have stabilized yet. Accounting for this are gradual shifts in the proportions of the population in the zero to fourteen and fifteen to 64 age ranges; as the proportion zero to fourteen begins to fall gently from about year twelve after HIV is introduced, the proportion fifteen to 64 begins to rise slowly with no change in the proportion 65 and older. These concurrent, opposite-sign changes in the numerator and denominator of the dependency ratio have the effect of bringing it down considerably. It has often been claimed that HIV will increase the dependency ratio by hollowing out the middle-aged portion of the age structure leaving the young and old unaffected. Clearly in light of the findings here one has to take into account the fertility-inhibiting impact of an HIV epidemic and the fact that the loss of young children may more than offset the loss of adults, resulting in little or, as presented here, negative impacts on the dependency ratio. This is not to say that there are not large impacts on the age structure that will adversely effect the elderly; there are, but they are perhaps more subtle than we have appreciated. Some of these are addressed below.

Panel 3 of Figure 5 presents the effects of introduction the behavioral preventative program in year 31 of the HIV epidemic. The now familiar impact of this program on fertility translates into a very rapid and precipitous decrease in the already reduced two-sex dependency ratio as the proportion of the population in the zero to fourteen age range is dramatically reduced. The two-sex dependency ratio reaches as low as

0.75 in year 40 of this simulation.

Panel 4 of Figure 5 displays the effects of the antiretroviral treatment program. The age structure distortions of this program are described 4.2 above in Population Age Structure. The reduction in the *proportion* of the population five to fourteen overwhelms the increase between zero and four combined with the *increase* in the proportion fifteen to 64, particularly at ages 25 to 44, has the joint effect of slightly reducing the dependency ratio allowing it to stabilize at about 0.9.

Panel 5 displays the combined effects of both of these intervention programs applied together, and it is pretty much what we expect. The two programs both cause reductions in the dependency ratio, and those add up to produce a still larger decrease, leaving the dependency ratio at just over 0.6 in year 40.

The impact of HIV on the dependency ratio is somewhat counterintuitive as are the impacts of both types of treatment program. The combined treatment program that is generally the least disruptive and most beneficial actually produces the sharpest and most disruptive change in the dependency ratio. This reflects the strong reshaping of the population age structure brought about through the HIV epidemic before the treatment programs and the additionally strong impact of the combined treatment programs – the net result being a significant reduction in the proportion of the population living in the zero to fourteen age range.

Now we turn to significant sex differentials that appear in the dependency ratio as HIV begins to effect the population. We will focus mainly on the results presented in Figure 7; Figure 6 presents the same trends in the sex-specific dependency ratio together with the trends in the sex-specific prevalence of HIV between ages fifteen and 64 in order to provide a reference for the stage of the epidemic. It is clear from panel 2 of Figure 6 that the distortions in the dependency ratio for both sexes begin at about the time the HIV epidemic gets going with a prevalence of a few percent.

Panel 1 of Figure 7 demonstrates that in the absence of HIV the sex-specific dependency ratios are constant at about 1.15 for males and 1.10 for females. When HIV is introduced to the population in panel 2 of Figure 7 the female dependency ratio *remains constant* at about 1.1 or slightly less, while the male dependency ratio begins to fall at a substantial rate to a level of less than 0.9 in year 40. Consequently, the decreases observed in the two-sex dependency ratio during an HIV epidemic are driven mainly by decreases in the male dependency ratio. The female dependency ratio stays roughly constant as the HIV epidemic grows because although the age composition of the zero to fourteen and fifteen to 64 age groups change dramatically, the *overall proportion* of the female population in those age groups does *not* change, while the proportion older than 65 changes very little under any circumstance. What happens in the zero to fourteen age range is that the proportion zero to four decreases rapidly at the same time and by about the same amount as the proportion ten to fourteen increases – changes in these two age ranges cancel each other out. A similar thing happens in the fifteen to 64 age range; the proportion fifteen to 24 increases dramatically while the proportion 30 to 59 decreases equally dramatically and by about the same amount.

The male dependency ratio decreases because the proportion zero to fourteen decreases while the proportion fifteen to 64 increases. This is mainly caused by the fact that *male HIV-related mortality occurs at older ages* than female HIV-related

mortality and consequently does not evacuate the fifteen to 64 age group as effectively as it does for females, particularly in the twenty to 34 age range. The result is an increase in the proportion of males fifteen to 64 accompanied by a balancing decrease in the proportion zero to fourteen; all of which results in a steady decline in the male dependency ratio.

Introducing the behavioral preventative treatment program in year 31 has the same impact on the dependency ratio for both females and males; it reduces it strongly by significantly reducing the proportion of the population living in the zero to fourteen age range, panel 3 of Figure 7. This treatment brings the dependency ratio for females down to about 0.75 and for males to about 0.65 by year 40.

The antiretroviral treatment program has a similar but less dramatic effect, also through slightly decreasing the proportion of the population in the zero to fourteen age range. Refer to the discussion under Population Age Structure (Findings) for why the effect of this intervention is more muted and gradually reverses over the course of the ten year intervention.

The combined effect of the two interventions on the sex-specific sex ratio, panel 5 of Figure 7, is to very dramatically reduce the dependency ratios for both females and males in line with what we would expect from the reductions in the two-sex dependency ratio in panel 5 of Figure 5.

The findings relating to the dependency ratio will surprise many observers who have argued that the dependency ratio will increase during an HIV epidemic. The opposite result emerges largely from the reductions in fertility that accompany a substantial HIV epidemic and from the subtle changes in the sex-specific age structure brought about by the sex differentials in the ages at which HIV-related mortality occurs – earlier for females.

4.5 Number of Orphans

Figure 8 and Figure 9 display trends in the numbers and percent of children who are orphans. Figure 8 contains the raw numbers of children and orphans *under the age of fifteen* in order to make clear how the numerator and denominator change in the percent of all children who are orphans. That percent is then displayed in Figure 9.

Panel 1 of Figure 8 demonstrates the steady proportionate growth of the total number of children under age fifteen in the stable population unaffected by HIV. That plot also displays a very small number, in comparison to the total number of children, of maternal and paternal orphans that also grows very slowly over the course of 40 years. The same plot in Figure 9 indicates that the percentage of children under the age of fifteen who are maternal, paternal and dual orphans remains constant in the population unaffected by HIV, and importantly that the percentage of children who are dual orphans is literally negligible in the HIV-free population. There are always a few more paternal orphans resulting from the slightly higher adult mortality of males.

We turn now to the second panel in each figure that displays the same numbers for the untreated population with HIV. Added to these plots is the trend in the adult (both sexes ages fifteen to 49) prevalence of HIV. Panel 2 of Figure 8 makes clear the fertility-inhibiting impact of an HIV epidemic that we have mentioned numerous times above; compared to panel 1, the total number of children zero to fifteen is significantly reduced, and this reduction begins to be felt between ten and fifteen years after HIV is

introduced into the population. About twenty years after HIV is introduced the number of orphans begins to rise significantly. Interestingly the number of orphans begins to increase about ten to fifteen years after the adult prevalence begins its rise and the number of orphans does not reach significant levels until HIV has been effecting the population for at least twenty years – a significant lag associated with the average time between infection and death of roughly ten years for adults. Given that most HIV epidemics in sub-Saharan Africa are less than twenty years old, big increases in the numbers of orphans may yet be coming. Looking at panel 2 of Figure 9 reveals the stunning fact that 37 percent of all children under age fifteen are maternal orphans by year 40, 30 percent are paternal orphans and *22 percent are dual orphans*. Compare this number to the figure of roughly zero percent in panel 1 of Figure 9. The fifteen year (or so) lag is very apparent in this plot when comparing the trends in the percent children who are orphans and adult prevalence. The dramatic increase in the percentage children who are orphans is driven both by the falling number of children and by the rising number of those who are orphans; working together these two movements create a large fraction of children who are orphaned.

Examining the impacts of the behavioral preventative treatment program in panel 3 of Figure 8 and Figure 9 reveals the familiar sharp drop in adult prevalence induced by the treatment accompanied by a strong reduction in the total number of children zero to fifteen but no big drop in the number of orphans until the latest years of the simulation, 38 through 40. This results from the fact that adult HIV-related mortality is unaffected by this treatment, and hence parents of existing children keep dying as they did until near the end of the ten year period of the treatment when the reservoir of HIV infected adults begins to thin and HIV-related mortality of adults begins to abate. Panel 3 of Figure 9 makes clear that there is a sharp downturn in the percentage children who are orphans toward the end of the treatment period.

The antiretroviral treatment program has the opposite effect on orphanhood. Immediately after the treatment begins the total number of children begins to climb again as the HIV-mediated reduction in fertility is ameliorated, and concurrently the numbers of orphans begins to decline because the treatment is deferring HIV-related adult mortality to older adult ages that are attained after the children have lived to age fifteen, and existing orphans age out of the zero to fifteen age range, panel 4 Figure 8. These changes translate into a very sharp decline in the percentage of children who are orphans, panel 4 Figure 9 that softens and flattens out toward the end of the treatment period as the system begins to stabilize.

Combining the two treatment programs produces a result similar to the antiretroviral treatment program, except accompanied by a clear decrease in the total number of children and in adult HIV prevalence.

Clearly HIV-induced changes in the numbers and percentage children who are orphaned are very significant and will produce substantial intergenerational tensions in the population, in addition to the necessity to reallocate resources to the caregivers of these orphans. Now we examine this same question from the point of view of those caregivers. In this case adults aged 50 and over.

4.6 Numbers of Children and Grandchildren

Figure 10 describes the number and percentage of adults aged 50 and older who have

surviving grandchildren *less than 15 years of age* but no surviving children. Panel 1 indicates that in a population unaffected by HIV, the number of adults 50 and older rises at a constant proportionate rate with slightly fewer males than females – a natural result of male adult mortality exceeding that of females. The number and percentage of older people of either sex who have surviving grandchildren without surviving children is negligible and constant.

When HIV is introduced to the population in panel 2 the situation changes; about twenty to 25 years into the epidemic – ten years or so after prevalence begins rising – the number of adults 50 and older plateaus and immediately starts declining. At the same time the number and percentage of adults 50 and older with surviving grandchildren but no surviving children begins to rise. Because the total number of adults 50 and older is declining while the number with surviving grandchildren but no surviving children is rising, the percentage with surviving grandchildren but no surviving children rises very quickly. Roughly 22 percent of females 50 and older and roughly 12 percent of males are in this category by year 40. The discrepancy between the female and male percentages likely results from the fact the fact that women can only have children during the years when they are fertile while men can have children over a much wider range of ages. This results in an oldest age at which women (as a population) can reproduce, and as a consequence they cannot replenish their pool of children as they age and therefore more quickly lose *all* of their children than men who always have the potential to have another child and thereby avoid losing *all* of their children. In this way men are able to maintain their pool of children longer than women in the face of a force of mortality, such as HIV, that gradually kills their children.

Introducing the behavioral preventative treatment program in panel 3 has little impact because most of the transmission is happening at much younger ages that do not impact on the total number of adults 50 and older during the comparatively short period of ten years over which the treatment is applied. The existing HIV positive population at the time the treatment started continues to die as it did over the ten years of the treatment thereby continuing to create the orphans that contribute to the percentage of adults 50 and over who have surviving grandchildren but no surviving children, see panel 3 of Figure 8.

In contrast, the antiretroviral treatment program has an immediate and very positive impact on this indicator, panel 4 of Figure 10. The number of adults 50 and older stabilizes because HIV-related adult mortality is deferred, and the number of adults 50 and older with surviving grandchildren but no surviving children also stabilizes for the same reason. The net result is a stabilization of the percentage adults 50 and older with surviving grandchildren but no surviving children at a level of about 9 percent for females and 4 percent for males.

The combined treatment program produces a result almost identical to the antiretroviral program – as expected given the fact that the behavioral preventative program had little effect.

HIV-mediated changes in the number and percentage of adults 50 and older who are in a position to be required to care for young children are very significant and display a strong sex differential. By the time a vigorous HIV epidemic stabilizes, up to one quarter of women 50 and older may have surviving orphaned grandchildren who they must care for, while up to 15 percent of older men may be in the same position. The

40 years simulated here is not sufficient to see these indices stabilize; they are still rising steadily in year 40. Given the twenty to 25 year lag between the time HIV is introduced to the population and the beginning of the rise in this indicator, the future is ominous for those who will be 50 and older in sub-Saharan Africa over the next ten to twenty years.

5 Conclusions

Taken as a whole the findings presented here paint a nuanced picture of the impact of HIV on a population, and in particular of the consequences for older people. Some generally held beliefs about those impacts are questioned while it is clearly demonstrated that there will be substantial impacts. The most prominent of these is the assertion that the dependency ratio will rise. Contrarily, results presented here indicate that through subtle changes in the age structure, driven by both changes in fertility and mortality, the dependency ratio is actually likely to decline rather than rise, and that furthermore the decline is largely driven by changes in the male age structure. Even though the dependency ratio is likely to hold steady or fall, big changes are wrought on the age structure resulting in a stable age structure with a "stepped" shape after the epidemic has stabilized that is likely to be younger than the pre-HIV age structure. Because of the sex differential in age-specific incidence and prevalence, females die of HIV-related causes at younger ages than males leading to severe distortions in the age-specific sex ratio that result in a substantial predominance of females living at older ages in HIV infected populations. And overall the population growth rate is likely to fall substantially to the vicinity of zero percent per annum. The average time from infection to death of roughly ten years appears in many guises in the results presented here – mainly the typical duration of the lag between the initial rise in prevalence and the appearance of some other effect, such as the creation of excess orphans.

Perhaps the most salient results with respect to the older segment of an HIV infected population have to do with the creation of a large number of orphans of all three types – maternal, paternal *and dual.* The situation grows even grimmer when viewed from the point of view of the elderly themselves with the result that nearly 25 percent of women older than 50 live with surviving grandchildren but no surviving children after 40 years of an HIV epidemic. Taken together with the impact of HIV on the age-specific sex ratio which after 40 years of an epidemic results in two thirds of the population 50 and older being female, the final result is that fully one fifth of the total population over 50 years of age will be in a position of obligation to care for grandchildren. Acknowledging that each grandchild has two grandmothers, the entire burden will be diluted slightly, but the fact remains that a substantial fraction of older people, who will be largely women under any circumstance, will be caring for young children.

The impacts of the behavioral preventative and antiretroviral treatment programs are varied and often work in opposite directions. The most important impacts of the prevention program have to do with its effect on fertility, the fact that it can substantially reduce fertility. This feeds back through the age structure to affect other processes in the population and the indicators that we use to measure the impact of

HIV, such as the dependency ratio. The fertility impacts of the preventative programs are felt immediately while the drop in incidence does not work through to have an impact on HIV-related mortality for about ten years which produces a lag in the mortality impact; and because the treatment programs were only simulated for ten years, these mortality impacts were largely not evident.

In comparison the antiretroviral programs have little effect on fertility, and what little they do increases rather than decreases fertility. In contrast, they have an immediate impact on mortality by deferring it for an average of ten years or so in adults. These mortality reduction impacts were evident immediately and often led to positive changes in the trend of the epidemic. However, it is important to note that these changes are likely to be temporary and result from the immediate deferment of mortality. Again because the simulated treatment only lasted ten years, there was not sufficient time to see the rebound of HIV-related mortality as the deferment expired and HIV-related mortality began to reassert itself at a new level slightly less – because of the reduction in incidence brought about by the suppression of viral load and lowered transmission probability – than what had been before the treatment began.

In general it appears that some mixture of the two types of treatment program is best; the dual approach often leads to a smoother result with less dramatic short term changes, and often the two compliment each other.

An important next step in this investigation is to restart all of the fifteen simulations whose results are presented here and simulate another 40 years. This will provide ample time for the perturbations wrought by the treatment programs to stabilize and for the epidemics to reach new equilibria. In general there appear to be two important time scales in the temporal evolution of all of these epidemics. The first has to do with the latency between infection and death – about ten years for adults. The second has to do with fertility, the latency between a change in fertility and the time when the children who were born at the time of that change reach reproductive ages themselves, or about one generation. This is the period over which the perturbations in fertility reverberate through the age structure, after about one generation the fluctuations in the age structure are significantly reduced in magnitude, but at least one to two more generations are required for the echoes of a fertility perturbation to completely disappear.

The most important impression provided by the findings presented here is that the impacts of HIV on a population are subtle and multifaceted and *must be understood together* in the context of their joint impact on a whole population. Ignoring one or more of the major impacts results in a distorted view of the other impacts. It is also clear that the timescales over which an epidemic grows and treatments have their impacts are on the order of decades or quarter centuries, and that many will echo through the age structure for on the order of 50 to 100 years. These long time scales are important in motivating urgency in treatment but also cautioning patience after a program is instituted because it may take significant time for the effects to be strongly felt and even longer for a new equilibrium to be reached.

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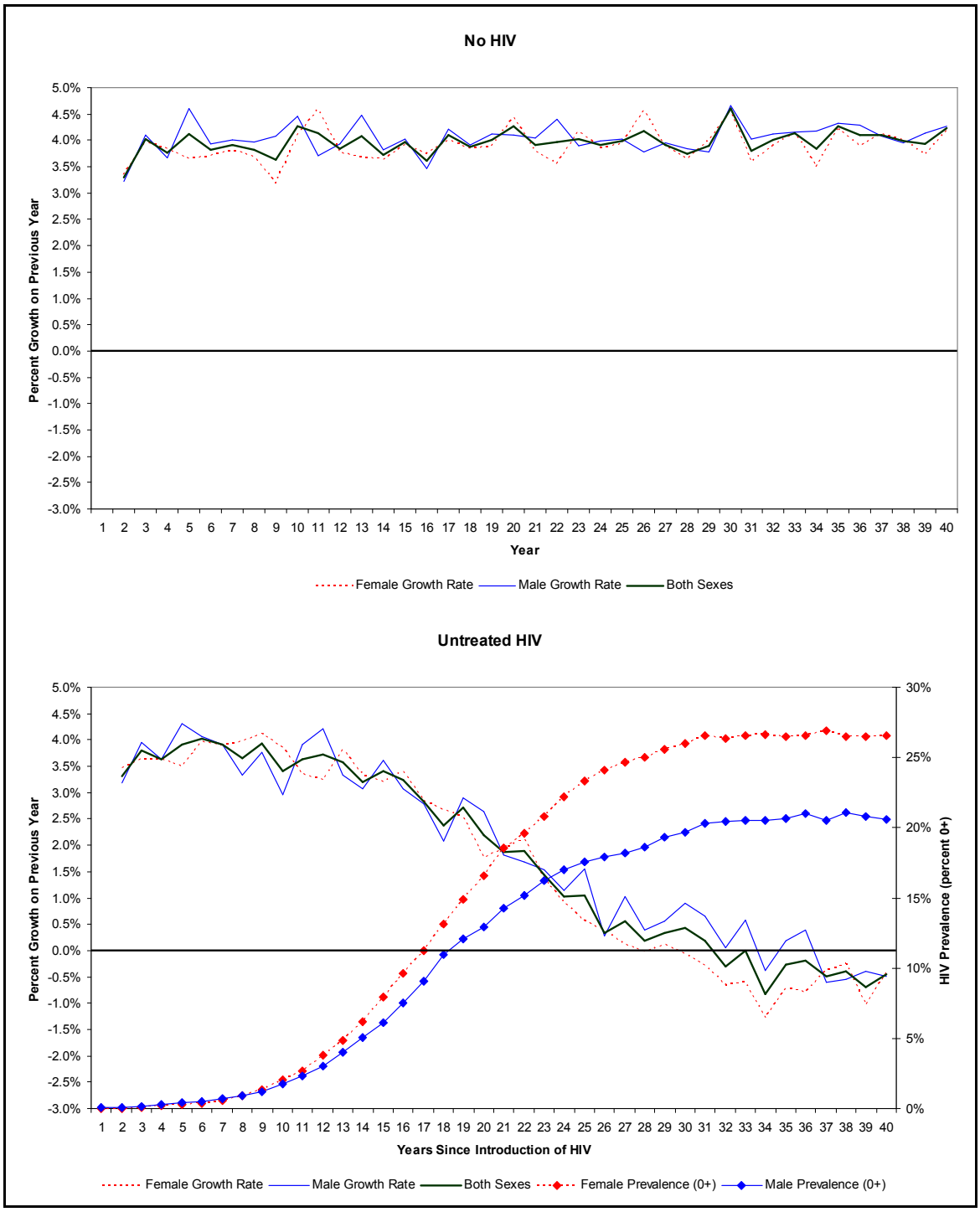

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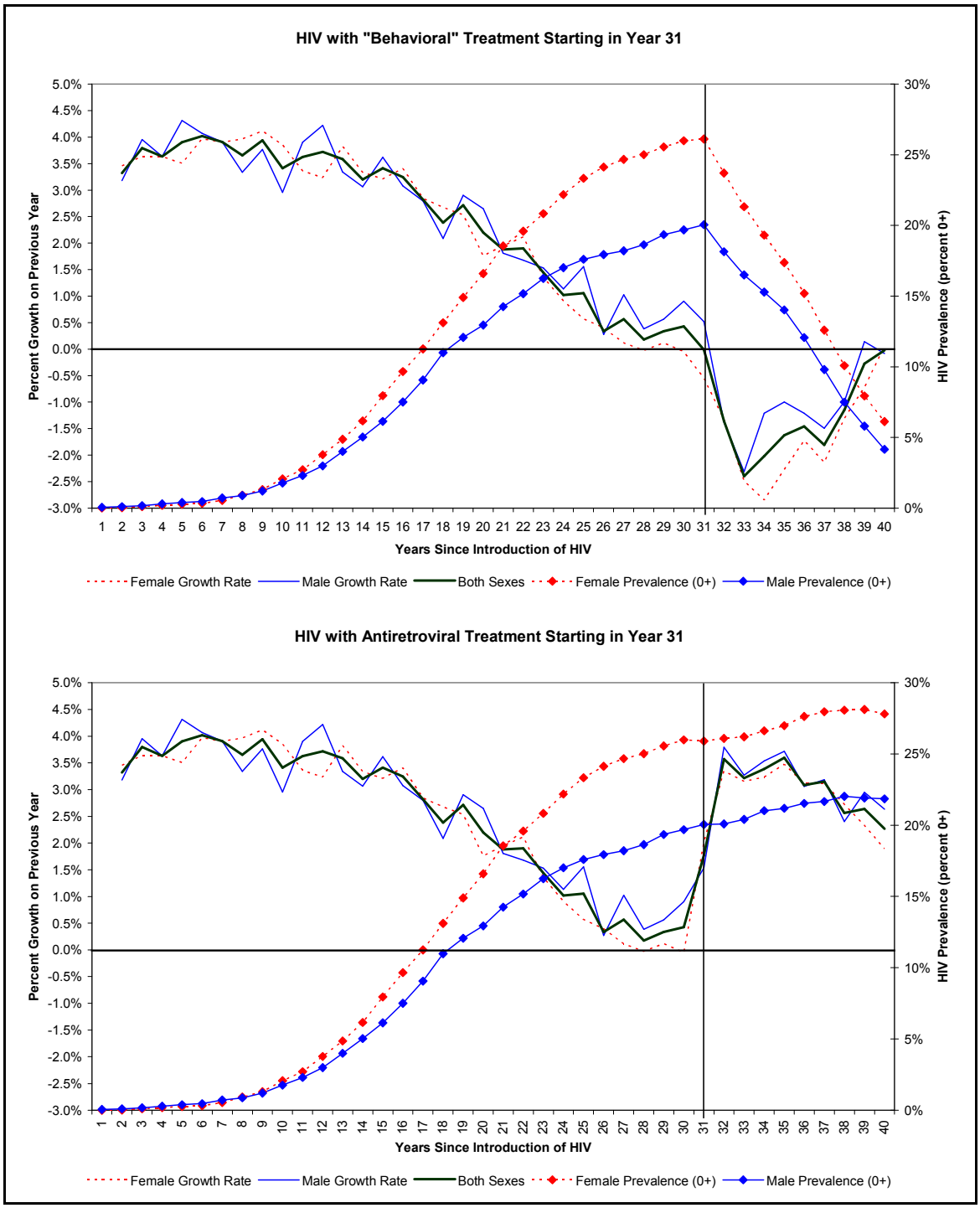

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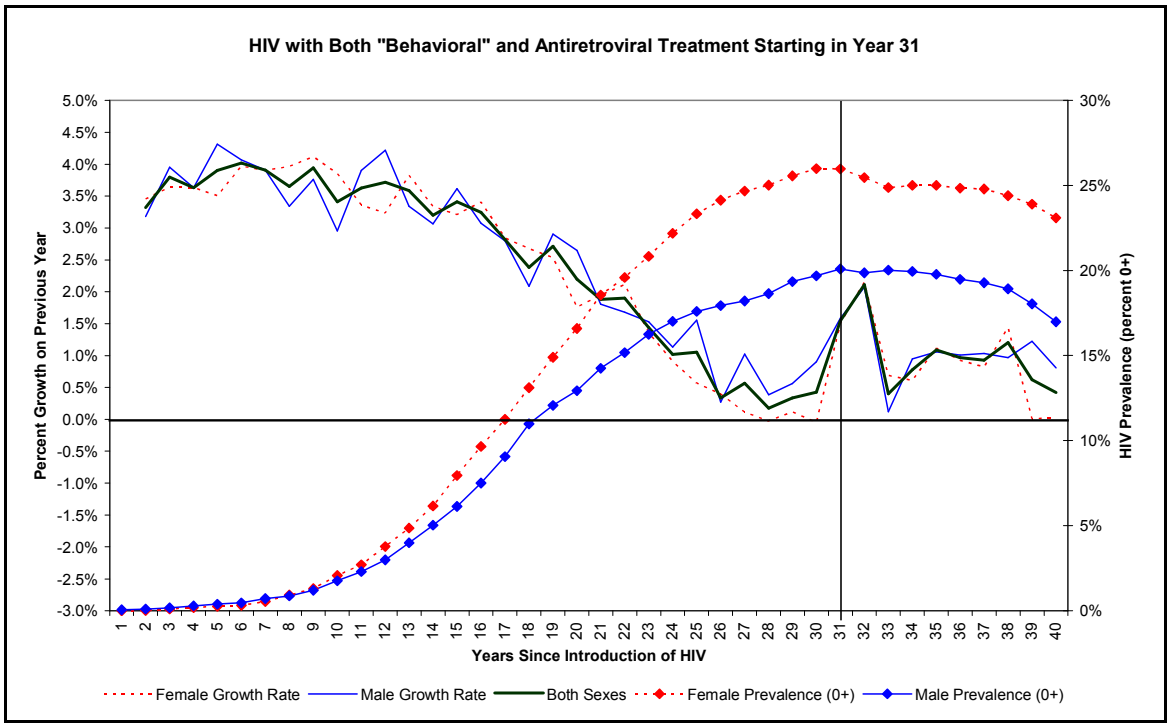
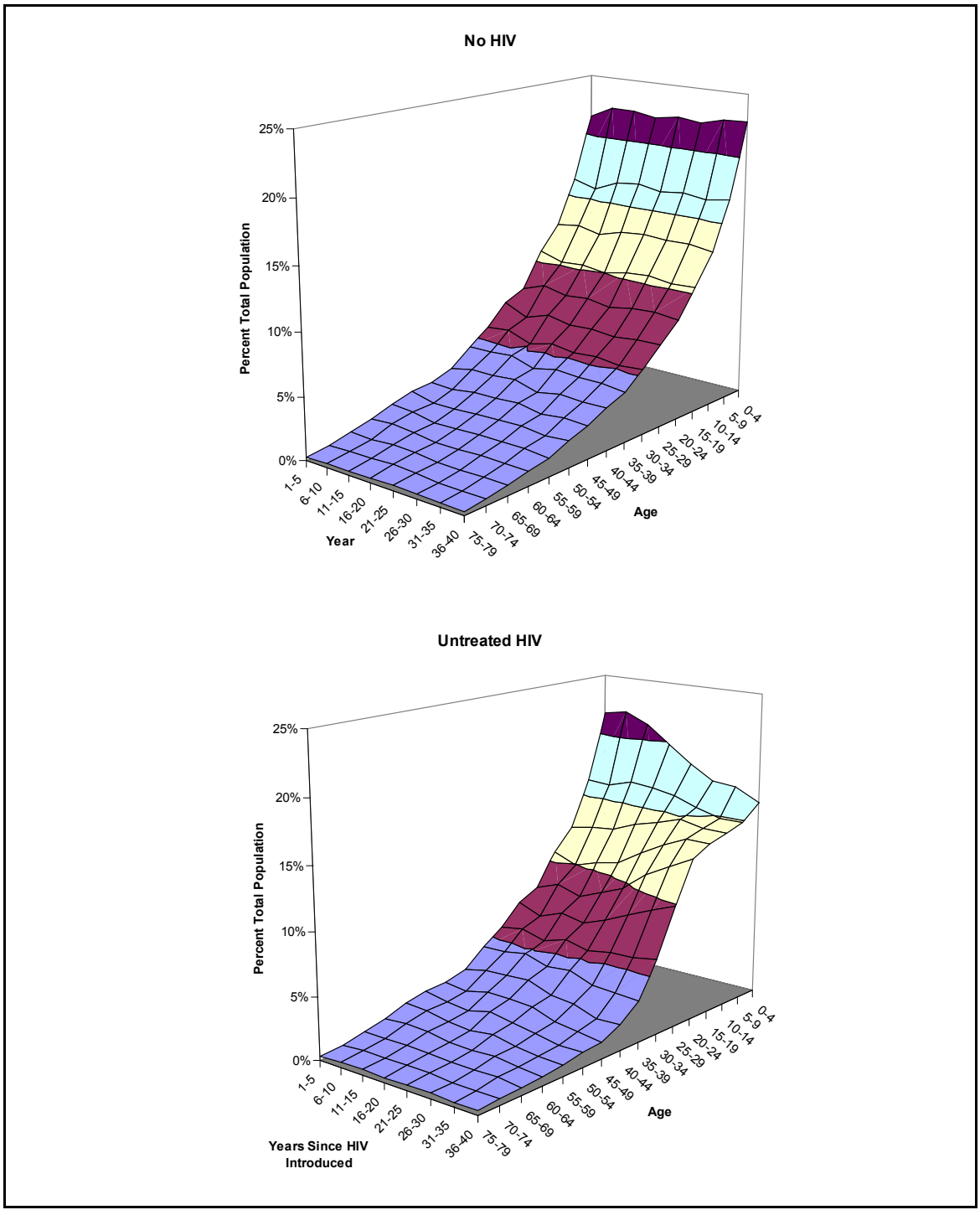


Figure 2: Male Proportional Age Structure



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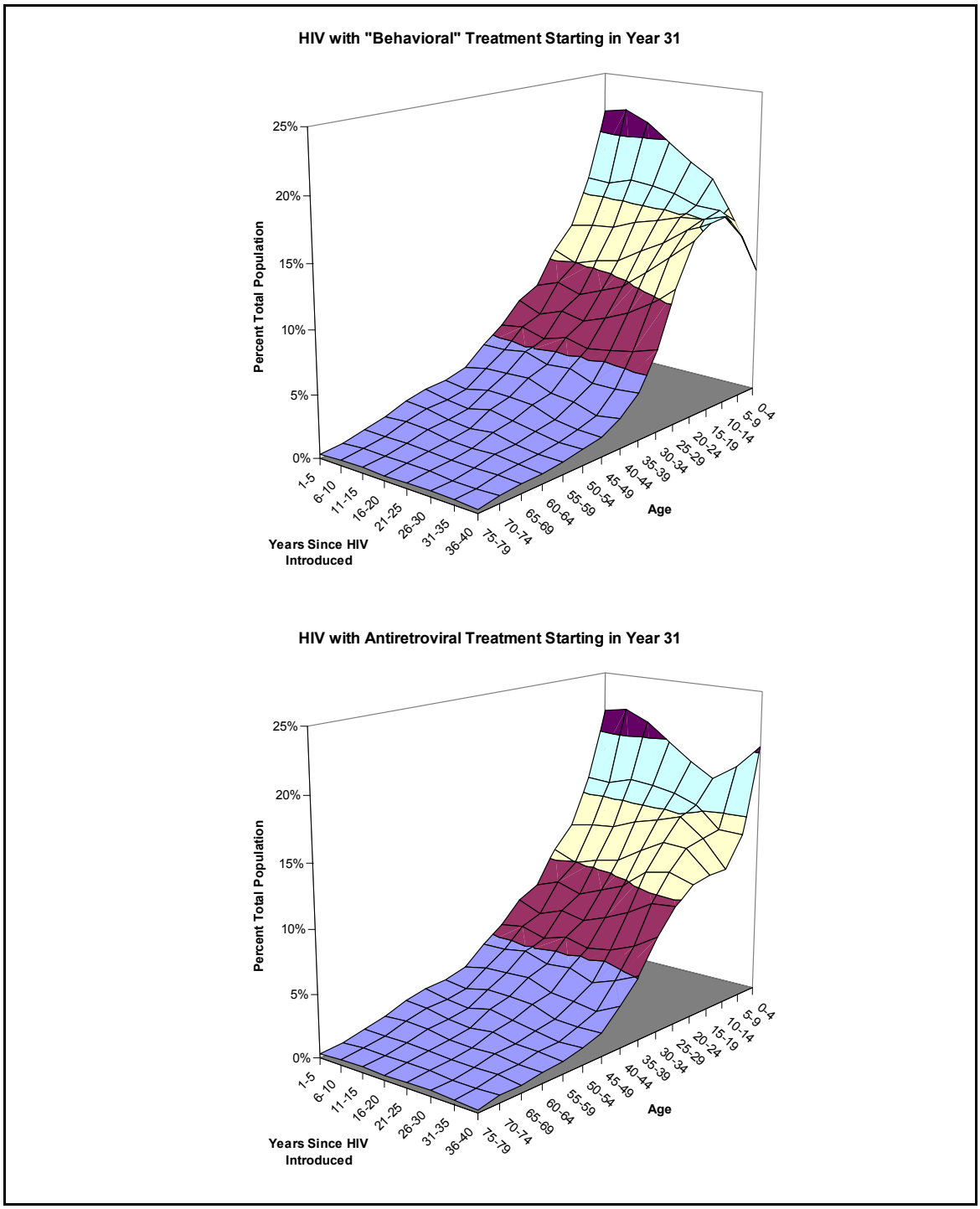

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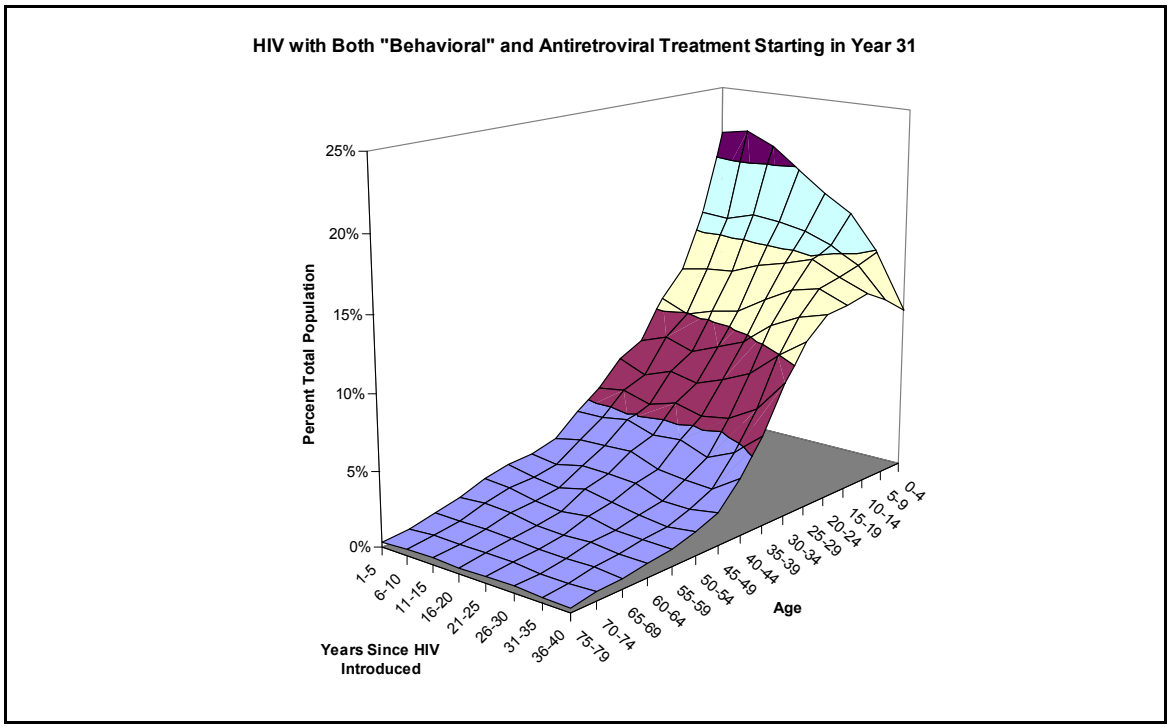


Figure 3: Female Proportional Age Structure

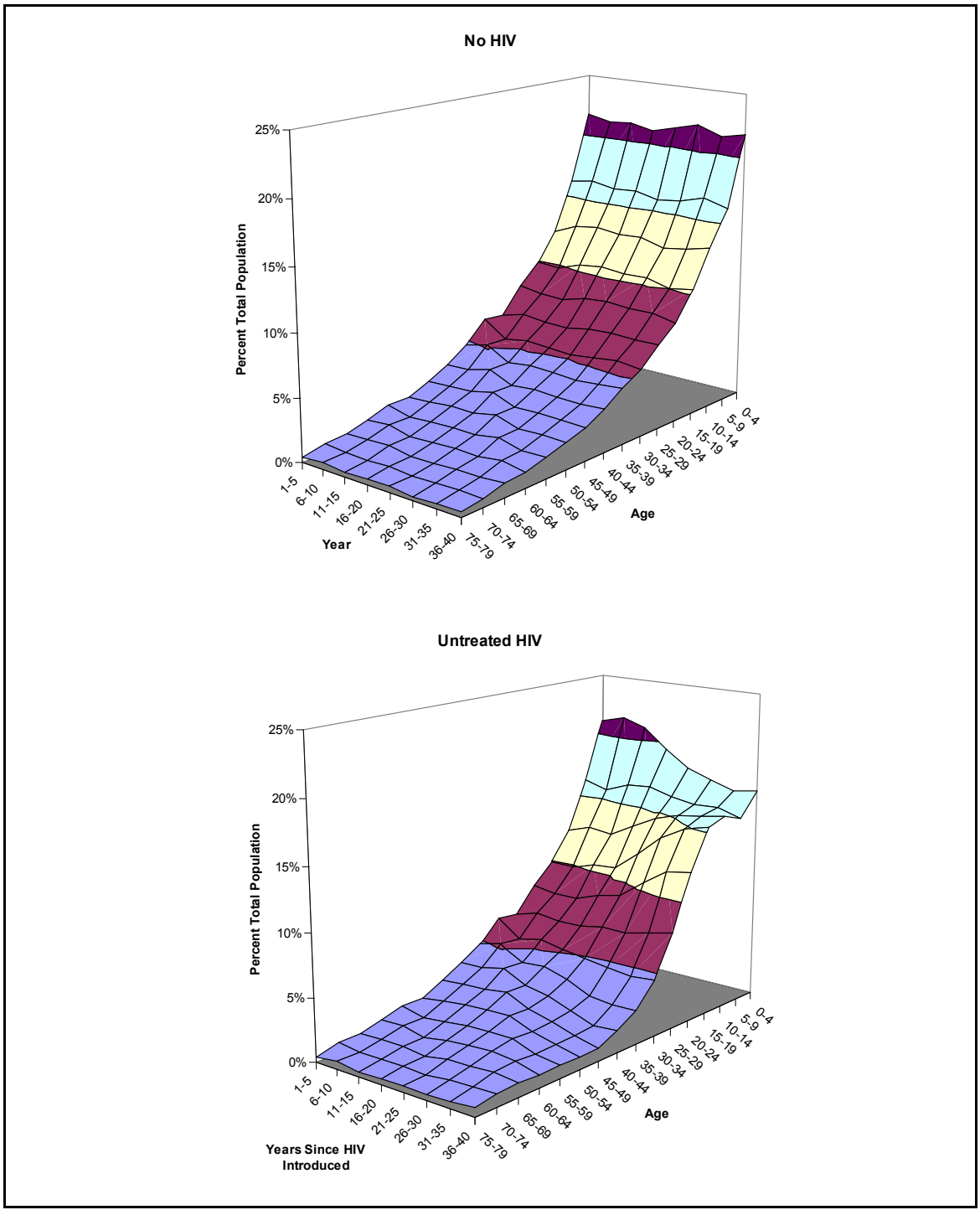
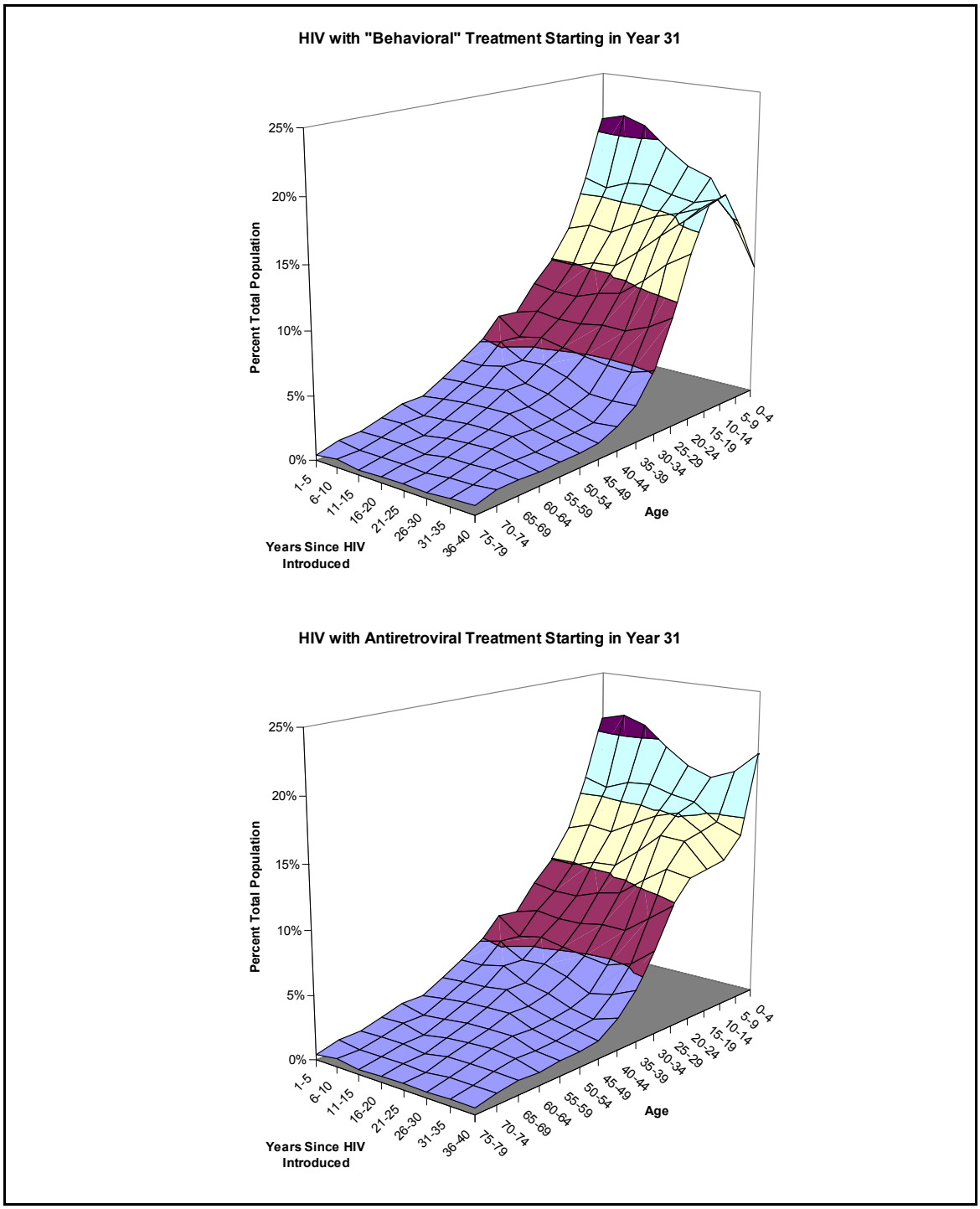


Figure 3: Female Proportional Age Structure



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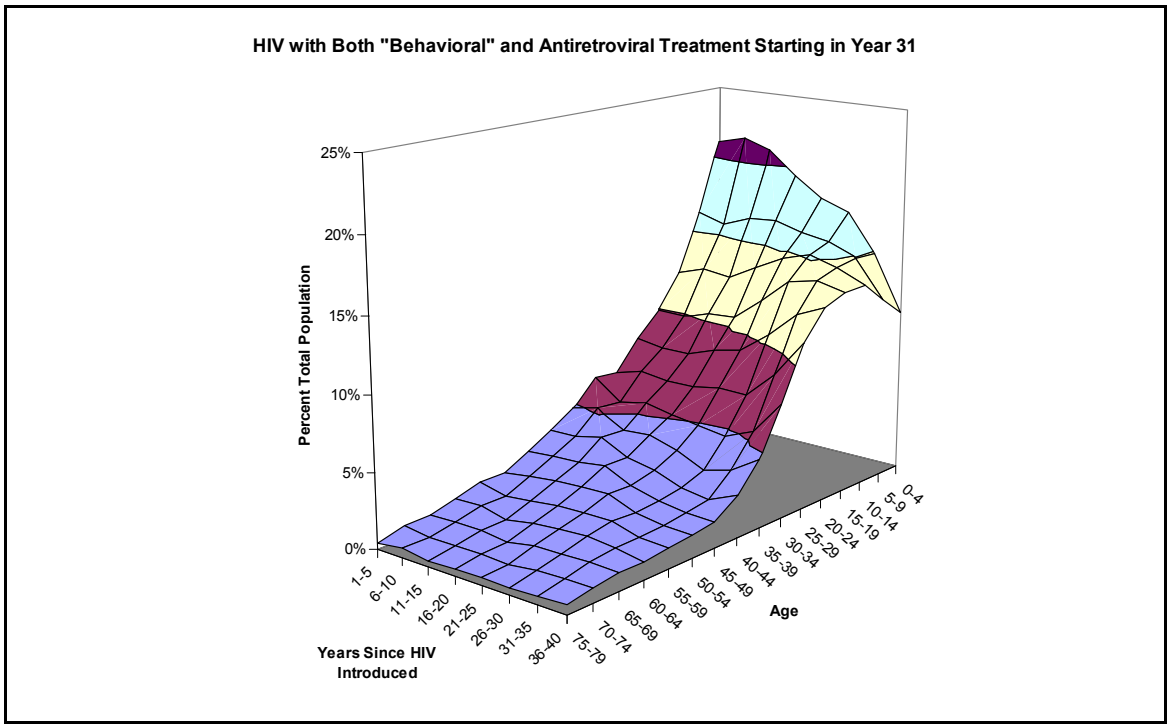
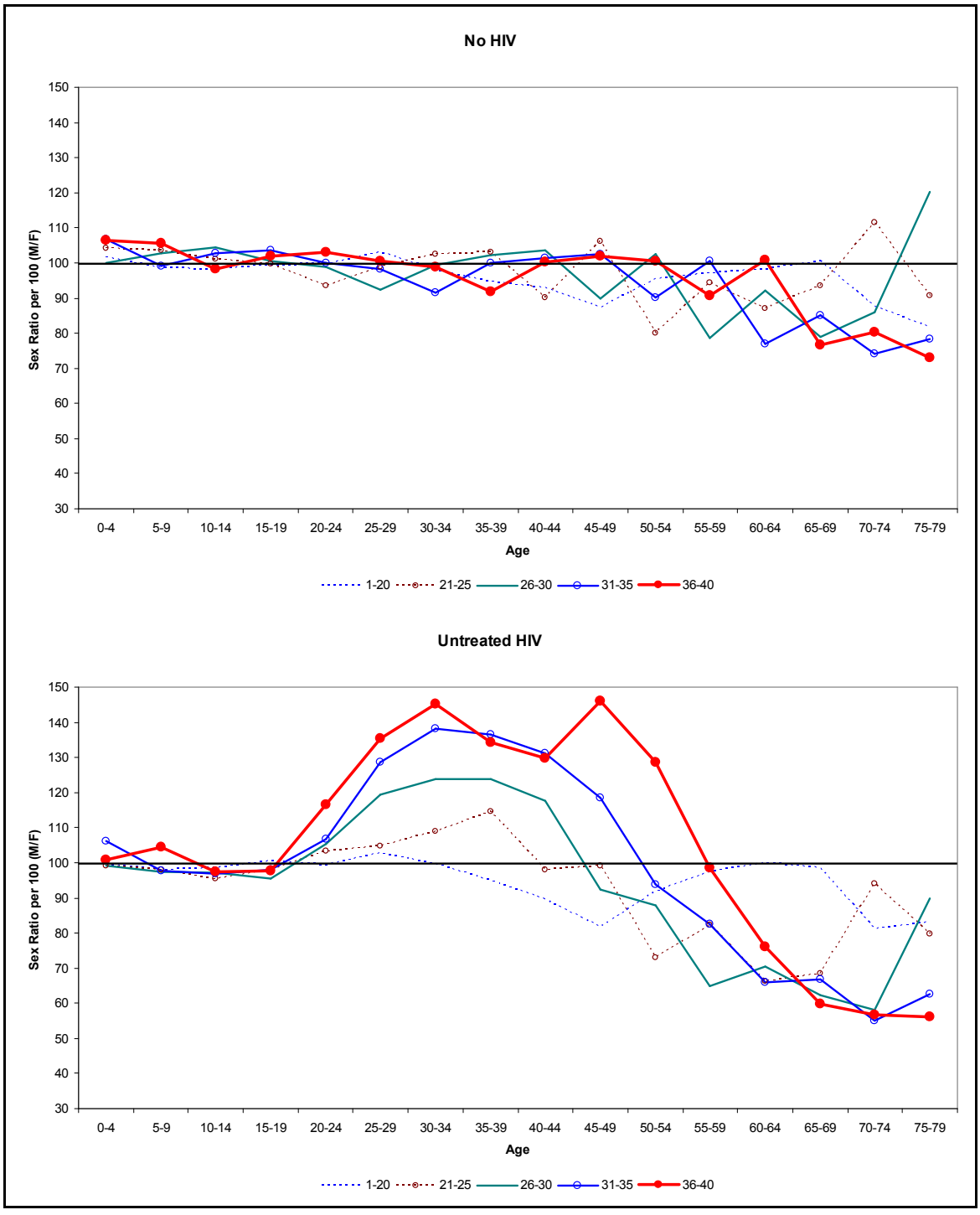


Figure 4: Age-Specific Sex Ratio by Years Since HIV Introduced



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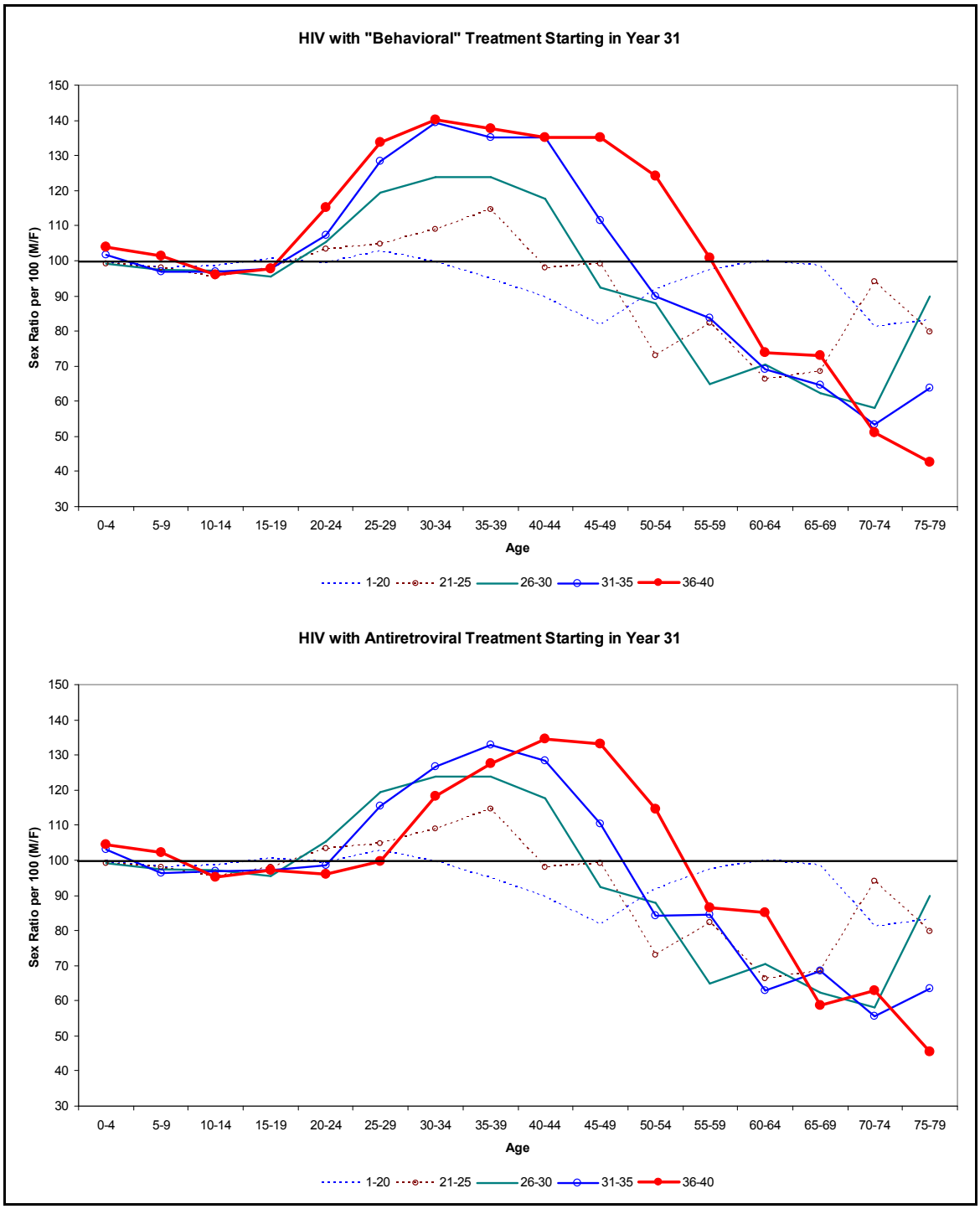

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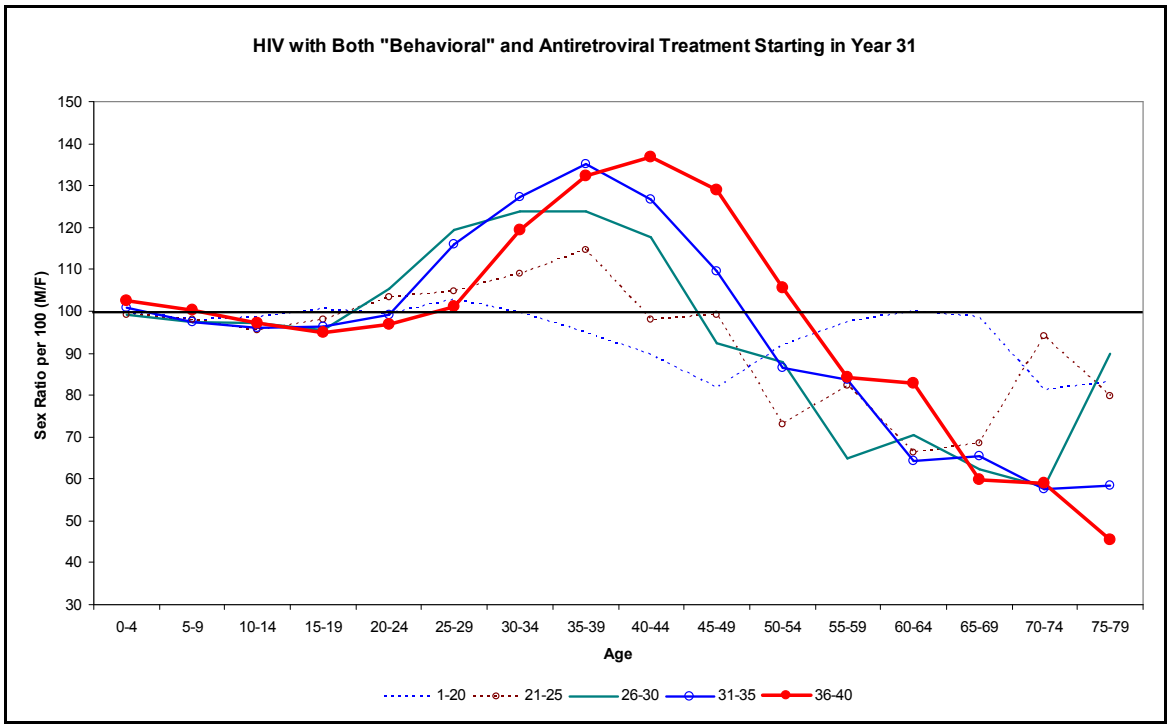

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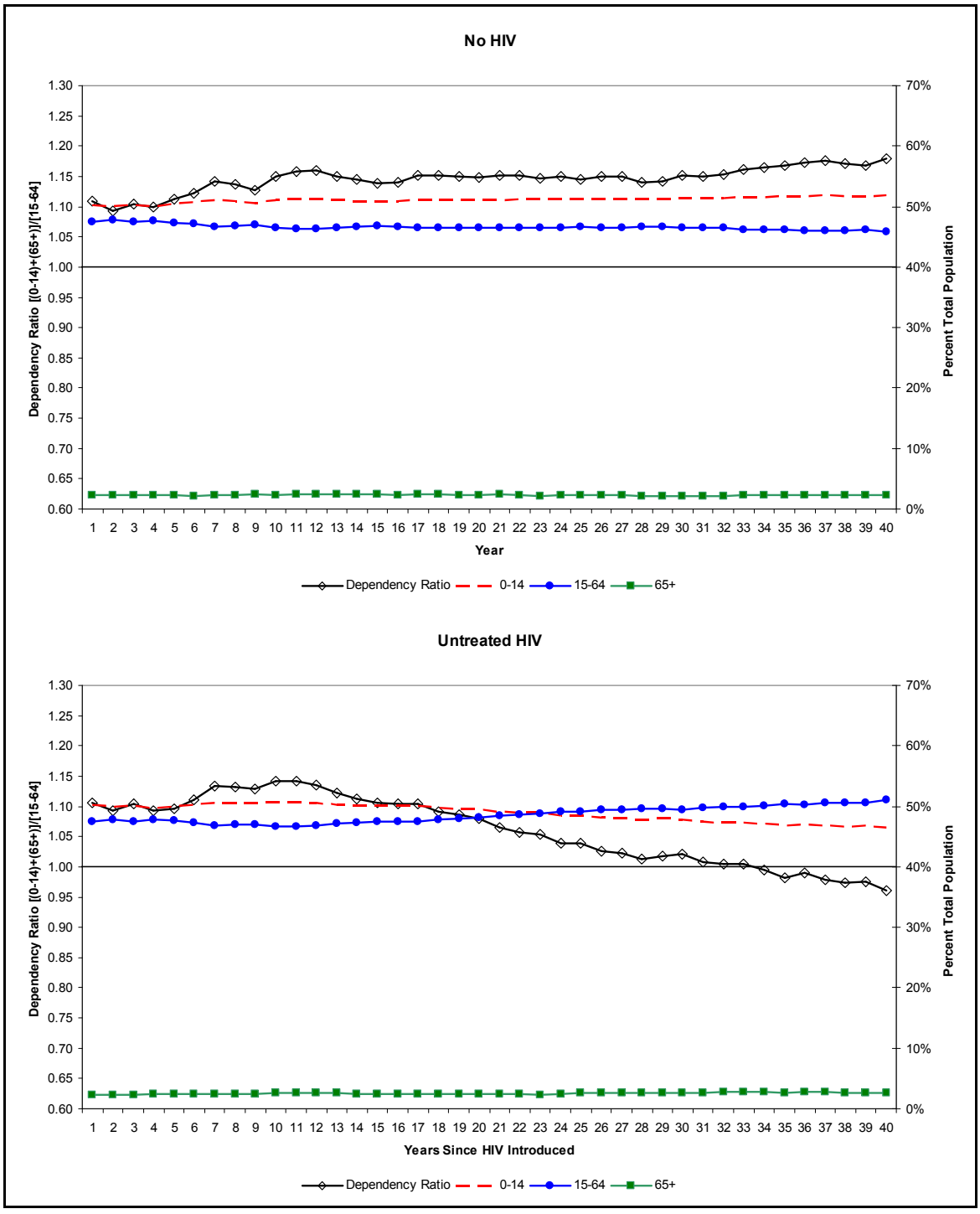

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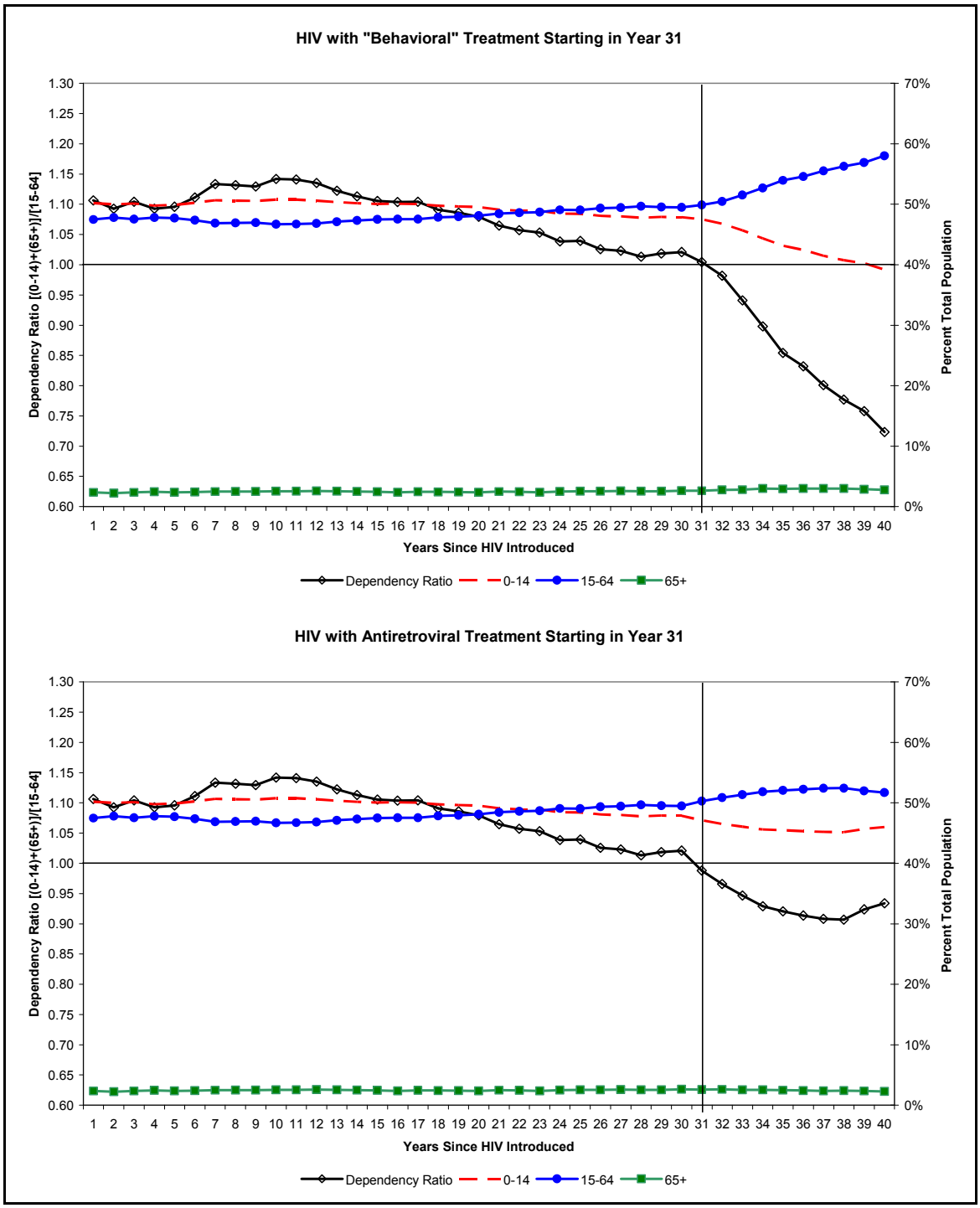

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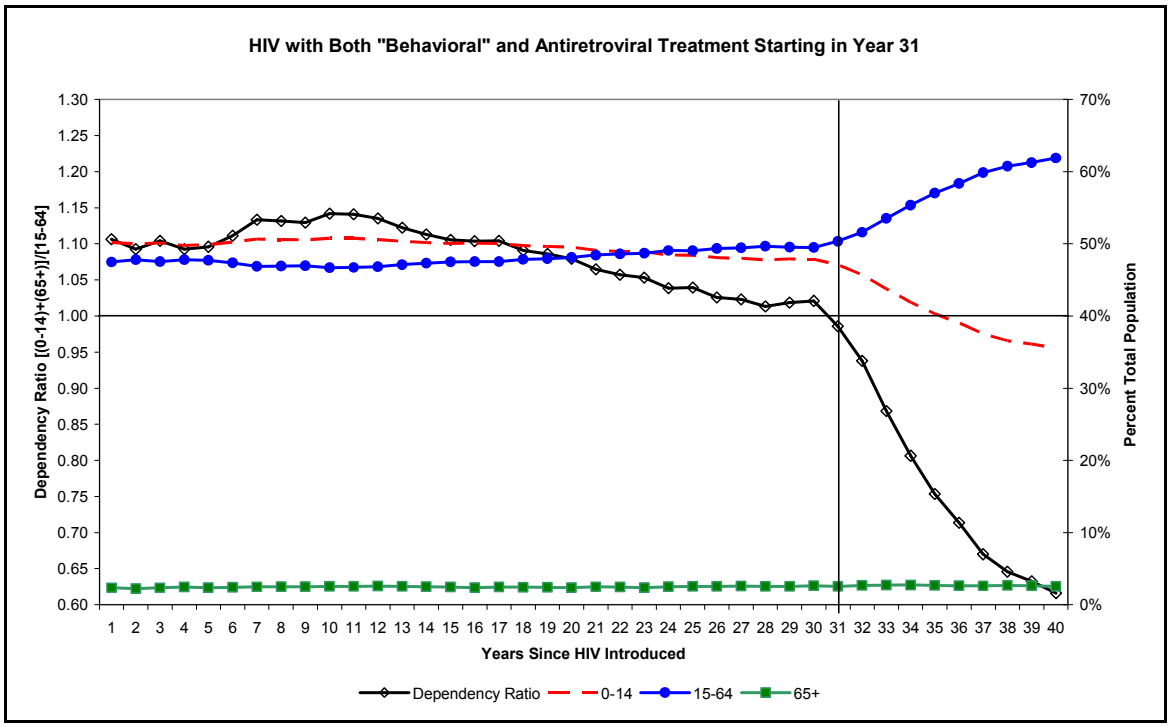

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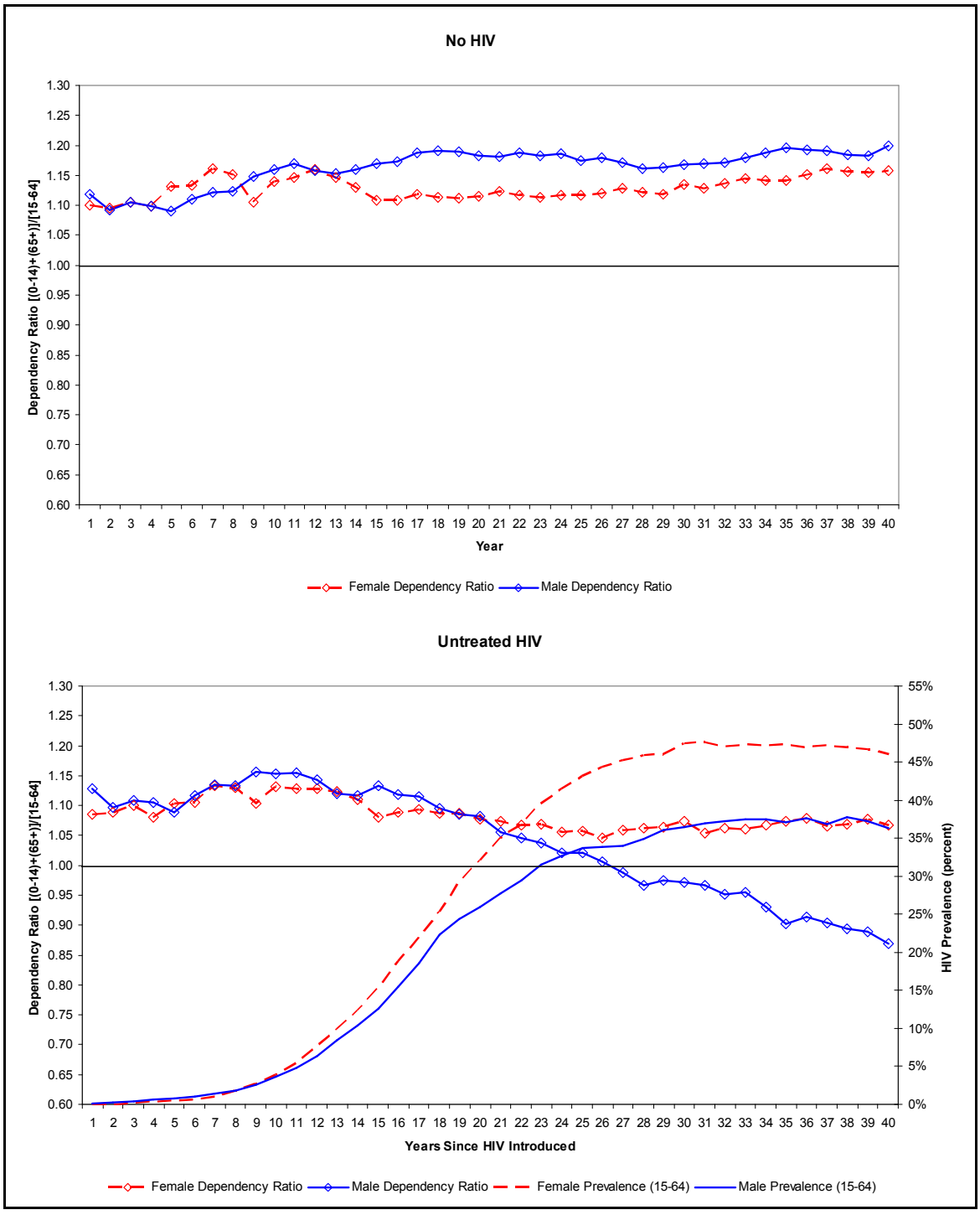

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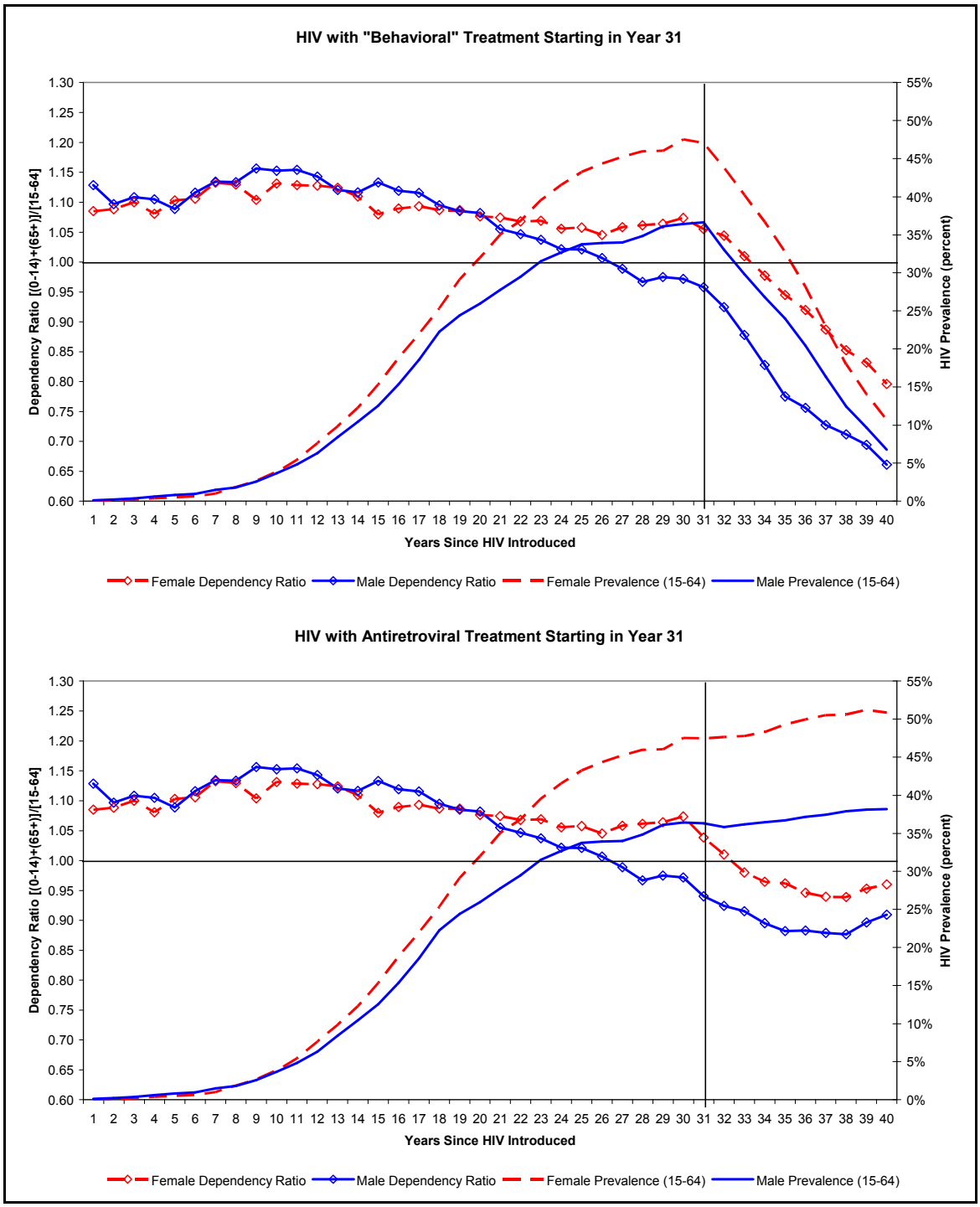

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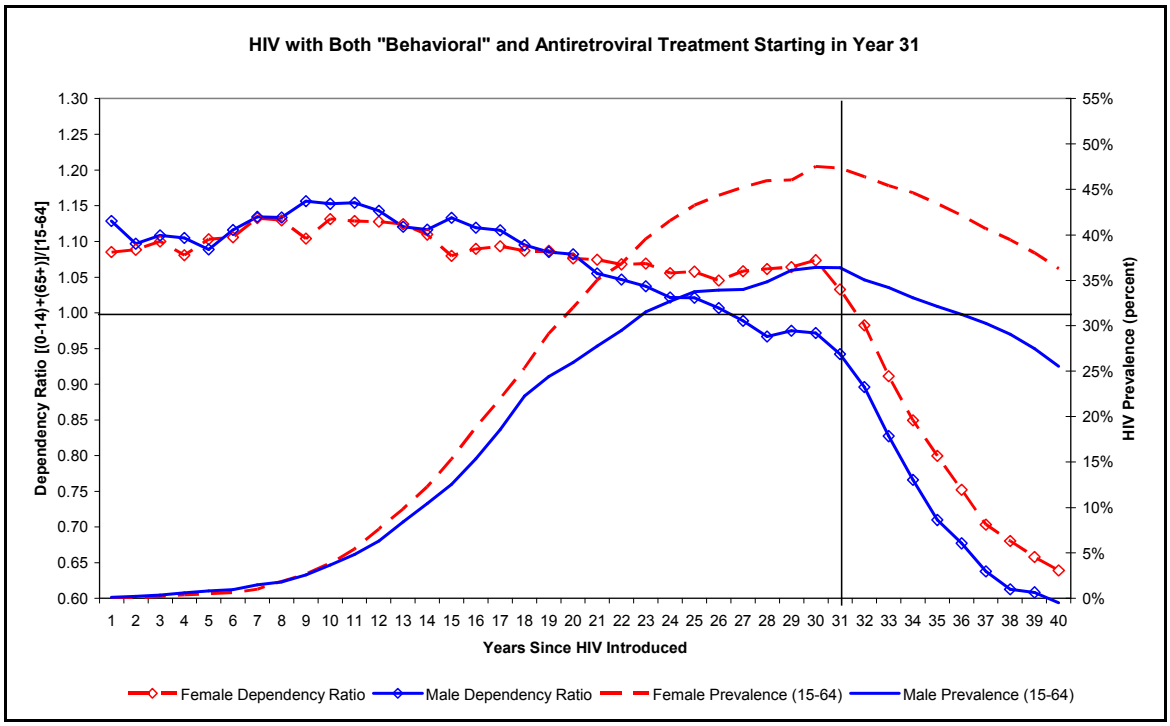
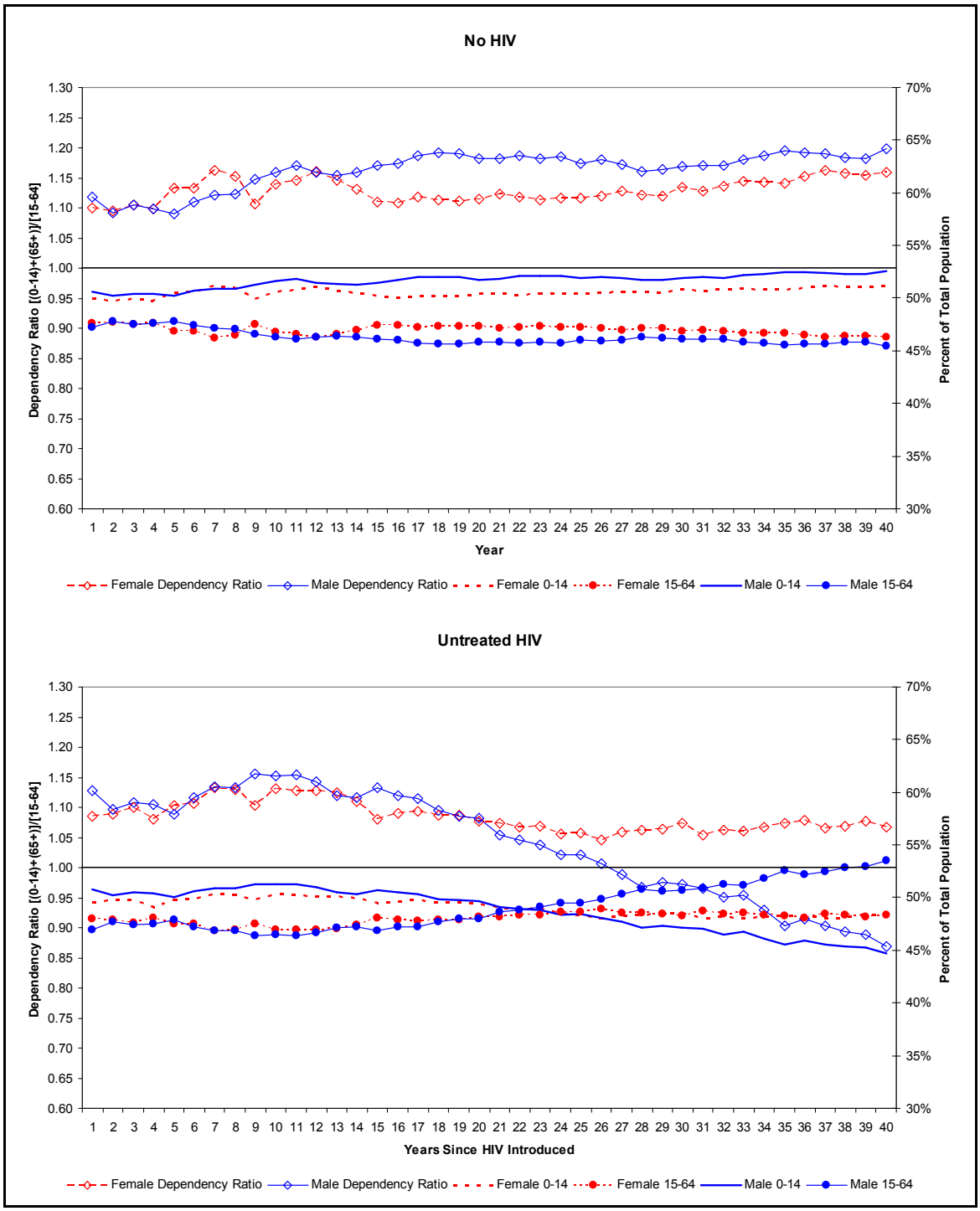


Figure 7: Trends in Dependency Ratio and Percent Children, Working-Age Adults and Adults Aged 50+



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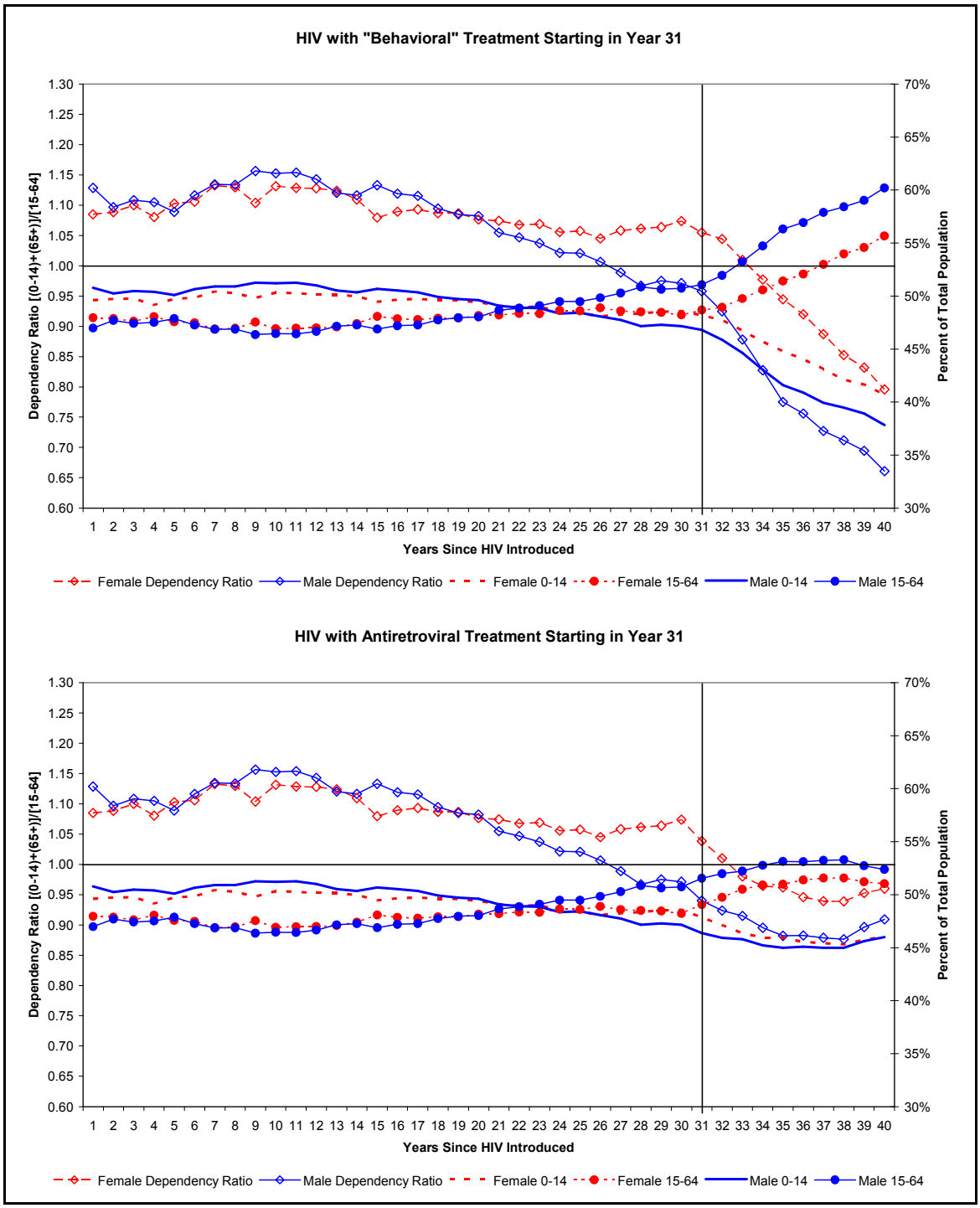

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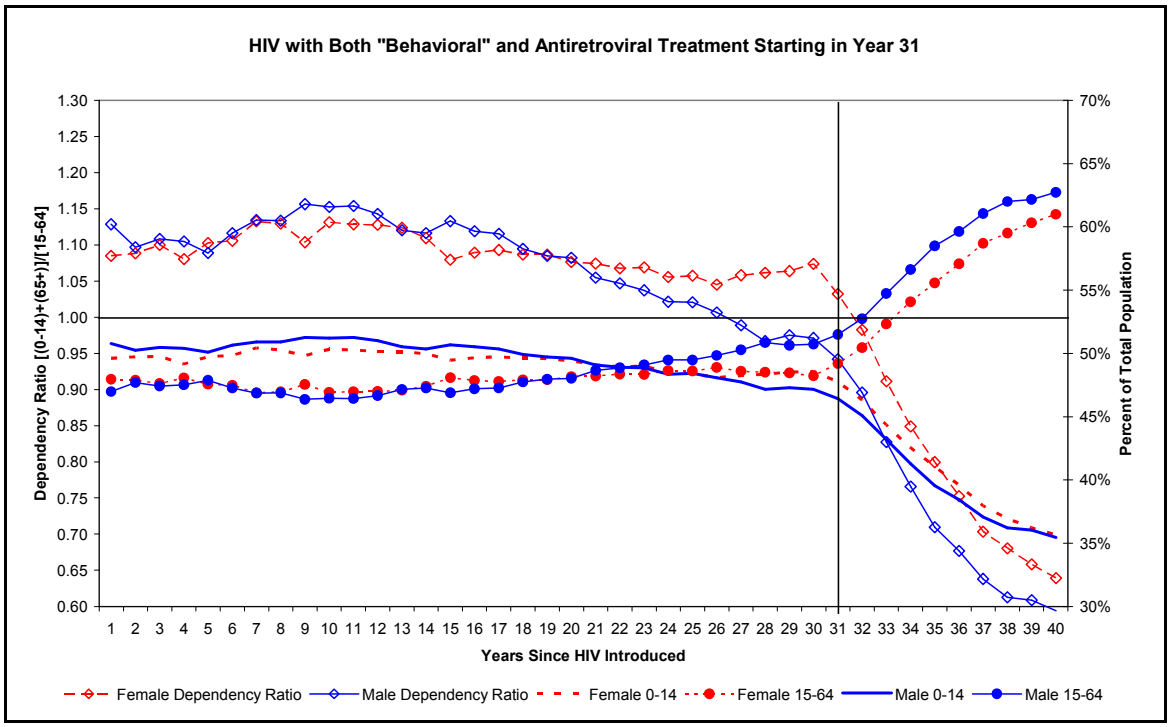
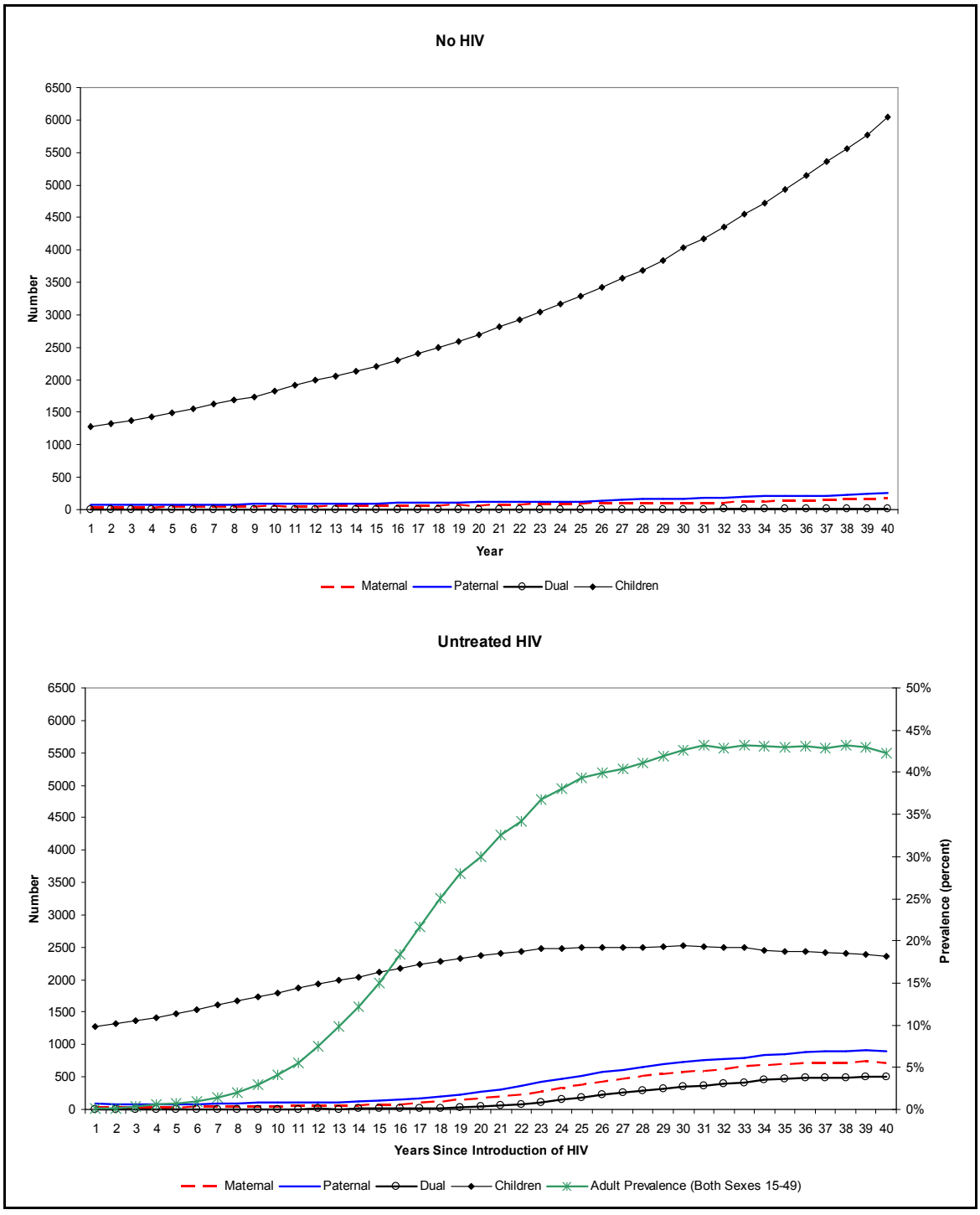


Figure 8: Trends in Number of Orphans and Adult Prevalence



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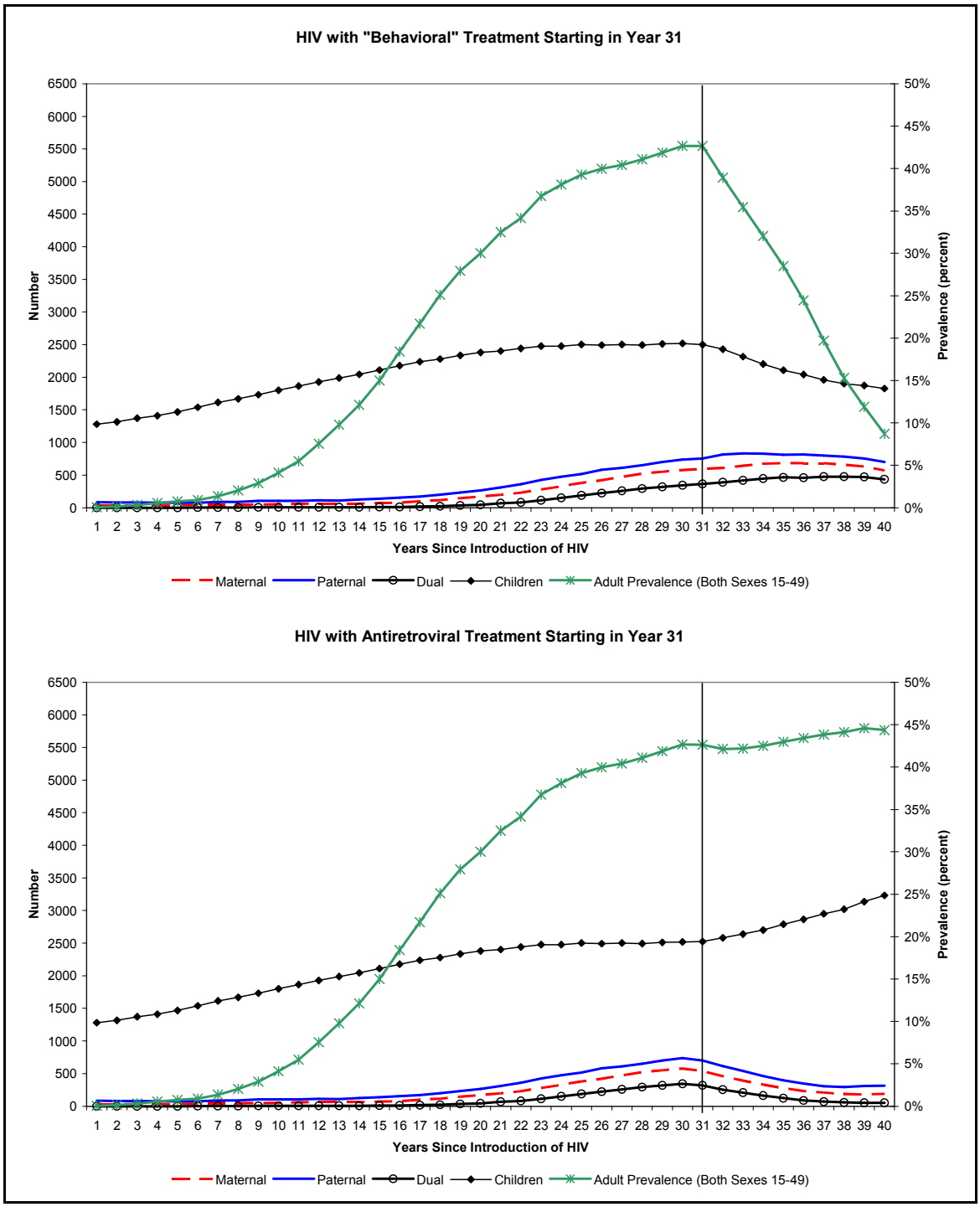

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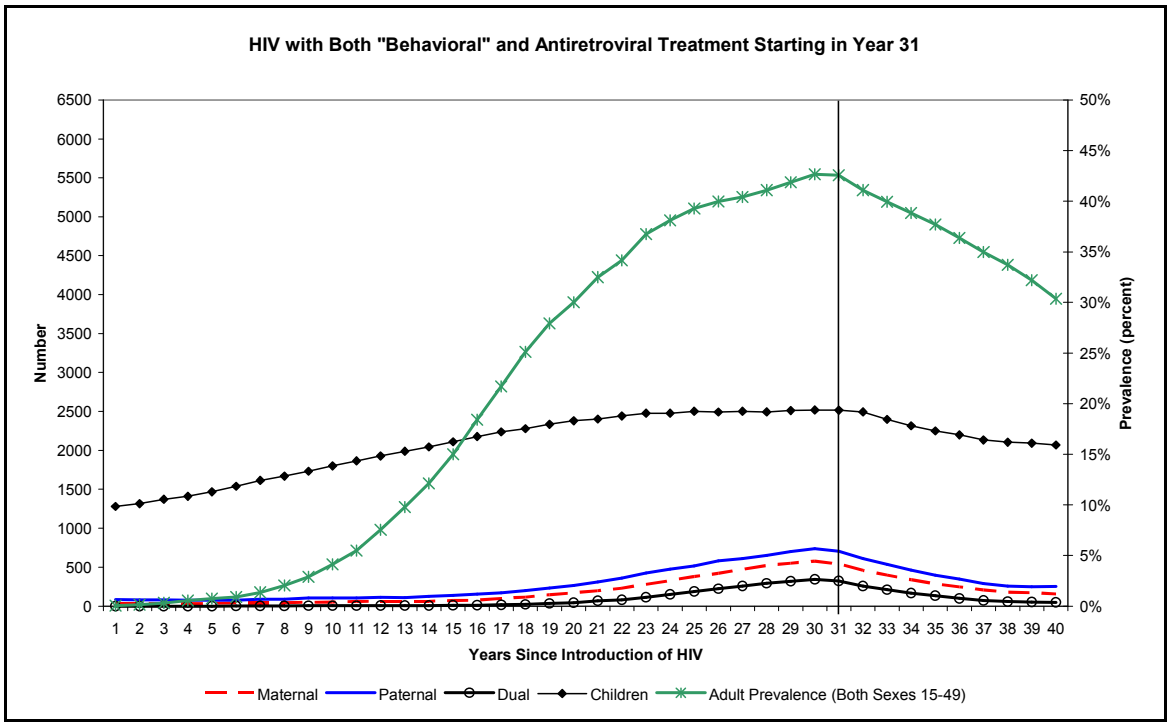

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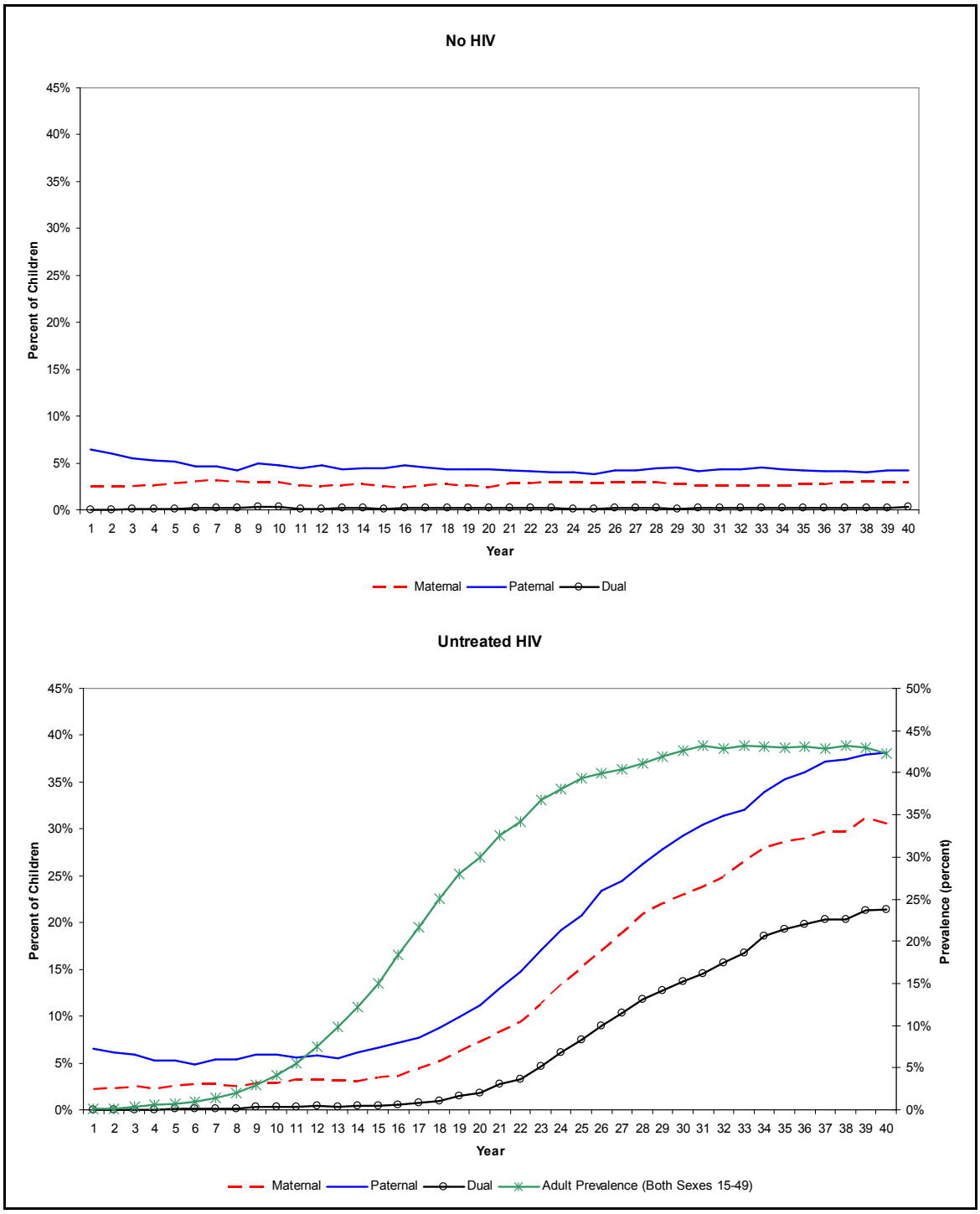

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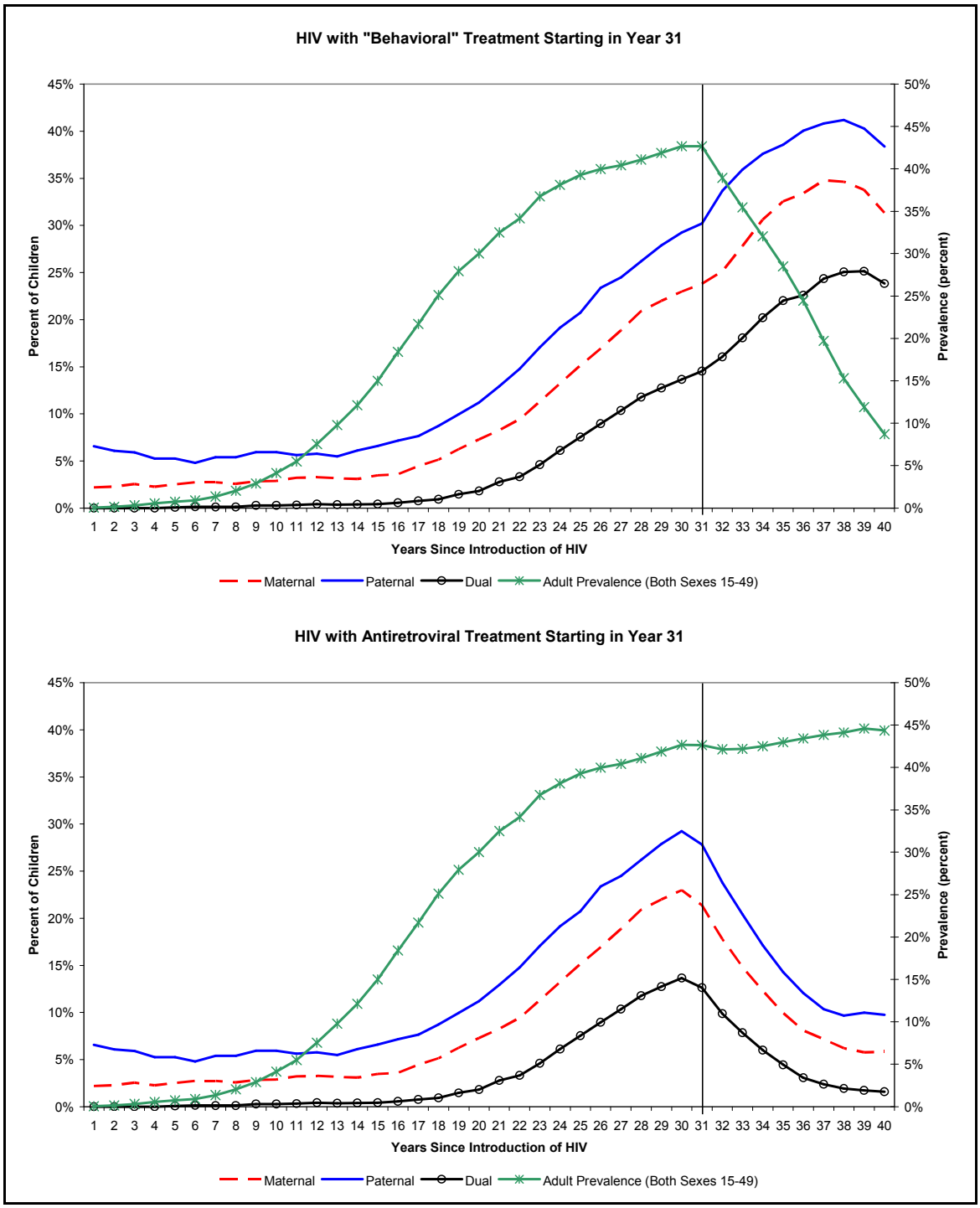

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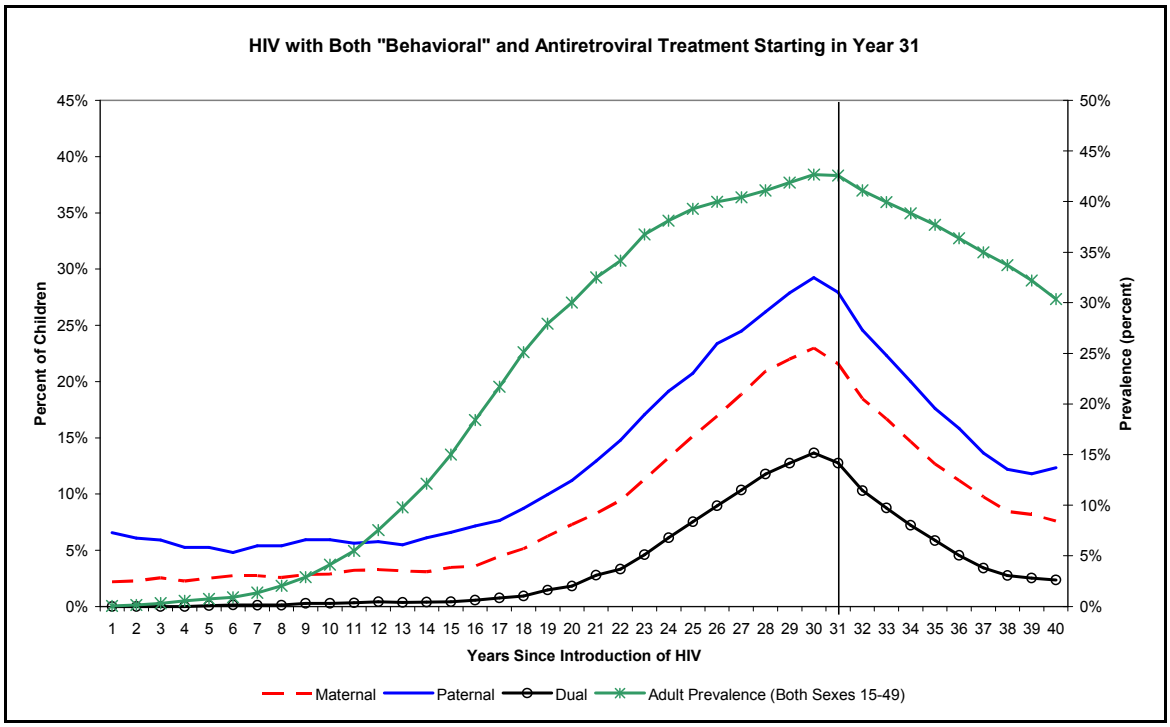

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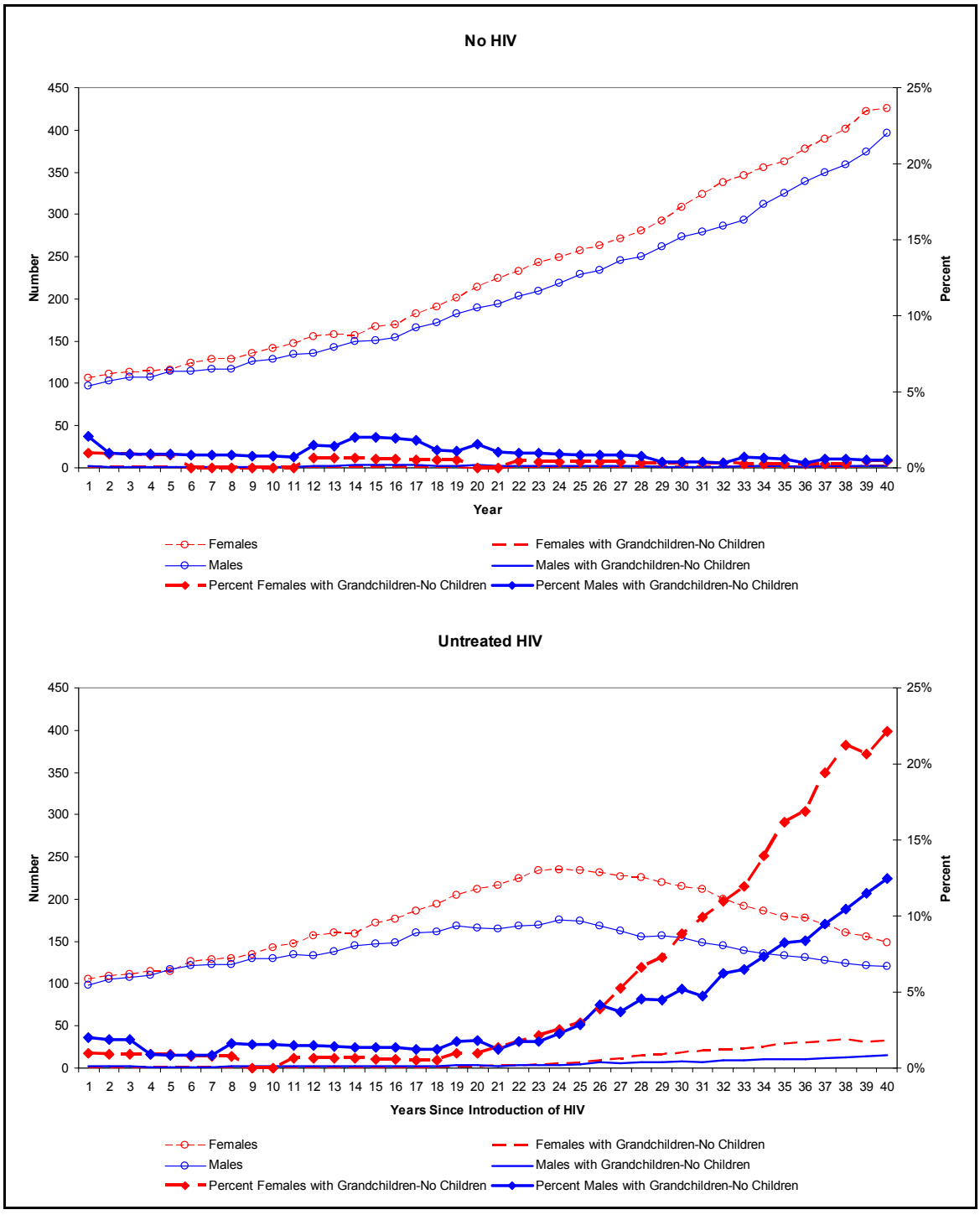

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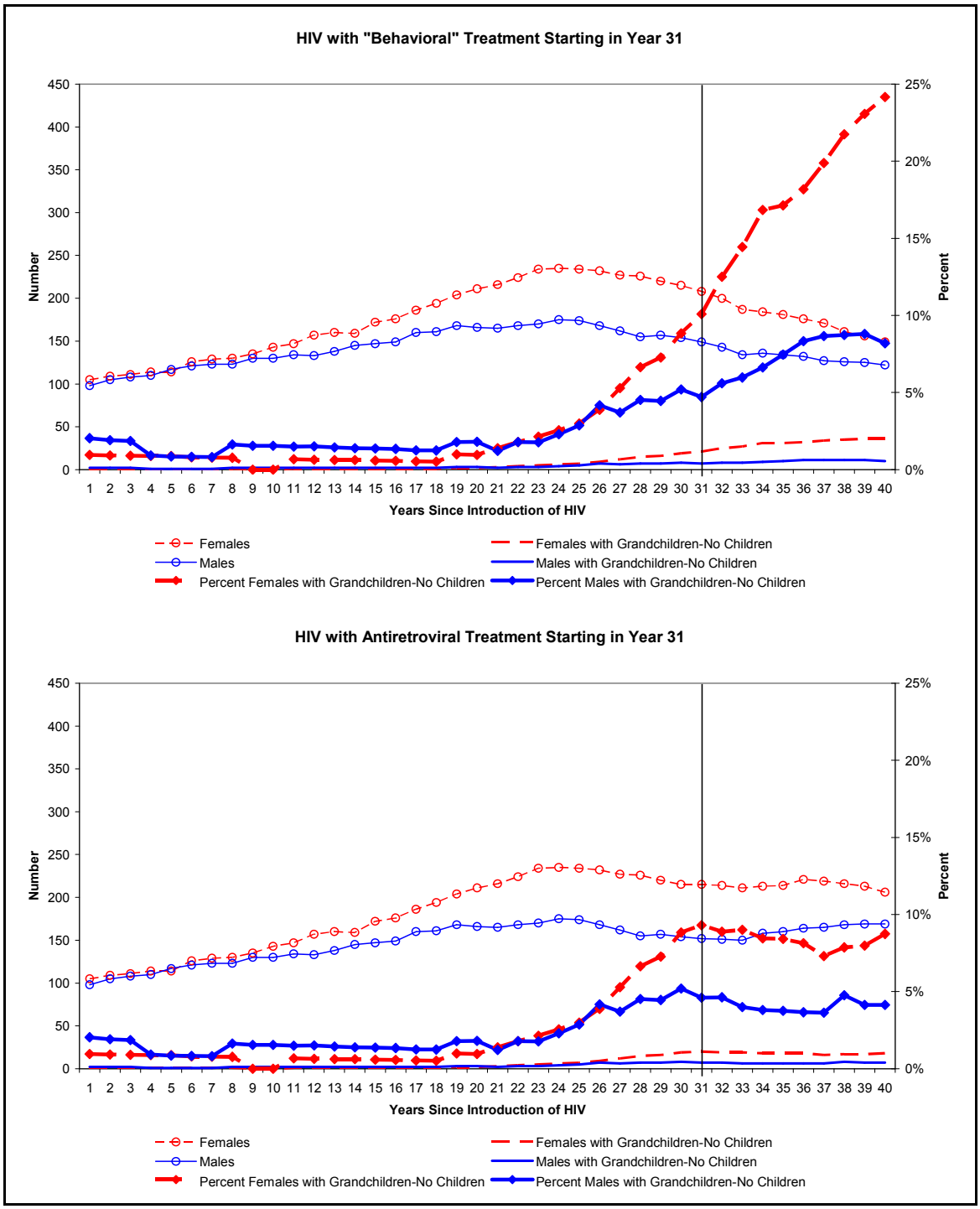

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