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THE EFFECT OF THE HIV/AIDS EPIDEMIC ON AFRICA'S TRUCK DRIVERS

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Abstract. The AIDS epidemic is having a growing impact on the transport sector of the economy of sub-Saharan Africa, where long-distance truck drivers are at an increased risk of infection due to their frequent contacts with commercial sex workers. The spread of AIDS in the transport industry is especially significant to the economy, as truck drivers are largely responsible for transporting crops and supplies needed for daily subsistence. In this paper we analyze these effects via two models, one employing a switch and the other a Verhulst saturation function, to describe the rate at which new drivers are recruited in terms of the supply and demand for them in the general population. Results provide an estimate of the epidemic's economic impact on the transportation sector through the loss of truck drivers (an estimated 10% per year, with endemic levels near 90%).

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1. Introduction. HIV/AIDS, one of the leading causes of death in the world, is especially destructive in Africa. At the end of 2003, an estimated 26.6 million people in sub-Saharan Africa (SSA) were infected with the HIV virus, making up two thirds of the world's HIV-infected population. The estimated number of adults and children newly infected with HIV in 2003 was a startling five million, over three million of which were in SSA. Over three quarters of the estimated three million adult and child deaths due to HIV/AIDS in 2003 occurred in SSA [30]. In fact, the levels of infection are so high that the number of deaths in the next decades may result in population decline [19].

An epidemic of such magnitude has serious repercussions for the African society and economy. The AIDS epidemic is particularly damaging to the transportation sector, where long distance truck drivers (TDs) are at an increased risk of infection due to the migratory nature of their job and their prolonged absence from home. As a result, TDs are more likely to have sexual interactions with commercial sex workers (CSWs), who often provide them with affordable food and lodging during their journeys. The spread of AIDS is further exacerbated by the highly sexually active lifestyles of both the TDs and the prostitutes they visit. Many of the TDs and CSWs have multiple sexual partners (a study of one truck stop [9] found 87% of the TDs reported having a different partner [CSW] every time they visited), and CSWs report that many of their clients insist on not using condoms [4]. A number of studies show a high prevalence of AIDS among long-distance TDs in SSA, and a corresponding high prevalence of the virus among CSWs and their clients [2, 3, 21, 17]. Ramjee and Gouws found an average prevalence of 56% in each of these the two high-risk groups; at one truck stop the prevalence rates were as high as 95% [21]. A 2001 study in Uganda [9], where incidence rates are much lower than in most of Africa, found a 40% overall incidence rate at one truck stop, compared to an overall incidence rate of 8% in the surrounding province.

The spread of AIDS in the transport industry is especially significant to the SSA economy. The TDs are largely responsible for transporting a majority of the goods and supplies needed for daily subsistence. Badly affected areas are losing a large percentage of these valuable skilled drivers to the AIDS epidemic. As more experienced drivers are lost, it may become costly to hire and train new recruits. Moreover, the prevalence of AIDS among experienced TDs is higher than that among the less experienced drivers since these TDs generally have higher wages and can afford repeated visits with CSWs, thereby greatly increasing their chances of infection [2, 3]. Once TDs have contracted HIV, their physical health diminishes, resulting in reduced efficiency of the transport industry as a whole.

In this study, we investigate the effect of the HIV/AIDS epidemic on the population of TDs and CSWs in SSA and more specifically its impact on the transport industry economy. The simple models presented in the following sections track the relationship between the number of men available to work as truck drivers and the number of drivers the transportation industry needs, as the HIV/AIDS epidemic progresses within the TD population. Section 2 considers a model which switches explicitly between cases in which each of these two populations (supply and demand) is greater than the other, while section 3 considers a model where the transition between dominance of supply and demand is smooth (provided by a Verhulst-type function). Finally, we estimate parameters to provide some concrete predictions and discuss implications.

FIGURE 1. Flow diagram for the two-sex SI model

2. The switching model. We begin our investigation with a two-sex SI model as shown in Figure 1. We assume for simplicity that all new TD and CSW recruits are HIV-negative (this is not necessarily true, but our results will thus provide a conservative estimate of the epidemic's impact). TDs and CSWs enter the population under study at respective rates of $f_1(I_1)$ and $f_2(I_2)$ and remain in their jobs an average of $1/\mu_1$ and $1/\mu_2$ units of time, respectively (in the absence of HIV). TDs and CSWs become infected at respective rates of β_{21} and β_{12} , and progress to AIDS at respective rates of δ_1 and δ_2 . (The infection rates incorporate sexual activity and risk levels, and the progression rates include all screenings which may remove individuals before progression to AIDS.)

In models for diseases transmitted by heterosexual contact, it has been established (e.g., [5]) that parameters describing sexual contact rates between men and women are constrained to obey certain balance laws when the populations form a closed network of contacts. In the present context, however, it is important to note that both TDs and CSWs have regular sexual contacts outside the study population: TDs with wives and girlfriends at home and CSWs with other clients and partners. Balance laws, which state that the total number of male sexual contacts in unit time must equal the total number of female sexual contacts in unit time, typically constrain sexual activity rates (here incorporated into the β_{ij}) as functions of the population sizes. Studies (e.g., [2, 10]) suggest, however, that this open network of sexual contacts may prevent TD and CSW sexual activity levels from fluctuating dramatically with changes in population sizes. Instead, CSWs may adjust the proportion of their contacts with TDs versus other clients and partners to meet the demand of TD clients (who may pay more than other clients), while the CSWs' overall activity level, driven primarily by economic need, remains more or less constant. It is the sexual activity level of the outside population which therefore accommodates these fluctuations. The impact of the HIV/AIDS epidemic is magnified by the connections between the TD-CSW core group and the general population, but for simplicity we shall not model explicitly the complete network of sexual contacts.

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FIGURE 2. A graph comparing recruitment rates based on demand for and supply of drivers, as a function of the number of infected drivers.

Recruitment into both populations depends in practice upon two economic factors: the number of jobs the market can support and the number of candidates willing and able to fill them. For TDs we denote these quantities by N_d and N_s and assume that $N_d \langle N_s;$ that is, under normal circumstances truck driving is a desirable enough job that all positions are filled. We also define $\Lambda_d = \mu_1 N_d$ and $\Lambda_s = \mu_1 N_s$ as the recruitment rates corresponding to them. (We similarly define \tilde{N}_d , \tilde{N}_s , $\tilde{\Lambda}_d$ and $\tilde{\Lambda}_d$ for the CSWs.) Therefore, in the absence of HIV, recruitment is driven by the job market; that is, $f_1(0) = \Lambda_d$. However, as the AIDS epidemic invades the TD population, infected drivers begin to quit their jobs sooner than normal because of their illness. At first, the number of excess candidates $N_s - N_d$ can quickly replace the TDs who develop AIDS. However, as the epidemic increases in size, recruitment becomes limited by the pool of candidates N_s .

In this section we consider a model that switches from a demand-driven recruitment to a supply-driven recruitment at the point where the demand for jobs overcomes the supply of candidates. We define this switch point mathematically as $I_{1_{sw}} = \frac{\mu_1}{\delta_1}(N_s - N_d)$, with a corresponding term $I_{2_{sw}} = \frac{\mu_2}{\delta_2}(\tilde{N}_s - \tilde{N}_d)$ for the CSWs. Figure 2 shows the supply-demand graph for $f_1(I_1)$. Figure 3 shows how implementing two such switches (one in each population) divides the model into four distinct regions of operation. In Case I $(I_1(t) < I_{1_{sw}}, I_2(t) < I_{2_{sw}})$ the prevalence of HIV is low enough in both populations that their sizes remain constant. In Case II $(I_1(t) > I_{1_{sw}}, I_2(t) < I_{2_{sw}})$, although the CSW population remains constant, TDs are being lost to AIDS fast enough that recruitment is limited by the number of available candidates. The reverse is true in Case III $(I_1(t) < I_{1\ldots}$, $I_2(t) > I_{2_{sun}}$, while in Case IV both populations' recruitment is affected by the epidemic.

The full switching model is therefore given as follows. Let

$$
f_1(I_1) = \min(\mu_1 N_d + \delta_1 I_1, \mu_1 N_s), \quad f_2(I_2) = \min(\mu_2 \tilde{N}_d + \delta_2 I_2, \mu_2 \tilde{N}_s)
$$

be the respective recruitment rates. Then

$$
I_1' = \beta_{21} \frac{I_2}{N_2} (N_1 - I_1) - (\mu_1 + \delta_1) I_1, \tag{1}
$$

$$
I_2' = \beta_{12} \frac{I_1}{N_1} (N_2 - I_2) - (\mu_2 + \delta_2) I_2, \tag{2}
$$

$$
N'_1 = f_1(I_1) - \mu_1 N_1 - \delta_1 I_1, \tag{3}
$$

$$
N_2' = f_2(I_2) - \mu_2 N_2 - \delta_2 I_2. \tag{4}
$$

Model parameters are summarized in Table 2 in section 4, where their values are estimated.

2.1. Analysis of component submodels. We begin our analysis by determining the behavior of each of the four component models contained in the switching model. If we define $x_1 = I_1/N_1$ and $x_2 = I_2/N_2$, then equations (1) and (2) give us equilibrium conditions $(x_1^*, x_2^*) = (0,0)$ and

$$
(x_1^*, x_2^*) = \left(\frac{\beta_{12}\beta_{21} - (\mu_1 + \delta_1)(\mu_2 + \delta_2)}{\beta_{12}[\beta_{21} + (\mu_1 + \delta_1)]}, \frac{\beta_{12}\beta_{21} - (\mu_1 + \delta_1)(\mu_2 + \delta_2)}{\beta_{21}[\beta_{12} + (\mu_2 + \delta_2)]}\right),
$$
 (5)

the latter (endemic) equilibrium existing only when $R_0 > 1$, with

$$
R_0 = \sqrt{\frac{\beta_{12}\beta_{21}}{(\mu_1 + \delta_1)(\mu_2 + \delta_2)}}.
$$
\n(6)

 $(R_0$ can be calculated using the *next-generation operator* method originated in [7] and detailed in [6].)

In Cases I and III, $f_1(I_1) = \mu_1 N_d + \delta_1 I_1$, so that $N'_1 = \mu_1 (N_d - N_1)$. The solution to this equation is $N_1(t) = N_d + [N_1(0) - N_d]e^{-\mu_1 t}$, so that $N_1(t) \to N_d$ regardless of R_0 . Likewise, in Cases I and II, we find that $N_2(t) \to \tilde{N}_d$. ´

In Cases II and IV, $f_1(I_1) = \mu_1 N_s$, so that $N'_1 = \mu_1 \left(N_s - N_1 - \frac{\delta_1}{\mu_1} I_1\right)$, which has the unique equilibrium $N_1^* = N_s/(1 + \frac{\delta_1}{\mu_1} x_1^*)$. Likewise, in Cases III and IV, we find that $N_2^* = \tilde{N}_s/(1 + \frac{\delta_2}{\mu_2} x_2^*)$. We shall defer the question of stability here to a more general examination (below) and merely observe here that for $N_1 > N_s$, $N'_1 < 0$, while for $N_1 < \frac{\mu_1}{\mu_1 + \delta_1} N_s$, $N'_1 > 0$, so that N_1 remains in $\left[\frac{\mu_1}{\mu_1 + \delta_1} N_s, N_s\right]$ and similarly for N_2 .

The Jacobian matrix at the disease-free equilibrium (henceforth DFE) is

$$
\begin{bmatrix}\n-(\mu_1 + \delta_1) & \beta_{21} \frac{N_1^*}{N_2^*} & 0 & 0 \\
\beta_{12} \frac{N_2^*}{N_1^*} & -(\mu_2 + \delta_2) & 0 & 0 \\
f'_1(0) - \delta_1 & 0 & -\mu_1 & 0 \\
0 & f'_2(0) - \delta_2 & 0 & -\mu_2\n\end{bmatrix}.
$$
\n(7)

The eigenvalues corresponding to the third and fourth rows/columns are negative $(-\mu_1, -\mu_2)$, so that local stability depends on the eigenvalues of the upper left 2×2 submatrix, whose trace is negative and whose determinant is positive precisely when $R_0 < 1$. Therefore, the DFE is locally stable when $R_0 < 1$, regardless of the form(s) of the f_i .

N[∗]

The Jacobian matrix at the endemic equilibrium (henceforth EE) has the form

$$
\begin{bmatrix}\n-(\beta_{21}x_2^* + \mu_1 + \delta_1) & \beta_{21}\frac{N_1^*}{N_2^*}(1 - x_1^*) & \beta_{21}x_2^* & c_{14} \\
\beta_{12}\frac{N_2^*}{N_1^*}(1 - x_2^*) & -(\beta_{12}x_1^* + \mu_2 + \delta_2) & -\beta_{12}\frac{N_2^*}{N_1^*}x_1^*(1 - x_2^*) & \beta_{12}x_1^* \\
0 & -\Delta_1 & 0 & -\mu_1 & 0 \\
0 & -\Delta_2 & 0 & -\mu_2\n\end{bmatrix},
$$
\n(8)

where

$$
c_{14}=-\beta_{21}\frac{N_1^*}{N_2^*}(1-x_1^*)x_2^*,
$$

$$
\Delta_1=\left\{\begin{array}{ccc} 0 & \text{Cases I, III,} \\ \delta_1 & \text{Cases II, IV} \end{array}\right. \text{ and } \Delta_2=\left\{\begin{array}{ccc} 0 & \text{Cases I, II,} \\ \delta_2 & \text{Cases III, IV} \end{array}\right..
$$

In Case I, the eigenvalues corresponding to the third and fourth rows/columns are again negative $(-\mu_1, -\mu_2)$, reducing our analysis to a study of the eigenvalues of the upper left 2×2 submatrix, whose trace is again negative and whose determinant $\beta_{12}\beta_{21}(x_1^* + x_2^* - x_1^*x_2^*)$ (calculated using (5)) is always positive. The Case I EE is thus always locally stable when it exists.

Determining local stability for the EE in general is more complicated. The identities

$$
(\beta_{21}x_2^* + \mu_1 + \delta_1)(\beta_{12}x_1^* + \mu_2 + \delta_2) = \beta_{12}\beta_{21}
$$
\n(9)

and

$$
\beta_{12}\beta_{21}(1-x_1^*)(1-x_2^*) = (\mu_1 + \delta_1)(\mu_2 + \delta_2),\tag{10}
$$

derived from (5), facilitate computations throughout. The characteristic equation has the form $\lambda^4 + a_1 \lambda^3 + a_2 \lambda^2 + a_3 \lambda + a_4 = 0$, where

$$
a_1 = (\beta_{21}x_2^* + \mu_1 + \delta_1) + (\beta_{12}x_1^* + \mu_2 + \delta_2) + \mu_1 + \mu_2 > 0,
$$

\n
$$
a_2 = (\mu_1 + \mu_2)[(\beta_{21}x_2^* + \mu_1 + \delta_1) + (\beta_{12}x_1^* + \mu_2 + \delta_2)] + \mu_1\mu_2
$$

\n
$$
+ [\beta_{12}\beta_{21} - (\mu_1 + \delta_1)(\mu_2 + \delta_2)] + \Delta_1\beta_{21}x_2^* + \Delta_2\beta_{12}x_1^* > 0,
$$

\n
$$
a_3 = (\mu_1 + \mu_2 + \Delta_1x_1^* + \Delta_2x_2^*)[\beta_{12}\beta_{21} - (\mu_1 + \delta_1)(\mu_2 + \delta_2)]
$$

\n
$$
+ \mu_1\mu_2[(\beta_{21}x_2^* + \mu_1 + \delta_1) + (\beta_{12}x_1^* + \mu_2 + \delta_2)] + \mu_1\Delta_2\beta_{12}x_1^* + \mu_2\Delta_1\beta_{21}x_2^* > 0,
$$

and

$$
a_4 = (\mu_1 + \Delta_1 x_1^*)(\mu_2 + \Delta_2 x_2^*)[\beta_{12}\beta_{21} - (\mu_1 + \delta_1)(\mu_2 + \delta_2)] > 0.
$$

The fourth-order Routh-Hurwitz criteria are $a_1 > 0$, $a_4 > 0$, $a_1 a_2 > a_3$ and $a_3(a_1a_2-a_3) > a_1^2a_4$. The first two are demonstrated above, and calculations show

$$
a_1a_2 - a_3 = (\mu_1 + \mu_2)[(\beta_{21}x_2^* + \mu_1 + \delta_1) + (\beta_{12}x_1^* + \mu_2 + \delta_2)]^2 + \mu_1\Delta_1\beta_{21}x_2^*
$$

+
$$
(\mu_1 + \mu_2)^2[(\beta_{21}x_2^* + \mu_1 + \delta_1) + (\beta_{12}x_1^* + \mu_2 + \delta_2)] + \mu_2\Delta_2\beta_{12}x_1^*
$$

+
$$
[(\beta_{21}x_2^* + \mu_1 + \delta_1) + (\beta_{12}x_1^* + \mu_2 + \delta_2)][\beta_{12}\beta_{21} - (\mu_1 + \delta_1)(\mu_2 + \delta_2)]
$$

+
$$
\Delta_1x_1^*[(\beta_{21}x_2^* + \mu_1 + \delta_1)^2 + (\mu_1 + \delta_1)(\mu_2 + \delta_2)] + \mu_1\mu_2(\mu_1 + \mu_2)
$$

+
$$
\Delta_2x_2^*[(\beta_{12}x_1^* + \mu_2 + \delta_2)^2 + (\mu_1 + \delta_1)(\mu_2 + \delta_2)] > 0.
$$

Proof of the final inequality is technical and is relegated to the appendix. With these four conditions satisfied, we see that the EE is always locally stable when it exists.

A result by Thieme (Theorem 1.6 in [27], Theorem 1.5 in [28]) provides a Poincaré-Bendixson type trichotomy that can be applied to the Case I model. Here

the limiting system is equations (1)–(2) with $N_1 = N_d$, $N_2 = \tilde{N}_d$, where by inspection we can see that solutions are bounded in $[0, N_d] \times [0, \tilde{N}_d]$ and Bendixson's criterion rules out periodic solution. Thus local stability extends to global stability in the Case I model.

Finally, a Lyapunov function approach can provide a sufficient condition for the global stability of the DFE when $R_0 < 1$ in Cases II and III. For Case II, let $V = \frac{x_1}{\mu_1 + \delta_1} + \frac{\beta_{21}x_2}{(\mu_1 + \delta_1)(\mu_2 + \delta_2)}$. Then $V > 0$ except at the DFE, and

$$
\frac{dV}{dt} \le x_1 (R_0^2 - 1) + x_1 \left(\frac{\mu_1 + \delta_1 x_1}{\mu_1 + \delta_1} - \frac{\mu_1 + \delta_1 x_1^*}{\mu_1 + \delta_1} \cdot \frac{N_1^*}{N_1} \right) + \frac{\beta_{21}}{\mu_1 + \delta_1} x_2 \frac{\mu_2}{\mu_2 + \delta_2} \left(1 - \frac{N_2^*}{N_2} \right)
$$

Since, as seen above, $N_1' < 0$ for $N_1(t) > N_s$, for any given $\epsilon > 0$ there exists some finite moment $\tau_1(\epsilon)$ after which $N_1(t) < N_s + \epsilon$, so that $-N_1^*/N_1 < -\frac{\mu_1}{\mu_1 + \delta_1 x_1^*}(1-\tilde{\epsilon})$ (where $N_1^* = N_s/(1 + \frac{\delta_1}{\mu_1} x_1^*)$, $\tilde{\epsilon} = -\sum_{n=1}^{\infty} (-\epsilon/N_s)^n < \epsilon/N_s$) and

$$
\frac{dV}{dt} < x_1 \left(R_0^2 - (1 - \tilde{\epsilon}) \frac{\mu_1}{\mu_1 + \delta_1} \right) + \frac{\beta_{21}}{\mu_1 + \delta_1} \, x_2 \frac{\mu_2}{\mu_2 + \delta_2} \left(1 - \frac{N_2^*}{N_2} \right).
$$

Now, since $N_2(t) \to N_2^* = \tilde{N}_d$ monotonically, if $k_{II} = R_0^2 - \frac{\mu_1}{\mu_1 + \delta_1} < 0$, then there Exists some finite moment τ_2 after which $\left(1 - \frac{N_2^*}{N_2}\right) < x_1 |k_{II}| \beta_{12}/R_0^2 \mu_2$, so that for $t > \max(\tau_1(\epsilon), \tau_2), dV/dt < x_1 \frac{\mu_1}{\mu_1 + \delta_1} \tilde{\epsilon}$. Since this is true for any ϵ no matter how small, we conclude that $dV/dt \leq 0$. That is, for $R_0 < \sqrt{\mu_1/(\mu_1 + \delta_1)}$, the Case II DFE is globally stable. By interchanging subscripts, we can obtain a similar result for Case III.

2.2. Switching behavior. We now return to the switching model to assemble a picture of its behavior using the analyses of the component submodels. We first observe that, since equations (1) and (2) are unaffected by the switches in f_1 and f_2 , the values of x_1^* and x_2^* , and therefore the expression for R_0 , are the same as derived above.

To see which equilibria from the component models exist in the switching model, we must compare each $I_1^* = x_1^* N_1^*$ with $I_{1_{sw}}$, and each $I_2^* = x_2^* N_2^*$ with $I_{2_{sw}}$. We see immediately (see Figure 3) that the only DFE that exists in the switching model comes from Case I (and is stable for $R_0 < 1$). For the EE (5), some algebra shows that

$$
x_1^* N_d < I_{1_{sw}} \Leftrightarrow N_d < \frac{N_s}{1 + \frac{\delta_1}{\mu_1} x_1^*} \quad \text{and} \quad x_1^* \frac{N_s}{1 + \frac{\delta_1}{\mu_1} x_1^*} > I_{1_{sw}} \Leftrightarrow N_d > \frac{N_s}{1 + \frac{\delta_1}{\mu_1} x_1^*},
$$

so that each of the two possible values for I_1^* falls on the appropriate side of $I_{1_{sw}}$ (to exist in the switching model) precisely when the corresponding value of N_1^* is the lesser of the two possible values for N_1^* . A similar result holds for population 2. Therefore, the switching model exhibits precisely one EE (which exists, and is stable, if and only if $R_0 > 1$, for which $I_1^* = x_1^* N_1^*$ and $I_2^* = x_2^* N_2^*$ with (x_1^*, x_2^*) given by (5) and

$$
N_1^* = \min\left(N_d, \frac{N_s}{1 + \frac{\delta_1}{\mu_1} x_1^*}\right), \ \ N_2^* = \min\left(\tilde{N}_d, \frac{\tilde{N}_s}{1 + \frac{\delta_2}{\mu_2} x_2^*}\right).
$$

The switching model therefore predicts classical threshold behavior with respect to R_0 (i.e., a forward bifurcation at $R_0 = 1$) and an impact on the overall sizes of populations 1 and 2 based upon the extent to which the epidemic reduces the .

FIGURE 4. A graph comparing $f_{1_{sw}}(I_1)$ and $f_{1_{sat}}(I_1)$ for $A = I_{1_{sw}}$

supply of available drivers (in population 1) or sex workers (in population 2). When the prevalence (5) rises high enough to make $N_s/(1+\frac{\delta_1}{\mu_1}x_1^*)$ fall below N_d , then the number of truck drivers will begin to fall, making transportation of essential goods more difficult.

3. The smooth saturation model. Now suppose that instead of taking a twopart model, we use a single continuous Verhulst-type function to describe the recruitment functions. A function \overline{a} \mathbf{r}

$$
f_{1_{sat}}(I_1) = \mu_1 \left(N_d + (N_s - N_d) \frac{I_1}{I_1 + A} \right)
$$

satisfies the properties $f_1(0) = \Lambda_d$, $f'_1(I_1) \ge 0$, $f''_1(I_1) \le 0$ [a.e.], $\lim_{I_1 \to \infty} f_1(I_1) =$ Λ_s , just as the switching version $f_{1_{sw}}$ does, and in addition it is smooth at A. Since $f_{1_{sat}}(A) = (\Lambda_d + \Lambda_s)/2$, A provides a halfway mark, a measure of how fast saturation occurs (e.g., $A = 0$ gives the "immediately saturated" recruitment function $f_1(I_1) =$ (Λ_s) . We can choose any positive value for A, but if we match derivatives of the two f_1 's at $I_1 = 0$ (which makes $f_{1_{sat}}(I_1) \leq f_{1_{sw}}(I_1) \ \forall I_1$), then we find that A corresponds to the switch point, $A = I_{1_{sw}} = \frac{\mu_1}{\delta_1}(N_s - N_d)$. Figure 4 compares the two recruitment functions.

Now we have a model with I'_1 and I'_2 as in (1) and (2), and

$$
N_1' = \mu_1 \left[N_d + (N_s - N_d) \frac{I_1}{I_1 + A} - N_1 - \frac{\delta_1}{\mu_1} I_1 \right],
$$
 (11)

$$
N_2' = \mu_2 \left[\tilde{N}_d + (\tilde{N}_s - \tilde{N}_d) \frac{I_2}{I_2 + \tilde{A}} - N_2 - \frac{\delta_2}{\mu_2} I_2 \right].
$$
 (12)

This model has the same two equilibrium values for x_1^* and x_2^* , and consequently the same expression for R_0 , as the switching model. The equilibrium condition for N_1 , \overline{a} \mathbf{r}

$$
N_d + (N_s - N_d) \frac{x_1^* N_1^*}{x_1^* N_1^* + A} - \left(1 + \frac{\delta_1}{\mu_1} x_1^*\right) N_1^* = 0,
$$

yields $N_1^* = N_d$ for $x_1^* = 0$ (i.e., for the DFE) and for the EE can be rewritten as

$$
N_1^{*2} - \left(\frac{N_s}{1 + \frac{\delta_1}{\mu_1} x_1^*} - \frac{A}{x_1^*}\right) N_1^* - \frac{N_d}{1 + \frac{\delta_1}{\mu_1} x_1^*} \frac{A}{x_1^*} = 0,
$$

which has the unique positive solution

$$
N_1^* = \frac{1}{2} \left[\left(\frac{N_s}{1 + \frac{\delta_1}{\mu_1} x_1^*} - \frac{A}{x_1^*} \right) + \sqrt{\left(\frac{N_s}{1 + \frac{\delta_1}{\mu_1} x_1^*} - \frac{A}{x_1^*} \right)^2 + 4 \frac{N_d}{1 + \frac{\delta_1}{\mu_1} x_1^*} \frac{A}{x_1^*}} \right]. \quad (13)
$$

We observe that since $N_d \langle N_s \rangle$, replacing N_d in (13) with N_s would increase its value; that is, $N_1^* \leq N_s/(1+\frac{\delta_1}{\mu_1}x_1^*)$. We can also show¹ that for $A = I_{1_{sw}},$ $N_1^* < N_d$. In other words, $N_{1_{sat}}^* \leq \overline{N}_{1_{sw}}^*$, consistent with the fact that $f_{1_{sat}} \leq f_{1_{sw}}$. The expression for N_2^* is similar.

The Jacobian matrices at the DFE and EE likewise follow (7) and (8), respectively, with Δ_1 redefined as

$$
\Delta_1 = \delta_1 - \mu_1 \frac{(N_s - N_d)A}{(x_1^* N_1^* + A)^2},
$$

with x_1^* as in (5) and N_1^* as in (13), and similarly for Δ_2 . Here we observe that, as in the switching model, $\Delta_1 < \delta_1$ and $\Delta_2 < \delta_2$, and if we take $A = I_{1_{sw}}$ and $\tilde{A} = I_{2_{sw}}$, then we also have that

$$
\Delta_1 = \delta_1 \left[1 - \frac{I_{1_{sw}}^2}{(x_1^* N_1^* + I_{1_{sw}})^2} \right] \ge 0,
$$

and similarly for Δ_2 . Therefore, the stability analysis for the EE of the switching model holds here also, and we see that the DFE is again locally stable whenever $R_0 < 1$, while the EE exists and is locally stable for $R_0 > 1$.

4. Parameter estimates. In this section we review studies and data on the model parameters, so as to develop some quantitative estimates and predictions of the epidemic's effect on the populations under study. By necessity all estimates are ballpark figures, as what little data is available relies largely on self-reporting and self-identification and vary widely by location and study.

Contact rates. The rate of infectious contact, β_{ij} , depends on the total number of contacts per person per year and the probability of transmission per contact. According to [23], the per-contact probability of female-to-male transmission ranges in general from 0.0003–0.0060, and the per-contact probability of male-to-female transmission ranges from 0.0005–0.0080. Estimates vary significantly among regions where the epidemic is in different stages, however [23], and one study in Uganda (which did not distinguish between genders and considered only monogamous couples) found an overall per-contact infection probability of 0.0011 [8]. As we are considering the high-risk groups of TDs and CSWs, we will take the probabilities of transmission to be the upper bounds of these ranges. One longitudinal study of a trading town on the trans-Africa highway [20] found CSWs had an average of 5.8 contacts per week, while the male clients studied reported an average of 3.1 contacts per week. This is very likely an undercount for the TD population, as the men in the study were not all TDs, and the usual figure of 2 trips per month [2, 3]—coupled with short periods of only a few days between trips and studies that report high likelihood of CSW contacts while on the road—suggests between 3 and 5 contacts per week; however, we will use it as it

¹We derive the inequality by observing that $N_d - \left(N_s/(1 + \frac{\delta_1}{\mu_1}x_1^*) - A/x_1^*\right) > (N_s (N_d)/\frac{\delta_1}{\mu_1}x_1^*(1+\frac{\delta_1}{\mu_1}x_1^*)$, by building the right-hand side into the radicand of (13), and by taking the square root.

is a rare instance of a direct determination of the figure. We therefore estimate $\beta_{12} = 0.0080$ infections/contact × 5.8contacts/wk × 52wk/yr \approx 2.4infections/yr and $\beta_{21} = 0.0060$ infections/contact × 3.1contacts/wk × 52wk/yr ≈ 0.97 infections/yr. The estimate of approximately 300 contacts per year per CSW is consistent with the range of roughly 63 to 364 calculated from data on four large African cities in [18], although this same study notes that few of the clients of CSWs in large cities are TDs (in one of the cities, only 15% of the clients were TDs [16]).

Natural loss rates. We let the natural (i.e., due to other factors than HIV/AIDS) TD loss rate μ_1 be the reciprocal of the average remaining lifetime of a healthy TD (without AIDS) following recruitment. Assuming that the life expectancy of a healthy South African individual is 62 years [30], and the average age of a TD at recruitment is 29 years (average age of 37 years minus average time on the job of 8 years [21]; this is consistent with Mbugua et al. [17], who report a mean age of 33.5 years for drivers and 28.5 years for assistants), we have $1/\mu_1 = 33$ yr and $\mu_1 \approx 0.03 \text{ yr}^{-1}$.

Gysels, Pool, and Nnalusiba report [10] that CSWs who work at truck stops and bars on the main road are generally in their 20s or early 30s. We will use the estimate of 22.5 years as the age at which CSWs begin working (based on an average age of 25 years and an average time in the profession of 2.5 years given in [21]—this is compatible with an average age of 27.5 years given in [20] and the age range of 19–25 years for first sex work given in [18]), and a heuristic estimate of 40 years as the average age at which they effectively stop working as CSWs. (Gysels, Pool, and Nnalusiba interviewed CSWs as old as 55 but included in their sample women who owned their own bars and therefore had the financial security to negotiate lower risk levels. They classify these women separately from the high-risk CSWs working at truck stops, and we also exclude them from our model.) This provides an estimate of $1/\mu_2 = 17.5$ years and $\mu_2 \approx 0.057$ yr⁻¹.

AIDS-related removal rates. Estimates of rates for progression to AIDS continue to change with time, both because of fuller knowledge of the distribution and because of improving treatments. Here we follow the lead of Hyman et al. [14] in assuming a mean duration of infection of 8.6 years; many more recent estimates are higher but also reflect improved healthcare. Here we assume that since health care in sub-Saharan Africa is often poor or prohibitively expensive, the average remaining working life of an infected TD or CSW (which may end before full progression to AIDS) is relatively short. So we take $\delta_i^{-1} = 8.6$ years, which translates to an AIDS-related death rate of $\delta_i = 0.116$ yr⁻¹ for $i = 1, 2$.

Population of TDs. Many different scales are possible here—a single country, a single stretch of a given highway, or the entire network of sub-Saharan Africa's roads. Here, as an illustration, we begin with the 1996 population of truck drivers in South Africa as reported in the official occupational survey [25], 73,000. As this number does not include the assistants who commonly accompany TDs on longdistance trips and who are equally likely to have sexual contacts with CSWs, we double it to arrive at an estimate of $N_1(0) = 156,000$. We also assume the size of the current TD population to be determined by the number of jobs available, giving N_d this same value. To estimate N_s , we take the number of unemployed, economically active individuals in South Africa from the same year, 2,019,000 [26], and, assuming future HIV screening, reduce it by the 25% HIV prevalence among this population $[22]$ to get 1,514,250. (This figure includes women, so we might halve it, but it also excludes unemployed, economically inactive individuals who

	Est.	Est.	
	total	CSW	
City, country	pop.	pop.	Comments Source
Chirundu, Zimbabwe	2,700	100	31 permanent population only
Chirundu, Zimbabwe	4,000	300	$\left[31\right]$ includes transients
Diego-Suarez, Madagascar	80,000	2684	$\left\lceil 15 \right\rceil$ 12% of 15–49-year-old female pop.
Kisumu, Kenya	231,500	1374	[18] 19.5 CSWs/1000 men aged $15-59$
Ndola, Zambia	433,000	2288	[18] 19.1 CSWs/1000 men aged $15-59$
Cotonou, Benin	653,000	1915	[18] 10.1 CSWs/1000 men aged $15-59$
Bulawayo, Zimbabwe	1,000,000	7000	$[32] % \includegraphics[width=1\textwidth]{images/TrDiM-Architecture.png} \caption{The 3D maps of the estimators in the estimators in the image.} \label{fig:TrDiM-Architecture}$
Yaoundé, Cameroon	1,256,000	5600	$\left[18\right]$ 14.4 CSWs/1000 men aged $15-59$

Table 1. Some single-city estimates for CSW populations

might take a job as a TD if they could get it. The order of magnitude is the main idea here.) Estimates of HIV incidence among TDs varies but is generally reported to be high; we will use the figure of 56% reported in [21] to calculate $I_1(0)$ as of the year 2000.

Population of CSWs. Estimating the size of the CSW population in any area larger than a single town is considerably more difficult; even experts decline to make estimates beyond putting the order of magnitude in the tens of thousands in all of southern Africa [12]. The occupation tends to be driven by economic need, and women leave the profession when they can and return to it when they must [12]. Many CSWs move to follow demand—going to towns on the main road at times of the month when TDs come through [31] and moving to mines and construction sites when such sites arise [12]. For this reason the population of CSWs even in a single roadside town fluctuates according to many factors. Table 1 gives a review of single-city estimates found in the literature; however, only one of these (the first) is a roadside trading village—the rest are major cities, which are not likely to be representative of populations where truck stops and roadside bars are located. Furthermore, different studies used different estimation methods, so estimates may not be comparable. However, in general the estimates suggest that CSWs form approximately 0.3% to 0.7% of the population in larger cities and 3.7% to 7.5% (an order of magnitude higher) in smaller towns located along major commerce routes.

Although in reality any given population of TDs and CSWs is not a closed sexual network—members of both populations have outside sexual contacts—for simplicity we will take the initial proportion of CSWs to TDs to be determined by the balance law for sexual contacts: $3.1N_1(0) = 5.8N_2(0)$, i.e., $N_2(0)/N_1(0) \approx 0.53$. (Recall that 3.1 and 5.8 are the estimates cited earlier in [20] of the average numbers of sexual contacts per week made by CSW clients and CSWs, respectively.) For comparison, one study [29] describes a network consisting of the six major trucking and border towns in Zambia (Chirundu, Livingstone, Chipata, Nakonde, and Kasumbalesa on the borders and Kapiri Mposhi in the interior), which combined have an estimated 250,000 inhabitants, including 1,500 CSWs and 2,000 itinerant TDs (giving 0.75 CSWs per TD). Other estimates have given a ratio as low as 0.3 CSWs per TD. With $N_1(0) = 156,000$, a ratio of 0.53 CSWs per TD gives $N_2(0) \approx 83,000$. Since the CSW population is driven by the economic need of the individual women rather than the demand by TDs for CSWs, we will take $N_2(0)$ as an estimate for \tilde{N}_s . And, since some studies found a median number of clients per week as low as 1

in some areas [18], we will use this as a minimum contact rate in determining demand for CSWs. We therefore set $\tilde{N}_d \cdot 1$ contact/wk = $N_2(0) \cdot 5.8$ contact/wk = 481, 400. (Note that these estimates make $\tilde{N}_s < \tilde{N}_d$, so that the population and recruitment of CSWs is entirely supply-driven regardless of HIV prevalence, in accordance with observations.) To determine an initial condition for I_2 , we will use the HIV prevalence among CSWs reported by [21], which was also 56%.

Table 2 summarizes the estimates of model parameters. These parameters yield an estimate of 9.6 for R_0 and predict a rapid rise to an endemic state in which 86% of TDs and 92% of CSWs are HIV-positive, 10% of the TD population is lost to AIDS per year (bringing the average job lifetime below 8 years), and the CSW population is reduced to about 35% of its current size. The switching model predicts a steady TD population with the current potential recruit population, but the smooth saturation model predicts a drop to about 61% of the available number of jobs. If we change the model to discard the assumption that all new recruits are HIV-negative, endemicity levels rise even further. The rise in prevalence of HIV among TDs may appear sharp—numerical analysis suggests it will require only ten years—but is consistent with the growth of HIV prevalence in this population suggested by some studies in the last decade of the twentieth century (from 26% in 1990–1 in two studies [17] to 56% in 2000 [21]).

However, there are several caveats for interpreting these predictions. The fact that many of the parameters connected with the general population are changing as the AIDS epidemic spreads makes it unlikely that the epidemic will reach a constant level in this study population in the years to come. For example, N_s is likely to decrease over time as the epidemic affects the larger population. A decrease in N_1 is also likely to decrease N_d . We have also not incorporated any behavior changes in the models (see, e.g., $[18]$, p. S67), even to the extent of keeping the CSW activity level constant. In general, however, our simplifying assumptions tend to make our estimates conservative, and the overall conclusion remains valid that sexual network nodes such as truck stops will continue to grow in significance as the AIDS epidemic spreads.

5. Discussion. The spectrum of systems delimited by the two models analyzed in this paper extend the classical two-sex SI epidemic model to situations where disease-induced mortality limits or decreases population size and recruitment. Although the simple caricatures presented here lack the complexity of real sexual networks, the oversimplifications tend toward a conservative estimate of the impact of the HIV/AIDS epidemic on these populations and suggest a way to incorporate the inevitable external effects of the disease on these core groups into estimates. The high prevalence of HIV predicted for the TD and CSW populations is in line with the rapid rise of HIV seen there in the last years of the twentieth century.

Despite the difficulty in estimating the exact sizes of the populations involved in this HIV transmission cycle, it is clear that the growth of the AIDS epidemic among TDs and CSWs is far outpacing that of the general population in Africa. At some point it is possible that TDs will be lost to AIDS faster than they can be replaced. The size of the recruitment rate/pool is critical to maintaining the TD population and thereby the delivery of goods—and, indirectly, the economy as well. An annual loss of even the projected 10% of the TD population means an enormous cost to the economy in terms of training expenses and lost time. By official statistics [24], 690 million metric tons of goods were transported by South African road and rail enterprises in 2003, a number which has been steadily increasing since 1999. One newspaper article [11] claimed that about 870 million metric tons per year of goods are transported by South African trucks,² with an estimated value of about R2000 billion (US\$300 billion). With the estimated TD population given in the previous section, this means that between 4,000 and 6,000 metric tons of goods and supplies are transported per TD annually. As the number of working TDs decreases, the amount of goods transported decreases, posing a major threat to the economic status of the continent. These goods include food and medical supplies, the lack or ineffective distribution of which may cause severe hardships in many small towns and communities throughout SSA.

So what should be done? African countries are already struggling to deal with the AIDS epidemic and its effects. However, this particular population highlights areas that tend to fall between the cracks of traditional public health interventions: truck drivers tend to work under demanding conditions and timetables for many different companies of varying sizes and nationalities, while roadside sex workers carry out a socially stigmatized and typically undocumented business, with few resources available to them. The only approach presently available to these groups with the potential to slow or stop the spread of HIV/AIDS is education to reduce the risk of infection, typically by promoting condom use and providing condoms, information, and referrals.

Sociological studies have identified strategies that may maximize the effects of a targeted education campaign for these populations. Gysels et al. [9, 10] found that middlemen are employed at many truck stops to arrange and mediate TD-CSW interactions, claiming to offer some level of CSW screening to TDs while negotiating price for the CSWs. At places where they are employed, middlemen therefore have some potential influence on both groups. Ramjee and Gouws [21] identified toll plazas and border crossings as places where education campaigns could have an important effect on TDs, as all drivers pass through them regularly. There is also an important opportunity for the TDs' employers to shoulder some responsibility for this education, as it is in their own best interests to take measures which will reduce their turnover and loss rates.

²This figure includes goods transported by enterprises whose primary business is not transport, such as retail companies. The official statistics cited include only transport companies.

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Among CSWs, studies have identified the newest and poorest of the women as those at greatest risk of being infected [1, 10], as they are the ones least available to negotiate safer arrangements for themselves. Indeed, in general the CSW population is driven by economic desperation and feelings of powerlessness, with individual women negotiating better situations for themselves as their financial situations improve. Campbell [4] cites several factors that impede CSWs from insisting on condom use, including clients' refusal to do so, competition for clients, lack of a common language between CSWs and their clients, and lack of self-confidence stemming from early life experiences. To empower CSWs, several studies have recommended developing peer support networks of CSWs through which information and education could be transmitted [4, 10, 12]. In fact, in some areas the TDs and CSWs themselves have been trained as peer educators (e.g., Swaziland's Corridors of Hope initiative [12]).

Therefore, although resources are extremely limited, it is possible to maximize their effect through highly targeted interventions. Future modeling work may be able to predict or clarify the effects of these targeted interventions further, as has been done in other areas (e.g., Hethcote and Yorke's work with the role of core groups in the transmission of gonorrhea [13]). For example, a model which incorporates age distribution for CSWs could predict the effects of interventions targeted at new recruits. Also, as both populations have significant sexual contact with other populations (e.g., 70% of the TDs interviewed by Ramjee and Gouws [21] had wives and girlfriends in rural areas), modeling the wider sexual network may help relate the growth rates of the epidemic in the concentrated and wider populations.

Appendix. Stability of EE for switching model. The fourth and final Routh-Hurwitz criterion for the local stability of the EE of model $(1)-(4)$ is $a_3(a_1a_2-a_3)$ $a_1^2 a_4$, with the a_i as given in section 2.1. Mathematica software was used to calculate an expression for $a_3(a_1a_2 - a_3) - a_1^2a_4$ using the definitions of the a_i but keeping $K \equiv [\beta_{12}\beta_{21} - (\mu_1 + \delta_1)(\mu_2 + \delta_2)]$ intact as a single constant (positive since $R_0 > 1$ when the EE exists) where it appears in a_2 , a_3 , and a_4 . The resulting expression (after cancellations and grouping of identical terms) involves over 400 terms, 28 of which are negative. Proof that the desired inequality holds therefore reduces to finding positive terms which dominate the 28 negative terms. Table 3 identifies the 28 negative terms and some of the corresponding positive terms that dominate them.

Line	Negative term	Positive $term(s)$
	$\Delta_1 \delta_2 \mu_2^2 K^2$	
$\mathbf{1}$	$\frac{1}{\beta_{12}(\beta_{21} + \mu_1 + \delta_1)} = \Delta_1 \delta_2 \mu_2^2 K x_1^*$	$[2]\delta_1\delta_2\mu_2^2K$
	$\delta_1 \Delta_2 \mu_1^2 K^2$	
$\,2\,$		$[2]\delta_1\delta_2\mu_1^2K$
	$\frac{1}{\beta_{21}(\beta_{12} + \mu_2 + \delta_2)} = \delta_1 \Delta_2 \mu_1^2 K x_2^*$ $2\Delta_1 \mu_2^3 K^2$	
3		$[3]\delta_1\mu_2^3K$
	$\frac{\frac{2}{\beta_{12}(\beta_{21} + \mu_1 + \delta_1)} = \Delta_1 \mu_2^3 K x_1^3}{2\Delta_2 \mu_1^3 K^2}$	
$\overline{4}$	$\frac{1}{\Lambda} = \Delta_2 \mu_1^3 K x_2^*$	$[3]\delta_2\mu_1^3K$
	$\frac{\beta_{21}(\beta_{12}+\mu_2+\delta_2)}{\beta_{21}(\beta_{12}+\mu_2+\delta_2)}$ $\Delta_1 \mu_2^2 K^3$	
5		$[2]\delta_1\mu_2^2K^2$
	$\frac{\beta_{12}(\beta_{21}+\mu_1+\delta_1)^2}{\Delta_1\mu_2^2K^2}$	$\beta_{21} + \mu_1 + \delta_1$
	$=\frac{\frac{1}{\beta_{21}+\mu_1+\delta_1}x_1^*}{\frac{\Delta_2\mu_1^2K^3}{4}}$	$\sqrt{[2]\delta_2 \mu_1^2 K^2}$
6		$\beta_{12} + \mu_2 + \delta_2$
	$\frac{\beta_{21}(\beta_{12} + \mu_2 + \delta_2)^2}{\Delta_2 \mu_1^2 K^2}$	
	$=\frac{2\mu_1\Lambda}{\beta_{12}+\mu_2+\delta_2}x_2^*$	
7	$\delta_1^2 \Delta_1 \overline{\Delta_2 K^3}$	$\delta_1 \overline{\Delta_1 \Delta_2 K^3}$
		$\frac{}{\beta _{21}(\beta _{12}+\mu _{2}+\delta _{2})^{2}}$ (& see l. 18)
	$\frac{\beta_{12}(\beta_{21}+\mu_1+\delta_1)\beta_{21}(\beta_{12}+\mu_2+\delta_2)}{<\frac{\delta_1\Delta_1\Delta_2K^3(1-x_1^*)}{\beta_{21}(\beta_{12}+\mu_2+\delta_2)^2}}$	
8	$\delta_2^2 \Delta_1 \Delta_2 K^3$	$\delta_2 \Delta_1 \Delta_2 K^3$ $\frac{1}{\beta_{12}(\beta_{21}+\mu_1+\delta_1)^2}$ (& see l. 19)
	$\frac{\beta_{12}(\beta_{21}+\mu_1+\delta_1)\beta_{21}(\beta_{12}+\mu_2+\delta_2)}{<\frac{\delta_2\Delta_1\Delta_2K^3(1-x^*_2)}{\beta_{12}(\beta_{21}+\mu_1+\delta_1)^2}}$	
	$4\delta_1\Delta_1\overline{\Delta_2\mu_1K^3}$	$3\delta_1\Delta_1\Delta_2\mu_1K^2$
9		
	$\beta_{12}(\beta_{21}+\mu_1+\delta_1)\beta_{21}(\beta_{12}+\mu_2+\delta_2)\\ 3\delta_1\Delta_1\Delta_2\mu_1K^2x_2^*$	$\beta_{12}(\beta_{21}+\mu_1+\delta_1)$ $\delta_1 \Delta_1 \Delta_2 \mu_1 K^2$
	\leq	
	$\beta_{12}(\beta_{21} + \mu_1 + \delta_1)$ $\delta_1\Delta_1\Delta_2\mu_1K^2$	$\overline{(\beta_{21}+\mu_1+\delta_1)(\beta_{12}+\mu_2+\delta_2)}$
	$\frac{+\frac{1}{(\beta_{21}+\mu_1+\delta_1)(\beta_{12}+\mu_2+\delta_2)}}{4\delta_2\Delta_1\Delta_2\mu_2K^3}$	$3\delta_2\Delta_1\Delta_2\mu_2K^2$
10		$\beta_{21}(\beta_{12} + \mu_2 + \delta_2)$
	$\substack{\beta_{12}(\beta_{21}+\mu_1+\delta_1)\beta_{21}(\beta_{12}+\mu_2+\delta_2)\\3\delta_2\Delta_1\Delta_2\mu_2K^2\,x_1^*}$ \lt	$\delta_2 \Delta_1 \Delta_2 \mu_2 K^2$
	$+\frac{\frac{\delta_2\Delta_1(\beta_{12}+\mu_2+\delta_2)}{(\beta_{21}+\mu_1+\delta_1)(\beta_{12}+\mu_2+\delta_2)}}{\frac{2\delta_2\Delta_1\Delta_2\mu_1K^3}}$	+ $\frac{\overline{(\beta_{21} + \mu_1 + \delta_1)(\beta_{12} + \mu_2 + \delta_2)}}$
		$\Delta_1 \delta_2^2 \mu_1 K^2$
11		
	$\begin{array}{c} \beta_{12}(\beta_{21}+\mu_{1}+\delta_{1})\beta_{21}(\beta_{12}+\mu_{2}+\delta_{2}) \\ \delta_{2}\Delta_{1}\Delta_{2}\mu_{1}K^{2} \, x_{2}^{*} \end{array}$	$\beta_{12}(\beta_{21}+\mu_1+\delta_1)$ $\delta_1 \delta_2 \Delta_2 \mu_1 K^2$
	$\beta_{12}(\beta_{21} + \mu_1 + \delta_1)$ + $\frac{\delta_2 \Delta_1 \Delta_2 \mu_1 K^2 x_1^*}{x_1^*}$	$\beta_{21}(\beta_{12}+\mu_2+\delta_2)$
	$+\frac{92}{\beta_{21}(\beta_{12}+\mu_2+\delta_2)}$	
	$2\overline{\delta_1\Delta_1\Delta_2\mu_2}\overline{K^3}$	$\delta_1^2 \Delta_2 \mu_2 K^2$
12		
	$\frac{\beta_{12}(\beta_{21}+\mu_1+\delta_1)\beta_{21}(\beta_{12}+\mu_2+\delta_2)}{\delta_1\Delta_1\Delta_2\mu_2K^2x_2^*}+\frac{\delta_1\Delta_1\Delta_2\mu_2K^2x_2^*}{\delta_1\Delta_1\Delta_2\mu_2K^2x_2^*}$	$\begin{array}{c} \beta_{21}(\beta_{12}+\mu_2+\delta_2) \\ \delta_1\Delta_1\delta_2\mu_2K^2 \end{array}$
	$\beta_{21}(\beta_{12} + \mu_2 + \delta_2)$	
	$\frac{1-\frac{2\mu_{2}K^{2}x_{2}^{*}}{\sigma_{2}}}{4\Delta_{1}\Delta_{2}\mu_{1}^{2}K^{3}}$	$\frac{\beta_{12}(\beta_{21}+\mu_1+\delta_1)}{4\Delta_1\delta_2\mu_1^2K^2}$
13		$\beta_{12}(\beta_{21}+\mu_1+\delta_1)$
	$\frac{\beta_{12}(\beta_{21} + \mu_1 + \delta_1)\beta_{21}(\beta_{12} + \mu_2 + \delta_2)}{4\Delta_1\Delta_2\mu_1^2K^2 x_2^*}$	
	$=\frac{\frac{4\Delta_1-2r_1}{\beta_{12}(\beta_{21}+\mu_1+\delta_1)}}{4\Delta_1\Delta_2\mu_2^2K^3}$	
14		$4\delta_1\Delta_2\mu_2^2K^2$
		$\beta_{21}(\beta_{12}+\mu_2+\delta_2)$
	$\begin{array}{r} \hline \beta_{12}(\beta_{21}+\mu_1+\delta_1)\beta_{21}(\beta_{12}+\mu_2+\delta_2) \\ = \frac{4\Delta_1\Delta_2\mu_2^2K^2\,x_1^*}{\beta_{21}(\beta_{12}+\mu_2+\delta_2)} \end{array}$	
	$6\Delta_1\Delta_2\mu_1\overline{\mu_2K^3}$	$3\Delta_1\delta_2\mu_1\mu_2K^2$
15		$\beta_{12}(\beta_{21}+\mu_1+\delta_1)$
	$\frac{\beta_{12}(\beta_{21}+\mu_1+\delta_1)\beta_{21}(\beta_{12}+\mu_2+\delta_2)}{3\Delta_1\Delta_2\mu_1\mu_2K^2x_2^*}$	$3\delta_1\Delta_2\mu_1\mu_2K^2$
		$^{+}$ $\beta_{21}(\beta_{12}+\mu_2+\delta_2)$
	$+\frac{\frac{\beta_{12}(\beta_{21}+\mu_1+\delta_1)}{\beta_{21}(\beta_{12}+\mu_2K^2 x_1^*}}{+ \frac{3\Delta_1\Delta_2\mu_1\mu_2K^2 x_1^*}{\beta_{21}(\beta_{12}+\mu_2+\delta_2)}}$	

Table 3. The 28 negative terms (signs omitted) in the expression for $a_3(a_1a_2 - a_3) - a_1^2a_4$ and corresponding positive terms in this expression that dominate them

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