

# Mortality impact of AIDS in Abidjan, 1986-1992

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**Objectives:** To quantify the mortality impact of AIDS in the city of Abidjan (Côte d'Ivoire) by a full scale analysis of mortality trends before and after the onset of the epidemic.

**Design:** Data on deaths registered in the 10 vital registration centers of the city between 1973 and 1992, and data on causes of deaths in the four public hospitals were coded and investigated. Data on deaths were compared with census data in order to compute death rates.

**Methods:** Life tables were computed for each of the 20 years of the study. The trends in death rates were analysed during the 10 years before the onset of the AIDS epidemic (1973-1982) and compared with the changing death rates in the following 10 years (1983-1992). Deaths attributable to AIDS were defined as those in excess of the original trends. The evolution in the number of deaths in the hospital allowed an analysis by cause of death.

**Results:** There was a marked increase in death rates starting in 1986, date of the first diagnosed AIDS cases in the city. This increase was significant for both sexes, but more pronounced among men. It was concentrated primarily among young adults (aged 25-44 years) and among older children (aged 5-14 years), and most of it was considered to be attributable to AIDS and related infections, tuberculosis in particular. When data were cumulated from 1986 to 1992, approximately 25 000 persons were estimated to have died of AIDS.

**Conclusions:** The high number of AIDS deaths estimated in Abidjan underlines the heavy toll already paid by African populations, and calls for intensive action.

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**Keywords:** HIV/AIDS, demographic impact, sub-Saharan Africa, mortality, developing countries, age patterns, sex differences, cause of death, Côte d'Ivoire, vital registration

## Introduction

The AIDS epidemic was first visible in the early 1980s in North America, Europe and sub-Saharan Africa, and somewhat later in South America and South Asia. It soon became a major cause of death among young adults, especially men, in the United States and in several European countries [1-3].

In sub-Saharan Africa, HIV seroprevalence rates in adults in the reproductive age group (14-49 years) vary widely among the many countries of the continent, and are among the highest in the world in some countries of Central, East and West Africa. Large cities and some rural areas, such as the Rakai district in Uganda and the Kagera district in Tanzania, seem to have been the hardest hit at the early stage of the pandemic. During recent years,

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however, social networks and population mobility have facilitated the spread of HIV, creating a mosaic of local epidemics of increasing complexity [4].

The potential mortality impact of AIDS is usually estimated using models based on seroprevalence data, patterns of transmission and probability of transitions from HIV to AIDS and from AIDS to death [5]. Models based on seroprevalence rates indicate that AIDS may have a major mortality impact, and may even reverse the positive population growth in extreme cases [6,7].

Vital registration data are usually defective in sub-Saharan Africa, and there is virtually no account of the mortality impact of AIDS outside a few countries where cause of death statistics are available, such as South Africa and Mauritius. Two small scale studies conducted in Uganda indicated that HIV-seropositive persons had a risk of death about 10–20-fold higher than HIV-seronegative persons living in the same conditions [8,9].

Abidjan, the capital city of Côte d'Ivoire, appears to be particularly badly affected by the AIDS epidemic. Both HIV-1 and HIV-2 viruses are present in the city, but HIV-1 seems to be responsible for the majority of the deaths [10–14]. Early studies conducted in 1988 indicated that AIDS was already the leading cause of death among adults in Abidjan hospitals and responsible for a mortality increase in the population [15,16]. At that time, the incidence of AIDS in the city was estimated to be among the highest in the world, higher than in Haiti or New York City, two places also badly affected by the virus.

This study builds on earlier work conducted in Abidjan [17]. It is, however, far more comprehensive than earlier studies, since it takes into account mortality trends over a long period of time (20 years) and throughout the whole population, instead of a simpler comparison of 2 years based on samples. It reviews the demographic evidence of the impact of AIDS in the city of Abidjan from all the available data, and is part of a more comprehensive work on the mortality impact of AIDS in three capital cities of West Africa [18,19].

## Materials and methods

The study drew information from demographic sources (vital registration and census data), and from medical sources (hospital records).

### Vital registration and census data

The primary source of data for this study was vital registration. Deaths in Abidjan are recorded in the 10 vital registration centers, one in each district ('commune') of the city. These 10 centers were visited, and all the non-confidential information available on the death registers was coded and entered into microcomputers for the 1973–1992 period. Approximately 150 000 deaths certificates were available for this study. A few registers were

missing, usually away from the center waiting to be bound in the printing office (fewer than 1% of the total number of deaths). More details on the data collection methods can be found elsewhere [18,19].

The main concern with vital registration data in sub-Saharan Africa is the lack of completeness. However, this was not the case for adult death registration in Abidjan. Abidjan, like a few other French-speaking West African cities (such as Dakar), has a long-standing tradition of good vital registration going back to the colonial period. This is in part due to the fact that registration is compulsory for burial in the city's cemeteries. The completeness was estimated first by comparing the data with the multi-round survey conducted in 1978 in Abidjan [20]. For adult men, the registration was found to be virtually complete, and for adult women, completeness was estimated to be 91%. However, this was not the case for children aged 0–4 years, for whom the completeness was estimated to be 70% in 1978, and may even have declined over time. Completeness of adult death registration was also estimated by using the Preston–Coale method, a standard indirect demographic technique that compares the age distribution of registered deaths with the age distribution at the census [21]. The comparison indicated that death registration in 1975 was 97% complete for adult men, and 81% complete for adult women, and there were no evidence of decline in completeness of adult death registration in 1988 [18,19].

The two censuses conducted in 1975 and 1988 were used for reconstructing the population by age and sex for each year between 1973 and 1992. A simple age and sex-specific log-linear interpolation between the two censuses was used to calculate the population by age and sex at each timepoint. This allowed us to compute age-specific death rates for each year, and therefore period life tables, which were the basis for all further computations.

### Hospital data

Similarly, the four main public hospitals of Abidjan were visited, and all the available information on causes of death, either from death certificates or from the admission registers, was coded and entered into microcomputers. This type of information was much less complete than the vital registration. First, original data could be located only for the 1987–1992 period. Prior to 1986, there were too many elements of information missing to allow a systematic analysis. Second, even with complete information, deaths in hospitals for the 1987–1992 period accounted for about one-half of the deaths in the vital registration: the other half of the deaths had occurred outside of the hospitals.

Despite this information being rather scanty, it was still useful for our study. In fact, the increase in the number of adult deaths between 1987 and 1992 in the hospitals (3974 deaths) closely matched that of the vital registration data over the same period ( $n = 4098$ ). It will be

shown later that this increase was probably due to AIDS and related conditions (opportunistic infections such as meningitis, pneumonia, diarrhea, tuberculosis, septicemia and encephalitis). This indicates that most deaths attributable to HIV/AIDS were likely to have occurred in the hospitals. Consequently, the analysis was restricted to the increase in the number of deaths in the hospital, which could be compared with the increase in the number of deaths in the vital registration. We did not consider the absolute distribution of deaths by cause, as normally done in other studies, since it was severely biased.

In hospitals, the cause of death information was of variable quality. Some of the available death certificates were properly filled in, with an underlying cause of death, an immediate cause, and an associated cause recorded as recommended by the World Health Organization. In other cases, only a single condition was available. In some cases, distinction was simply made between natural death and accidental or violent death. The cause of death remained unknown in a relatively large proportion (31%) of cases.

The first case of AIDS diagnosed in Abidjan hospitals occurred in December 1985. Therefore, the rationale for this study was that any departure from previous mortality trends since 1986 could be interpreted as the impact of AIDS. To further substantiate this demographic argument, the corresponding increase in deaths was matched with the cause-of-death information in the hospitals.

## Results

### Baseline mortality trends, 1973–1982

Some minor improvements in the adult mortality situation occurred between 1973 and 1985. In 1985, the life expectancy at age 15 years was slightly higher for men (50.0 years) than the 1973–1982 average (49.6 years), and likewise for women: 57.5 years in 1985 compared with 56.8 years average in 1973–1982. This mild mortality decline was concentrated in the 35–64-year age group for men and in the 25–54-year age group for women, among young adults of both sexes.

For children, the situation was markedly different. There was a rapid (exponential) decline of mortality over the 1973–1982 period, from an estimated probability of death between birth and age 5 years of 107 per 1000 in 1973 to 41 per 1000 in 1982. This mortality decline started long before 1973, and was consistent with other estimates from demographic surveys since 1963, even though the level of mortality was lower when computed from the vital registration data because of the low completeness of death registration for children.

### Departure from baseline trends

Among adults, death rates fluctuated around baseline trends from 1973 to 1985, then increased suddenly in 1986 (Fig. 1). This change was visible in most age groups and for both sexes, and was particularly strong among

young men and to a lesser extent among young women. A measure of mortality increase was computed by comparing the observed value of death rates in 1992 with expected value from previous 1973–1982 trends. For adult men, this mortality increase was 51% at age 15–24 years, 100% at 25–34 years, 101% at 35–44 years, 20% at 45–54 years, 19% at 55–64 years and 28% at 65–74 years. For adult women, this mortality increase was about one-half that of men: 24% at age 15–24 years, 50% at 25–34 years, 48% at 35–44 years, 29% at 45–54 years, 6% at 55–64 years and 29% at 65–74 years.

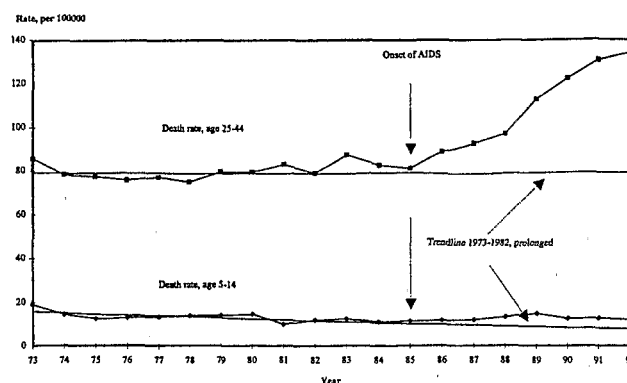


Fig. 1. Change in death rates for children (aged 5–14 years) and young adults (aged 25–44 years) in comparison with the 1973–1982 trend, Abidjan 1973–1992.

Among young children aged 0–4 years, the observed values of death rates were in general slightly above the trend line between 1986 and 1991, but slightly below in 1992. Among children aged 5–14 years for whom the quality of data was much better, there was again a marked departure from trends, starting also in 1986 (Fig. 1). By 1992, mortality of children aged between 5 and 14 years was 49% higher for boys and 56% higher for girls than anticipated from the 1973–1982 trend.

### Pattern of increase by age and sex

To indicate the differential impact of AIDS by age and sex, the observed value of the death rate in 1992 was compared with the expected value from the 1973–1982 trend by detailed age group, and for both sexes. Results revealed two peaks, which were similar for males and females: a first peak at age 5–9 years and a second peak at age 30–39 years (Fig. 2). The adult peak matched what was expected from previous empirical findings, from seroprevalence data and from models based on sexual transmission of AIDS [6,17]. In this case, the age group of 25–44 years is usually the most affected by AIDS. The children's peak was more surprising, which could be due in part to the acquisition of HIV by blood transfusion. In Abidjan, malaria anemia and sickle cell anemia are two frequent pathologies requiring blood transfusion in infants and young children. There is evidence that the supply of blood and blood products was seriously contaminated by HIV in the early years of the epidemic [22]. High levels of HIV infection in malaria and sickle

cell anemia patients who had received blood transfusion have been documented in Africa [23,24]. The increased mortality among children aged 5–14 years could also be due to resurgent tuberculosis among HIV-seronegative children.

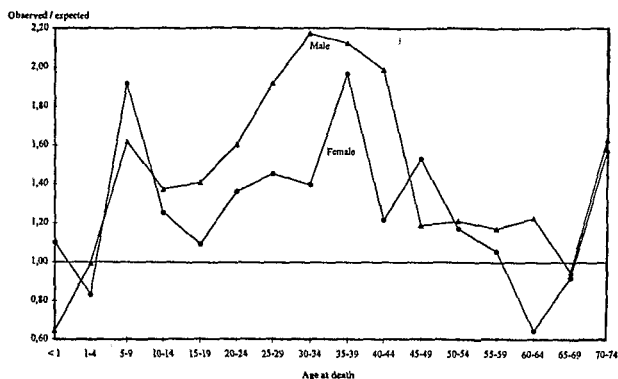


Fig. 2. Relative increase in age-specific death rates in 1992 compared with the 1973–1982 trend, Abidjan.

### Causes of death

Despite all its limitations, the cause of death information confirmed the conclusions of the demographic analysis. In 1992, 6925 deaths were reported in the four public hospitals compared with 2446 in 1987, a relative increase of 183%, which was far above the 33% increase in the population of the city over the same period of time. In absolute numbers, this increase was roughly equivalent to that noted in the vital registration. This excess number of deaths can be broken down by cause of death for broad age groups (Table 1).

For adults aged 15–44 years, 82% of the increase in the number of hospital deaths for which a cause was known (1717 deaths) could be attributed to certified AIDS (374

deaths) or opportunistic infections (1030 deaths). For adults aged 45 years and above, the corresponding proportion was 62% (523 out of 843 deaths). Other causes of death which contributed to the increase in adult deaths were malaria (2%), hypertension (5%), cardiovascular diseases (9%), cerebrovascular diseases (3%), cancers (4%), and accident and violence (1%). Whereas some cancers might be themselves the manifestation of AIDS, two causes were likely to be independent from this cause — malaria, and accident and violence — which combined accounted only for 4% of the increase of deaths in the 15–44-year age group, the most stricken by HIV/AIDS. Hypertension also played a minor role in this age group (3%). It is therefore plausible that HIV/AIDS was responsible for most of the increase in hospital deaths among adults.

Among children aged 5–14 years, certified AIDS and opportunistic infections contributed 59% to the increase in the number of hospital deaths with known cause (57 out of 97), which confirms that AIDS was acting in this age group and was probably responsible for a large part of the observed increase in mortality. This age group is also highly vulnerable to tuberculosis, a resurgent disease closely associated to the AIDS epidemic. Many of the 5–14-year old children may have died of tuberculosis without being HIV-positive. If this was the case, the increased mortality could be considered indirectly due to the HIV/AIDS epidemic. Malaria and violence also contributed significantly (32%) to the increase in the number of deaths in this age group, the other causes having a negligible effect.

Among children aged 0–4 years, the increase in the number of hospital deaths was smaller than the population increase. This appeared to be due to a combination

Table 1. Causes of death responsible for the increase in hospital deaths, Abidjan, 1987–1992.

	Age group (years)				Total
	0–4	5–14	15–44	≥ 45	
Population increase (1992/1987)	1.28	1.41	1.29	1.41	1.33
No. hospital deaths					
1987	908	227	762	549	2446
1992	1118	447	3383	1977	6925
Excess no. deaths, given population increase	– 45	126	2401	1201	3683
Main causes of deaths responsible for the excess no. deaths					
Certified AIDS	– 2	6	374	102	480
Opportunistic infections	– 94	51	1030	421	1408
Malaria	21	19	46	15	101
Vaccine-preventable	– 30	3			– 27
Other infectious/parasitic diseases	– 3	– 4	16	3	12
Hypertension			58	81	139
Cardiovascular			106	112	249
Cerebrovascular			20	60	83
Cancers	– 2	7	56	47	108
Early infancy	105				105
Malnutrition	– 98	2			– 96
Maternal			– 20	5	– 15
Accident and violence	12	12	31	– 4	51
Other/unknown	45	29	684	358	1082

Table 2. Estimations of deaths attributable to AIDS by age and sex in Abidjan, 1992 (deaths cumulated over the 1986–1992 period).

Age group (years)	Male deaths		Female deaths		Deaths attributable to AIDS (1–2+3–4)
	Registered (1)	Expected (2)	Registered (3)	Expected (4)	
0–4	7406	6914	4874	3842	1524
5–14	2993	1571	1665	908	2179
15–24	4102	2794	3091	2771	1628
25–34	9162	2430	3145	2359	7518
35–44	9450	2877	2217	1498	7292
45–54	6282	5004	1485	1099	1664
55–64	4792	4069	1405	1320	808
≥ 65	4306	2283	1549	1450	2122
Total	48493	27942	19431	15247	24735

Expected number of deaths were obtained by extrapolating the 1973–1982 trends in death rates to each year of the 1986–1992 period, and multiplied by the corresponding population in each age group. Expected deaths at age 0–4 years may be underestimated due to lack of completeness of the death registration. Expected deaths at age ≥ 65 years for men may be underestimated due to differential age misreporting in the 1975 and 1988 censuses. Therefore, the derived estimate of deaths attributable to AIDS at age 0–4 years and ≥ 65 years may be overestimated. Estimates for women may also be underestimated to lack of completeness of death registration. Deaths of Abidjan residents who died elsewhere could not be counted. See Results for further details.

of two factors: (1) the mortality decline visible in the vital registration data, and (2) a very small number of certified AIDS deaths in this age group. Furthermore, the number of certified AIDS deaths in 1992 (one case among children aged 0–4 years against 499 cases in other age groups) was even smaller than the number of confirmed AIDS cases in 1987 (three cases). AIDS was certainly present in this age group, and was in fact found in more detailed hospital studies, but in smaller numbers than with other causes of death [17].

### Mortality impact

Assuming that the increase in death rates since 1986 could be entirely attributable to HIV/AIDS, the mortality impact of AIDS was computed by summing over the 7 years from 1986 to 1992 the number of deaths in excess of those expected from the 1973–1982 trend. Results by age and sex indicate that the largest increase was among men aged 25–44 years and among children aged 5–14 years of both sexes (Table 2). In total, approximately 25 000 deaths were attributable to HIV/AIDS in the city of Abidjan since the beginning of the epidemic.

The number of deaths attributable to HIV/AIDS increased dramatically year by year, from 1986 to 1992 (Fig. 3). This pattern of increase was faster than a linear increase but slower than an exponential increase, which was consistent with what is known of the dynamics of the AIDS epidemic due to sexual transmission [25].

Applying a back-calculation model, the number of adult male deaths attributable to HIV/AIDS was used to estimate AIDS cases and HIV infections year by year from 1982 to 1992 [18,19]. The HIV epidemic curve was found to be accurately fitted by a gamma function, which has been used successfully to model the North American AIDS epidemic [26]. The model estimated that the cumulated total number of infections among survivors in 1992 was approximately 91 500 infected persons (plus 25 000 deaths) for an estimated population of 724 000 men — that is a seroprevalence of 13%. This

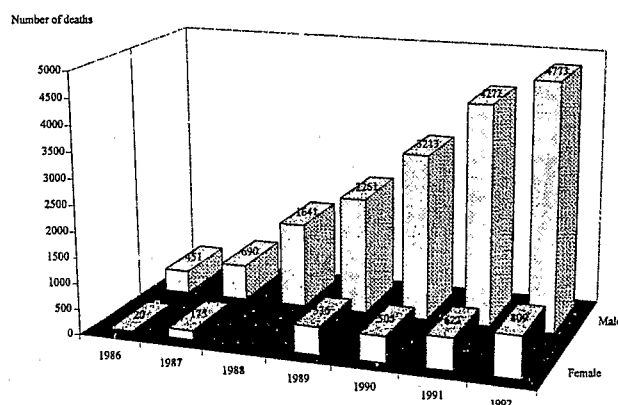


Fig. 3. Estimated number of deaths attributable to AIDS in those aged 15–74 years, Abidjan 1986–1992.

estimate of seroprevalence for men is consistent with two independent estimates of HIV-1 seroprevalence (11 and 14.8%) among pregnant women conducted in Abidjan in the same year (1992), and with an earlier estimate of HIV-1 seroprevalence (8%) among male blood donors in 1990 [27–29]. Similarly, the low estimate of death attributable to HIV/AIDS among children aged under 5 years was also consistent with the low estimate of HIV-1 seroprevalence among healthy children of 0.8% in 1989 [17].

### Discussion

Our crude estimate of the number of deaths attributable to AIDS may suffer from several biases. First, due to inaccuracy in some of the data, the number of deaths attributable to AIDS obtained by difference between observed and expected values of death rates may lead to a serious bias of the impact of AIDS among young children (aged 0–4 years), and in the elderly population (aged 65 years

and above). In contrast, it was certainly underestimated for women (because of underregistration of deaths), and for migrant workers of both sexes who went back to their country of origin in the interval between the onset of AIDS and death. Assuming that the tendency was to overestimate the impact among children and the elderly and to underestimate the effect among young adults, the crude estimate of 25 000 AIDS-related deaths could be taken as a plausible estimate, but should be given a wide confidence interval.

Similarly, we attributed all the increase from previous mortality trends to AIDS, although it is possible that mortality increased at the same time for a few causes of death unrelated to AIDS, such as malaria, accident, violence and hypertension. Unfortunately, data on mortality by cause of death were not available for the years prior to 1986, and were too incomplete for 1987–1992 to warrant such an analysis. Even if these other diseases had played a role in the mortality increase, it must have been minimal. Furthermore, if these diseases were increasing, others must have been decreasing in about the same numbers, since overall mortality was slightly decreasing prior to 1986. Adult mortality started to increase dramatically from 1986, shortly after the date at which AIDS was first clinically documented in Abidjan hospitals. It was therefore reasonable to attribute all of the mortality increase to HIV/AIDS, assuming that the small increase due to malaria, accident and violence, and hypertension tended to be compensated by the other causes which were declining at the same time. It would be surprising indeed if the other increasing causes of death all started to increase at the same time as AIDS, and that the decreasing causes all stopped decreasing in 1986.

The other alternative explanation to the changing trends in mortality may have been that mortality was rising because of the economic crisis and the structural adjustment policy. This can be ruled out. If it had been the case, most of the increase would have been among children and young women, and mostly among vaccine-preventable diseases, which are the most susceptible to this type of situation. The contrary was observed: mortality from vaccine-preventable diseases was still declining in 1992, and young men were the most affected by the mortality increase. It should be noted that great efforts were made in the mid-1980s to increase vaccine coverage, in particular for measles, an action that usually has a dramatic effect on mortality decline.

The order of magnitude of the mortality impact of AIDS in Abidjan, 25 000 AIDS deaths by 1992, is much higher than the official estimates based on certified AIDS cases: 14 655 for the whole country by 1992 [30]. This type of underestimation is considered classic for sub-Saharan Africa [2]. Extrapolated estimates from vital registration data imply a ratio of one certified case for seven in the population [18,19]. This is due to the fact that only one-half of the deaths occur in hospitals and that, among

these, not all are fully diagnosed and match the official definition of AIDS, which is known to be highly specific but to have a relatively low sensitivity [31].

In 1992, the number of registered deaths in the general population ( $n = 11\,639$ ) was more than twice that forecast by the 1973–1982 trends ( $n = 5725$ ). The mortality increase had cost 4.6 years in life expectancy at age 15 years for men, and 1.4 years for women, reversing for the first time in decades the declining trend in mortality in the country. This very rapid doubling of mortality in a few years has virtually no equivalent in the recent world history, outside of major political crisis, such as the dismantling of the Soviet Union and the changing political system which followed, or the massive destruction of the health system caused by a revolutionary movement such as in Mozambique [32,33]. However, it should be noted that, in 1992, the number of registered births ( $n = 71\,005$ ) was still largely in excess (by 59 366) of the number of registered deaths. Even knowing that death registration was not complete that year, a positive natural increase of population should continue to occur in the near future in Abidjan.

Fewer women than men were found to have died of AIDS. Among adults, the ratio of male-to-female AIDS deaths was about 6 : 1, slightly higher than in hospital data or observations of earlier studies [17]. At first glance, this observation seems plausible. First, the female population of young adults is smaller than the male population in Abidjan. Second, the early phase of the epidemic seems to have been characterized by a small pool of women (mainly commercial sex workers) who had sexual contacts with a larger pool of men (their clients) [17]. Furthermore, this sex differential might be exaggerated by biases in the data. First, deaths among women were less likely to be registered, and women were less likely to die in hospital. Second, women might also be more likely than men to go back to their place of origin outside of Abidjan, or even outside Côte d'Ivoire. They could be diagnosed as AIDS cases in Abidjan, but were unlikely to die and be registered in Abidjan. This point deserves further research.

The number of AIDS deaths among children aged 0–4 years was much lower than that predicted by models such as Epi-Model [34]. It may be argued that the small pool of women who became infected in the early phase of the epidemic was not representative of the general population. In particular, these women might be less likely than others to have a sustained relationship with a partner or a spouse, and less likely to give birth. Problems of heterogeneity in vulnerability to HIV/AIDS have been poorly studied so far, and may be crucial for understanding the dynamics of the epidemic during its first years. However, the poor quality of data in this age group may also be invoked to explain this discrepancy.



The back-calculation model used from the Abidjan data for adult men revealed a marked peak of incidence in 1987–1988 [18,19]. Incidence diminished quickly thereafter, which matches data on seroprevalence found to be stable between 1988 and 1992 [17]. This suggests a situation of strong heterogeneity, with a small pool of highly vulnerable individuals who became infected very rapidly, as was the case in the AIDS epidemics among homosexual men in North America. Used prospectively, the same model forecast an average yearly number of about 7500 AIDS cases for the few years after 1992, and about the same number of AIDS deaths.

Further studies are needed to explore the evolution of the epidemics, and the effect of social networks on the dynamics of HIV spread. The apparent first peak and decline of HIV incidence may be followed by a second rise, as HIV may spread to the larger population of adults who are not considered as being at high risk of acquiring infection. In this later phase of the epidemic, it is expected that the ratio of male to female cases will tend to balance, and that there will be an increasing number of pediatric AIDS cases.

By 1992, the cumulative number of deaths attributable to AIDS in Abidjan, a city of about 2 million people, was of the same order of magnitude as the cumulative number of AIDS deaths in France 3 years later (22 485 by June 1995), a country of about 57 million [35]. This estimate could provide clues to the extrapolation of mortality attributable to AIDS in cities where the seroprevalence is much higher than in Abidjan and where the mortality impact could not be measured. It gives a measure of the heavy toll already paid by African populations. The HIV/AIDS epidemic calls for powerful and sustained prevention and care programs.

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## References

- Mann J, Tarantola D (Eds): **Global overview: a powerful HIV/AIDS pandemic.** In *AIDS in the World, Vol II.* New York: Oxford University Press; 1996:5–40.
- Mann J, Tarantola D, Netter T (Eds): **The HIV pandemic: status and trends.** In *AIDS in the World.* Cambridge: Harvard University Press; 1992:9–132.
- Garrett L: *The Coming Plague: Newly Emerging Diseases in a World Out of Balance.* New York: Farrar, Straus and Giroux; 1994.
- AIDS Control and Prevention Program: *Final Report on the Workshop on the Status and Trends of the HIV/AIDS in Africa (Kampala, December 1995).* Cambridge: Harvard School of Public Health, François-Xavier Bagnoud Center for Health and Human Rights; 1995.
- Anderson RM: **Mathematical models of the potential demographic impact of AIDS in Africa.** *AIDS* 1991, 5 (suppl 1):S37–S44.
- Gregson S, Garnett GP, Anderson RM: **Is HIV-1 likely to become a leading cause of adult mortality in sub-Saharan Africa?** *J Acquir Immune Defic Syndr* 1994, 7:839–852.
- Mulder DW, Nunn AJ, Wagner HU, Kamali A, Kengeya-Kayondo JF: **HIV-1 incidence and HIV-1-associated mortality in a rural Ugandan population cohort.** *AIDS* 1994, 8:87–92.
- Sewankambo NK, Wawer MJ, Gray RH, et al.: **Demographic impact of HIV infection in rural Rakai District, Uganda: results of a population-based cohort study.** *AIDS* 1994, 8:1707–1713.
- Chin J, Lwanga SK: **Estimation and projection of adult AIDS cases: a simple epidemiological model.** *Bull WHO* 1991, 69(4):399–411.
- Gersh-Damet GM, Koffi K, Soro B, et al.: **Seroepidemiological survey of HIV-1 and HIV-2 infections in the five regions of the Ivory Coast [letter].** *AIDS* 1991, 5:462–463.
- Soro BN, Gersh-Damet GM, Coulibaly A, et al.: **Seroprevalence of HIV infection in the general population of the Côte d'Ivoire, West Africa.** *J Acquir Immune Defic Syndr* 1990, 3:1193–1196.
- Denis F, Barin F, Gersh-Damet G, et al.: **Prevalence of human T-lymphotropic retroviruses type III (HIV) and type IV in Ivory Coast.** *Lancet* 1987, i:408–411.
- De Cock K, Porter A, Odehouri K, et al.: **Rapid emergence of AIDS in Abidjan, Ivory Coast.** *Lancet* 1989, ii:408–410.
- De Cock KM, Odehouri K, Colebunders RL, et al.: **A comparison of HIV-1 and HIV-2 infections in hospitalized patients in Abidjan, Côte d'Ivoire.** *AIDS* 1990, 4:443–448.
- De Cock K, Barrere B, Dialy L, et al.: **AIDS — The leading cause of adult death in the West African city of Abidjan, Ivory Coast.** *Science* 1990, 249:793–796.
- De Cock KM, Barrere B, Lafontaine M-F, et al.: **Mortality trends in Abidjan, Côte d'Ivoire, 1983–1988.** *AIDS* 1991, 5:393–398.
- Djomand G, Greenberg AE, Sassan-Morokro M, et al.: **The epidemic of HIV/AIDS in Abidjan, Côte d'Ivoire: a review of data collected by project RETRO-CI from 1987 to 1993.** *J Acquir Immune Defic Syndr* 1995, 10:358–365.
- Garenne M, Madison M, Tarantola D, et al.: **Demographic Impact of HIV/AIDS in Three West-African Cities: Vol I, Abidjan [Working Paper Series No. 4].** Cambridge: Harvard School of Public Health, François Xavier Bagnoud Center for Health and Human Rights; 1995.
- Garenne M, Madison M, Tarantola D, et al.: **Conséquences Démographiques du SIDA en Abidjan: 1986–1992 [Etudes du CEPED No. 10].** Paris: CEPED; 1995.
- Antoine P, Herry C: **Enquête Démographique à Passage Répétés, Agglomération d'Abidjan.** Direction de la Statistique. Abidjan: ORSTOM; 1982.
- United Nations: *Manuel X: Techniques Indirectes d'Estimation Démographique.* Département des Affaires Economiques et Sociales Internationales [Etude Démographique No. 81]. New York: United Nations; 1984.
- Francis HL, Quinn TC: **Bloodborne transmission of HIVs in Africa.** In *AIDS in Africa.* Edited by Essex M, Mboup S, Kanki P, Kalengayi MR. New York: Raven Press; 1994:237–249.
- Greenberg A, Nguhen-Dinh P, Mann JM, et al.: **The association between malaria, blood transfusions, and HIV seropositivity.** *JAMA* 1988, 259:545–549.
- Izzia KW, Lepira B, Kayembe K, Odio W: **Syndrome d'immunodéficience acquise et drépanocytose homozygote.** *Annu Soc Belge Méd Trop* 1984, 64:391–397.
- Tarantola D, Mann J, Mantel C, Cameron C: **Projecting the course of the HIV/AIDS pandemic and the cost of adult AIDS care in the**

- world. In *Modeling the AIDS Epidemic, Planning Policy and Prediction*. Edited by Kaplan E, Brandeau M. New York: Raven Press; 1993:3-27.
26. Stoneburner RL, Lessner L, Fordyce EJ, Bevier P, Chiasson MA: **Insight into the infection dynamics of the AIDS epidemic: a birth cohort analysis of New York city AIDS mortality.** *Am J Epidemiol* 1993, **138**:1093-1104.
  27. Diallo MO, Traore V, Maran M, et al.: **Sexually transmitted diseases and HIV-1/HIV-2 infections among pregnant women attending an antenatal clinic in Abidjan, Côte d'Ivoire.** VII International Conference on AIDS in Africa. Yaounde, December 1992 [abstract TP-041].
  28. Sibailly TS, Adjorlolo G, Gayle H, et al.: **Prospective study to compare HIV-1 and HIV-2 perinatal transmission in Abidjan, Côte d'Ivoire.** VIII International Conference on AIDS/III STD World Congress. Amsterdam, July 1992 [abstract WeC 1065].
  29. De Cock KM, Gnaore E, Adjorlolo G, et al.: **Risk of tuberculosis in patients with HIV-I and HIV-II infections in Abidjan, Ivory Coast.** *BMJ* 1991, **302**:496-499.
  30. World Health Organization: **Acquired immunodeficiency syndrome (AIDS).** *Wkly Epidemiol Rec* 1994, **69**:5-8.
  31. Barré-Sinoussi F: **HIV virus variability.** In *AIDS in the World*. Edited by Mann J, Tarantola D, Netter TW. Cambridge, MA: Harvard University Press; 1992:267-275.
  32. Shkolnikov V, Meslé F, Vallin J: **La crise sanitaire en Russie.** *Population* 1995, **50**:907-944.
  33. Garenne M, Coninx R, Dupuy C: **Conséquences de la Guerre Civile au Centre-Mozambique, et Évaluation d'une Intervention de la Croix Rouge [Dossiers du Ceped, No 38].** Paris: Centre Français sur la Population et le Développement; 1996.
  34. Soro BN, Gershy-Damet GM: **The present and future course of the AIDS epidemic in Côte d'Ivoire.** *Bull WHO* 1992, **70**:117-123.
  35. Réseau National de Santé Publique: **Surveillance du SIDA en France: situation au 30 juin 1995.** *Bull Épidémiol Hebdom* 1995, **32**:141-148.



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