Human African Trypanosomiasis (HAT) or sleeping sickness (SS) continues to be a major cause of death in sub-saharan Africa with an estimated 60 million people in 37 countries at risk of the disease (WHO 1994; Cattand 1994). The disease is caused by two sub-species of the trypanosome parasite: Trypanosoma brucei gambiense Dutton 1902 which causes a chronic disease that last for years found in the western and central African countries and T. b. rhodesiense Stephens & Fantham 1910 that causes acute illness that last weeks and found in the eastern and southern Africa. The disease passes through an early stage, when the trypanosomes are mainly in the peripheral blood and lymphoid systems, and a late stage when they have invaded the nervous system. The early stage is characterized by malaise, lassitude and irregular fevers, followed by a range of symptoms including headache, anemia, joint pains and swollen tissues which become worse as the disease moves into the late stage. The advanced stage may be characterized by epileptic attacks, maniacal behaviours, somnolence and coma (Dumas & Bisser 1999.)

Tsetse flies are the major vectors responsible for cyclical transmission of the disease in Africa (Onyiah et al. 1983) of which there are 23 species all capable of transmitting it.

From early records (Baldry 1964), 11 species of tsetse are reported to infest about 75% of the landmass of Nigeria. Generally, the distribution of sleeping sickness in the country was solely dictated by the distribution of the riverine species involving Glossina tachinoides Westwood 1850 and G. palpalis palpalis Robineau-Desvoidy 1830. Nigeria is among the countries in Africa that suffered the devastation of the disease in the late 18th and early 19th century (Johnson & Lloyd 1923; Ford 1971), a situation that resulted in the death of tens of thousands of people from several outbreaks. Despite its size, the treatment and vector control infrastructure of the country was among the best in Africa.

The purpose of this paper is to evaluate the trends of the disease in Nigeria 1931-1990, identify the relationship between the infection rates and the control measures applied and estimate the annual percent change of the disease over the period. This is justified because Africa is passing through another cycle of devastation of HAT (Waisa et al. 2003; Utumba et al. 2005) which demands for such reflections as a prelude for proper planning.

Data Collection

The data for this paper were obtained from the unpublished records of epidemiological section of the Nigerian Institute for Trypanosomiasis Research (NITR) as well as the published annual reports.

Data and statistical analysis

Analyses were conducted using SPSS version 11.0 and Minitab version 7.2. Regression analysis was performed to predict the infection rates as measured by the control measures. The coefficient of determination (R²) was used as a measure of the variance in the infection rates that was attributable to the variation in control measures (Cohen & Cohen 1983). The relationship between the various control measures adopted at that time and infection rates recorded was obtained from Y = b0 + b1X + μ, with the assumption that the relationship between the control measures and infection rates is linear. This implies that variation in Y equals the systematic variation plus random variation.

Where Y = the dependent variable (infection rates)

b0 = the intercept

b1 = combined control measures

μ = unexplained variation

The ordinary linear regression equation used in this paper is R = a + by

Where R = the infection rates

a = the intercept

b = combined control measures

y = the infection period.

Let Yc represent combined control measures in the infection period.

Data stabilisation

In order to stabilise the data and evaluate the trend values of the infection rates, exponential equation in logarithmic form was used:

Yc = abx

LogYc = Log a + XLogb

The above gave a straight line in terms of X and LogY. The normal equations are:

∑ LogY = Nloga + Logb ∑ X

∑ XLogY = loga ∑ X + Logb ∑ X2

Since X origin may be taken at the middle of the period, ∑ X = 0; these equations may be written as:
\[
\sum \log Y = N \log a
\]
\[
\sum X \log Y = \log b \sum X^2
\]

Death prediction from HAT

Death from sleeping sickness was modelled using natural logarithm to identify the disparity between the predicted rates based on log-linear models which increases or decreases as the time periods being forecasted move further from the time of observation and also to identify the fluctuation across time and variability at each time point. These models have been widely used (Brillinger 1986; Brookmeyer & Gail 1988; Kuhn et al. 1994; Devesa et al. 1995; Davis et al. 1997; Chin & Lwanga, 1999).

The total number of persons diagnosed for HAT in Nigeria from 1931-1990 was 523,606 with prevalence of 1.37% (Fig. 1). The first peak was 1931-1940 when more than 370,000 persons were diagnosed as indicated by arrow 1. By the end of 1950, the number had dropped to about 40,000, which further declined to about 30,000 by the beginning of 1951. There was a slight increase in the number of cases between 1961-1970 which was sustained up to late 1970's. The period 1981-1990 witnessed a sudden upsurge in infection rates as indicated by arrow 2. The results showed that the number of cases detected was not directly correlated with the number examined.

Analysis of the mortality rate was only possible for 1970-1979 data. It showed a total mortality of 0.003% out of approximately 28,000 infected persons within that period (Fig. 2). The peak rate was in 1974 with 18.1% deaths followed by 1972 with 17.7% deaths. The overall mortality rate was 0.11 per 10,000 persons.

The prevalence of sleeping sickness according to age as recorded in 1932 is presented in Fig.3. Both sexes in all the age categories showed some degree of susceptibility to the disease except the very old persons aged 40 years and above. Male cases predominated in all age categories even though the sex ratio was not statistically significant (Mann-Whitney = 50.5, P>0.05). From the results of analysis, the degree of relationship between control measures and infection rates as measured by the coefficient of determination was 31.5%. There was a 3.2% reduction in the infection rates per decade.

Following analysis of the early epidemics recorded in the country, the number of persons diagnosed with HAT in Nigeria had continued to decline. However, recent events as indicated in Fig. 1 suggest that the disease may be on the increase unnoticed. This may be owing to a change in the way government perceived the disease following the overwhelming success achieved in its control. This unfortunate development resulted in dwindling funding over several decades, resulting in the breakdown of surveillance and vector control activities. The ecological conditions that could result to epidemic outbreak of HAT are still present. The reason why there has been no outbreak so far, as in other endemic countries is not known. Urgent attention needs to be taken to avoid catastrophe.
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REFERENCES


