A SYSTEM DYNAMICS MODEL FOR THE INTEGRATED CONTROL OF TSETSE FLIES AND AFRICAN TRYPANOSOMIASIS

Donald R. Drew

Professor of Systems Engineering
Virginia Polytechnic Institute and State University
and
Visiting Prof. of Systems Management
American University of Beirut

Abstract

The purpose of this study is to introduce systems thinking into the various aspects of the tsetse and trypanosomiasis problem which are presently dealt with from rather isolated viewpoints: the biological, the agricultural, the economic, the cultural, and the environmental, etc. In short, a systems approach to the problem implies the establishment of the quantification of long-range costs and benefits of combinations of alternative courses of action to deal with the problem, as well as the creation of development planning instrumentalities in African countries currently affected. The approach expostulated in this paper makes use of the computer model displayed in the causal diagram, "Dynamics of Tsetse Fly Control". The equations developed from the causal diagram are expressed in the DYNAMO simulation language. Using the model one can forecast not only the monetary but also the environmental and social costs of various management policies and control strategies. In the paper a range of strategies which focus on the disease nagana, the trypanosome parasite, the tsetse fly agent, and the host (cattle and wild game) are considered including curative drugs, immunization, avoidance, game reduction, brush clearing, biological control, male sterilization and insecticide spraying. By changing parameter values any combination of control techniques can be evaluated against any tsetse specie or trypanosome strain in any environment for various types of land use--pastoral, ranching, subsistence farming, etc.

INTRODUCTION

Socio-economic development in Africa's low-income countries depends to a high degree on the efficiency of public efforts to introduce change--especially in rural areas. A great deal of money is being spent on such rural services as veterinary measures, agricultural management, infrastructure, extension, water resources administration, land use planning, etc., but little is known about the economic efficiency and social, cultural, and environmental impacts of these services. Tsetse control is a case in point.

Tsetse flies occupy an immense area of tropical Africa, amounting to about 12 million square kilometers or about one and a half times the size of the U.S.A. Over much of this range, they are the vectors of trypanosomal diseases that have been responsible for the deaths of tens of thousands of Africans from "sleeping sickness" and of hundreds of thousands of domestic animals from a similar disease called "nagana".

While both diseases have exerted a tremendous influence on the history and progress of Africa south of the Sahara, it was nagana which mainly prevented the development of an indigenous civilization comparable to those associated with other regions of the world. The reason is that, for centuries, Africans have been denied the benefits of large domestic animals. Lack of draft animals for ploughing and pack animals for transport dictated that the African economy be based on the hoe and the head load. Lack of the milk and meat of herds to the extent they were available in other areas resulted in widespread protein deficiency, aggravated by the fact that the staple foods such as yams and cassava are much lower in protein content than cereals consumed in most other parts of the world.

The death of transport animals due to nagana limited the southward penetration of Islamic culture and postponed the introduction of western knowledge. For centuries, the interior was the scene of warfare and slave raids between tribes until huge tracts of country became depopulated and were rapidly invaded by the woodland tsetse. The people had to live in large communities for self-protection and cleared the surrounding vegetation for farms and firewood effectively separating them from the tsetse that infested the bush. The entry of the European powers into tropical Africa led to a return to the old pattern of settlement. Once law and order were established, the people tended to move out of the towns with their degraded farmlands and to form small villages in tsetse-infested bush. Sleeping sickness and nagana began to build up and to

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spread from the old endemic foci to other parts of the continent in epidemic proportions as people were able to make longer journeys over improved roads.

Today, officially, sleeping sickness is considered to be under control. Only about 10,000 new cases are reported annually out of a total of 35 million exposed. However, there are still many small endemic foci where the disease is liable to flare up. When public administration breaks down as happens during periods of political turmoil, then the spectre of epidemic sleeping sickness reappears. Only unremitting vigilance will keep it at the present "acceptable" level.

Presently, nagana is of far greater economic, and even medical, importance than sleeping sickness. Regarding the former, as the disease to which most domestic animals succumb; a major source of man's livelihood is threatened. As to latter, it is of much greater medical importance, if one takes the broader view, that a diet deficiency among the masses is far more critical than a specific disease among the few.

In short, the diseases described by the collective name of African trypanosomiasis, of which tsetse flies are the major transmitting agents, belong to the major factors shaping the environment of the vast region south of the Sahara, and they constitute a major obstacle to its socio-economic development.

BACKGROUND

In 1979 the Food and Agriculture Organization of the United Nations (FAO) decided to establish a Panel of Experts on Development Aspects of the FAO Trypanosomiasis Programme. The Panel held its first session in Lome, Togo, in December 1980, and one of the topics discussed was the possibility of a systems approach in the implementation of the Programme. The following quote is from the Panel discussion:

> "Considering the large number of variables which are likely to have an impact, a large computer simulation model made up of all relevant activities as well as the corresponding resources and constraints may promise the best results. This would permit the various interactions existing between different factors to be taken care of simultaneously. It would be possible to test the influence of changing conditions, e.g. of costs or prices or even new techniques using the model."

A cooperative agreement was entered into between the FAO and the American University of Beirut (AUB) whereby AUB would utilize data available in the FAO information system to develop a simulation model. The preliminary results of this research are reported in this paper.

APPROACH

The purpose of this study is to show how system dynamics can be employed to introduce systems thinking into the various aspects of the tsetse and trypanosomiasis problem which are presently dealt with from rather isolated viewpoints: the biological, the agricultural, the economic, the cultural, the environment, etc. In short, a systems approach to the problem implies the establishment of the quantification of long-range costs and benefits of combinations of alternative courses of action to deal with the problem, as well as the creation of development planning instrumentalities in African countries currently affected.

The approach expostulated here makes use of system dynamics, a methodology that helps disciplinary-oriented analysts and policy makers to think and communicate more clearly about complex issues, the complex issue being here the tsetse problem. The methodology employs the computer to perform scenario analyses by which the effects of the subjective judgements of the disciplinarians can be traced into the future for evaluation. Experience to date has indicated that collaborative decision making is enhanced permitting policy planning to be implemented as social learning, and allowing ethical and value judgements to be part of the development planning process.

The methodology utilizes three complementary forms of expression in the modeling of a problem: verbal, visual and mathematical. The verbal description is a mental model of the system expressed in words. The visual description is diagrammatic and shows cause-and-effect relationships between many variables in a simple, concise manner. The visual model, or "causal diagram", is translated into a mathematical model, the system equations. All forms are equivalent, with any one form merely serving as an aid to understanding for someone who is not fluent in the other languages. However, the verbal description does not lend itself to formal analysis. The visual causal diagram can only be analyzed qualitatively. The mathematical model is by far the most precise and is the only representation of the system that permits quantitative analysis and the evaluation of alternative solutions to a problem.

In the next section we begin by presenting a verbal description of the problem.

PATHOGEN-VECTOR-HOST DYNAMICS

African trypanosomiasis is the presence of trypanosomes, a form of protozoa, living as parasites in man, his domestic animals and game, transmitted by the tsetse fly. While several forms of trypanosomes are pathogenic to domestic stock, this is not true for game which form a natural reservoir from which the disease can be transferred to domestic animals by tsetse carrier. The details of the journeys undertaken by trypanosomes from the time they are sucked up from the warm-blooded host by the tsetse fly, and then returned to another animal when the tsetse bites it, provide us with the understanding needed to disrupt and control the cycle by intervening either directly or indirectly with respect to the parasite, the agent, or the host.

Tsetse flies feed solely on blood and they are always harmless when born. When they feed on a host infected with trypanosomes, some attach to

the walls of the fly's food-canal where they form compact colonies, multiply, and then continue their forward migration back to the fly's salivary glands where the mature, infective forms develop. Some species of tsetse-borne trypanosomes complete an even more complicated journey, traveling to the mid-gut before starting the journey back to become infected saliva. The duration of the cycle takes from 10 to 30 days depending upon the specie of trypanosome and the route taken.

In addition to this "cyclical transmission", transmission of trypanosomes from the host to another may also occur mechanically. The essence of "mechanical transmission" is that a fly (any specie of biting fly, as well as tsetse), when interrupted while feeding on an infected host, completes its meal on an adjacent beast and so mechanically infects the second animal.

Mechanical transmission being relatively unusual, it may assume that transmission of the trypanosomes is effected mainly by tsetse flies. There are 22 species of tsetse, all of which carry African trypanosomiasis. Classified as to habitat, the forest species of tsetse carry only nagana, the riverine species carry Gambian sleeping sickness and nagana, whereas the woodland savannah species carry Rhodesian sleeping sickness and a virulent form of nagana--either preventing the keeping of livestock or producing a crippling lose.

While the fraction of tsetse flies carrying trypanosomes is very high, the fraction with mature infections in which the cycle has been completed in the salivary glands in much lower, and is the relevant indicator if the danger potential of the local tsetse is to be assessed. Hence the longer the fly lives the more likely it is to complete the cycle and thus become dangerously infected. Therefore, it is important that the remarkable life history of the tsetse be understood.

Once inseminated the female tsetse is inseminated for life, as the male sperm is stored in the female and used when an egg requires fertilization. The female carries the young larva around inside her, in safety, until it is fully grown when the mother literally gives birth. By this time the female is about 20 days old. Thereafter the female will give birth about every 10 days until it dies. Since the pupal period is about 30 days, it takes the female about 50 days to produce herself in adult form. The life expectancies for the female and male are about 90 and 50 days, respectively, in addition to the 30 days spent burrowed in the ground as larva.

In addition to its curious life cycle, the ecology of the tsetse fly must be borne in mind when the subject of tsetse control is considered. Three factors in its ecology are climate, vegetation and food. Except in areas where there is little seasonal climatic variation, the fly population decreases throughout the dry season as climatic conditions become more severe, and then increases during the wet season to become maximal near the end of the rains.

It is believed that certain vegetation forms are permanently favored by tsetse flies, others only seasonally, while others are never popular. However, one thing is clear; no species of tsetse can live in treeless, shrubless, grassland, except for small penetrations in search of food, due to the severity of the climate and light intensity. The mother tsetse's reaction to heat is to seek the darkest place she can. The larva, with its own urge to escape light, burrows into moist earth where it will be protected from excessive heat, dryness, and many preditors and parasites.

It is important to know what kind of animals on which the different species of tsetse rely for food. Pigs of different species; cattle, ox and buffalo; rhinos, and elephants are favored hosts. Zebras, baboons, monkeys, dogs, cats, goats and sheep appear not to be favored. Factors affecting the host's suitability are availability, predicability, attractiveness of scent, thickness of skin, and complacency.

The manifold impact of the tsetse spread is pervasive and includes land use, ecology, culture, and economic development. All are interrelated and, ultimately, must be considered comprehensively. For the moment only the impact on the cattle industry will be treated since it affords considerable insight on the impact of the tsetse on all the rest. This is due to the fact that many of the great cattle-owing tribes are nomadic. Their confrontations with tsetse invasions often lead to serious land erosion because the cattle-owners, forced to retreat into small areas which soon became overstocked and degrade the land, refuse to reduce their herds. Their status in the eyes of the community depends upon numbers, not upon the condition of their beasts, nor on their land.

Severeness of the disease in cattle varies with the breed, the general condition of the animal, and with the strain of trypanosome. Under favorable conditions, with good grazing and no work, cattle can survive an acute phase of nagana and pass on into a chronic stage or even become premunized. Conversely, cattle with latent trypansomiasis are likely to die when forced to pull a plough, trek long distances, or are exposed to the elements or other diseases.

Premunity, mentioned above, refers to an equilibrium between the trypanosome and the host such that the animal host shows no clinical symptoms--emaciation, fever, etc.--and is apparently immune against new infection with the same trypanosome strain. This state of immunity is not a stable one and can be lost through stress, except in the case of game animals.

DESCRIPTION OF THE MODEL

The mental or verbal model of the tsetse problem as expressed in the previous section is presented in visual form in the causal diagram in Fig. 1. All the relevant parameter classes employed in the system dynamics methodology--level variables, rate variables, auxiliary variables, supplementary variables and constants are easily identifiable in this form of causal diagram. For example, a level variable is always at the head of a solid arrow and a rate variable is always at the tail of a solid arrow. The sign on the solid arrow tells us if the rate variable adds to or subtracts form the level variable. Whereas solid arrows denote physical flows, dashed arrows in the causal diagram define information flows from level variables to rate variables. Any intermediate variable on the path from a level variable or exogenous input to a rate variable is called an

auxiliary variable. The signs on dashed arrows have the following interpretation: If + it means that an increase in the parameter at the tail of the arrow will cause an increase in the variable at the head of the arrow; if - it means that an increase in the parameter at the tail of the arrow will cause a decrease in the parameter at the head of the arrow. Exogeneous inputs are easily identified on a causal diagram since they have no arrows leading to them, but have one or more dashed arrows emanating from them. Supplementary variables, in contrast, do not form part of the system itself, but merely indicate its performance, and therefore are always identificable as being at the head of a dashed arrow, and having no arrows emanating from them. In summarizing the causal diagramming convention: (1) the arrows describe the direction of causality between pairs of variables; (2) the lines (solid or dashed) denote (physical or information) flows; and (3) the signs tells us the nature of the relationship between a dependent-independent variable pair (direct or inverse).

The DYNAMO equations corresponding to the causal diagram appear in APPENDIX A and these represent the mathematical model of the tsetse-trypanosomiasis problem.

In difference equation terminology, any level variable L_i is expressed as functions of rate variables R_j and the previous value of the level,

$$L_i(t+dt) = L_i(t) + (dt) \sum_{i=1}^{n} R_j(t) \qquad \text{(Eq. 1)}$$

$$i = 1,\ldots,m;$$

with the R_j's assumed to be constant over the interval from t to t+dt. The rate variables are of the form

$$R_j(t) = F[L_i(t), E_k(t), A_{ij}(t), A_{kj}(t)] \qquad \text{(Eq. 2)}$$

where E_k are the set of exogenous inputs that affect R_j directly and A_{ij} and A_{kj} are the impacts of auxiliary variables in the causal streams from the ith level variable and kth exogeneous input, respectively. Since the exogenous inputs are known time functions or constants, if the initial values of the level variables are known, all other variables can be computed from them for that time. Then the new values of the level variables for the next point in time can be found from Eq. 1.

Because of the inability of any computer language to handle subscripts, DYNAMO uses a postscript notation in which .K stands for the present time t, .J stands for past time t-dt, and .L stands for future time t+dt. As in all computer programming upper case letters are used and DT is called the solution intervals, the time between successive computations in the simulation. Since rate variables are assumed to be constant over DT, the double postscript is used, .JK for rates on the right side of an equation and .KL for rates on the left side.

USE OF THE MODEL

A range of possibilities exists for dealing with the tsetse problem. These will be considered briefly with cattle in mind, as they are economically the most important of domestic animals. Strategies may be focused on the disease, on the parasite--the trypanosome, on the agent--the tsetse fly, or on the host--cattle and game. The attacks may be direct or indirect and involve medical, chemical, biological, or mechanical techniques. They include the following:

(1) Curative Drugs. In the early stages, nagana can be cured by drugs which can kill trypanosomes in the blood stream. The problems are that the disease is difficult to diagnose in the early stage and drug-resistant strains of trypanosomes develop.

(2) Immunization, Prophylactic drugs afford protection to cattle for two to four months. The problems are cross resistance as occurs with curative drugs and their doubtful effectiveness in areas of high tsetse infestation.

(3) Avoidance. The principle here is simply one of reducing cattle-fly encounters by keeping them out of infested areas.

(4) Substitution. Some breeds of cattle, though infested, are considerably more resistant to nagana than others. These are primarily small breeds that have the added disadvantage of maturing slowly.

(5) Game Reduction. The elimination of game represents a direct attack on the pathogen by destroying it in its natural host, and an indirect attack on the vector by destroying its source of food. However, Africa's great heritage is facing extinction and game reduction as an end in itself is out of the question.

(6) Wildlife Utilization. The idea here is to exploit, not destroy, the natural productivity of the tsetse-trypanosome-fauna ecosystem. The major possibilities of wildlife utilization include: game viewing, sport hunting, game cropping, and game farming. The problem is the failure of our accounting systems to measure and reconcile the concepts of utilization and conservation.

(7) Mechanized Bush Clearing. This indiscriminate removal of vegetation deprives the tsetse of shade and resting sites. However, its affect on wildlife is probably more devastating than game reduction achieved through hunting and there is the danger of erosion.

(8) Barrier-Clearings. Three kilometer-wide barrier-clearings can isolate tsetse-infested areas. However, periodic maintenance of the barrier and continuous operation of pickets along important routes is needed.

(9) Selective Clearing. Discriminate clearing usually entails the removal of only 2-10% of the vegetation and is therefore cheaper and less destructive. Unfortunately, there are many places where the method cannot work because of the ubiquity and uniformity of the vegetation.

(10) Biological Control. The basic idea here is to let natural enemies of the tsetse such as predators, parasites, and pathogens do the job. However, while the tsetse has enemies, none are outstanding and no attention has been made to introduce new ones.
(11) Laboratory Sterilization. Male tsetse born in the laboratory can be sterlized by irradiation and chemical means and then released systematically into a natural population in such numbers as to compete with the unsterilized males. However, because of its low reproductive potential, mass rearing of colonies of tsetse in the laboratory is difficult and time consuming.
(12) Field Sterilization. Flies would be sterilized by chemicals in the field. This would depend on efficient trapping methods for catching the flies.
(13) Insecticides. There are two main techniques of use. In one method, insecticides are sprayed with discrimination to particular parts of trees where the insects are known to rest. The other method involves aerial spray applications which kill all insects in a given area. Both must be repeated several times at about three week intervals so that adults that emerge from buried pupae are destroyed. Some insecticides, like endosulfan are toxic to fish, making their use in the rivarine tsetse habitat problematic.

The outputs of computer runs reflecting these control strategies are presented in the form of computer graphs in Appendix B.

IMPLEMENTATION

It is evident that no one control technique is ideal. What is needed is an integrated tsetse control concept in which several techniques are combined in such a way as to maximize control effectiveness for a given region. The integrated control concept against the disease and the fly must be designed in conjunction with the form of land use made possible by the control effects. Before implementing any control, one must understand the manifold effects on the ecosystem--the impacts on land-use, the environment, and on economic development. This understanding can be gained only slowly, and sometimes disastrously, by experimenting directly on the system.

In 1973, a famine struck six west African counties south of the Sahara, resulting in the death of some 100 thousand people and five million cattle. The famine was blamed on a drought that began in 1968, but in reality the drought was only the trigger that led to upset of the delicate ecological balance. The real cause was overzealous tsetse control permitting vast expansion of cattle herding leading to overgrazing which drastically altered the vegetation to an extent which may possibly reduce future rainfall and thus permanently impoverish this vast zone.

In contrast, a mathematical model of an ecosystem permits one to predict not only the monetary but also the environmental and social costs of various management policies and control strategies. The model presented in the Appendix is meant to be illustrative rather than conclusive. By changing parameter values, any combination of control techniques can be evaluated against any tsetse specie or trypanosome strain in any environment for various types of land use--pastoralism, ranching, subsistence farming, etc.

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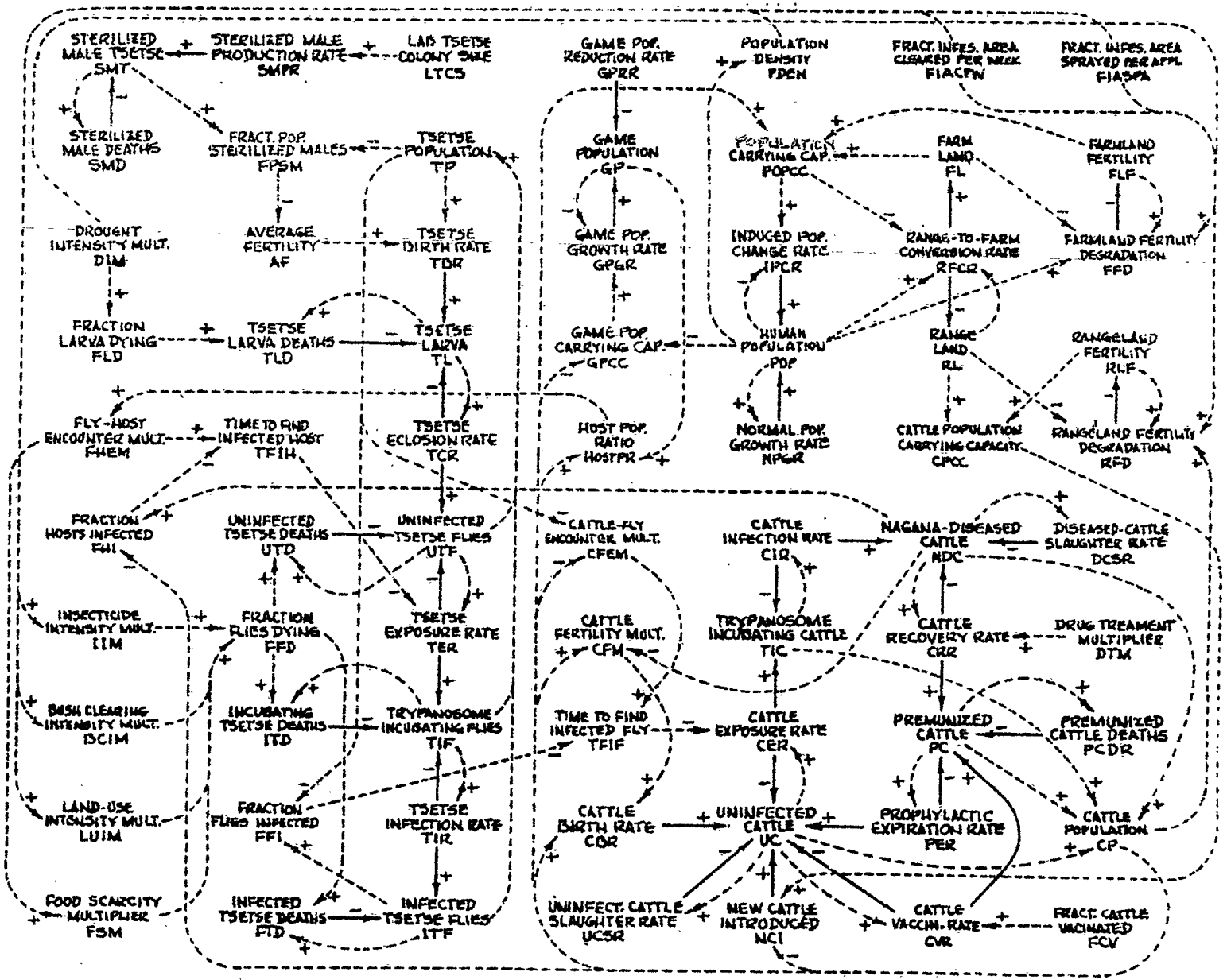

DYNAMICS OF TSETSE FLY CONTROL

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NOTE   TSETSE FLY LIFE CYCLE
L TL.K=TL.J+(DT)(TBR.JK-TLD.JK-TCR.JK)                    (1)
N TL=TLN                                                  (1.1)
C TLN=12E6                                                (1.2)
NOTE   TSETSE LARVA (FLIES)
R TCR.KL=TL.K/PP                                          (2)
NOTE   TSETSE ECLOSION RATE (FLIES/WK)
C PP=5                                                    (2.1)
NOTE   PUPAL PERIOD (WK)
L UTF.K=UTF.J+(DT)(TCR.JK-UTD.JK-TER.JK)                  (3)
N UTF=UTFN                                                (3.1)
C UTFN=6E6                                                (3.2)
NOTE   UNINFECTED TSETSE FLIES (FLIES)
R UTD.KL=UTF.K*FFD.K                                      (4)
NOTE   UNINFECTED TSETSE DEATHS (FLIES/WK)
R TER.KL=UTF.K/TFIH.K                                     (5)
NOTE   TSETSE EXPOSURE RATE (FLIES/WK)
L TIF.K=TIF.J+(DT)(TER.JK-ITD.JK-TIR.JK)                  (6)
N TIF=TIFN                                                (6.1)
C TIFN=2E6                                                (6.2)
NOTE   TRYPANOSOME INCUBATING FLIES (FLIES)
R ITD.KL=TIF.K*FFD.K                                      (7)
NOTE   INCUBATING TSETSE DEATHS (FLIES/WK)
R TIR.KL=TIF.K/CDT                                        (8)
NOTE   TSETSE INFECTION RATE (FLIES/WK)
C CDT=2.5                                                 (8.1)
NOTE   CYCLE DURATION IN TSETSE (WK)
L ITF.K=ITF.J+(DT)(TIR.JK-FTD.JK)                         (9)
N ITF=ITFN                                                (9.1)
C ITFN=4E6                                                (9.2)
NOTE   INFECTED TSETSE FLIES (FLIES)
R FTD.KL=ITF.K*FFD.K                                      (10)
NOTE   INFECTED TSETSE DEATHS (FLIES/WK)
A TP.K=UTF.K+TIF.K+ITF.K                                  (11)
N TPN=UTFN+TIFN+ITFN                                      (14.4)
NOTE   TSETSE POPULATION (FLIES)
R TBR.KL=TP.K*AF.K                                        (12)
NOTE   TSETSE BIRTH RATE (FLIES/WK)
A AF.K=AFN*TABLE(AFMT,FPSM.K,0,1,.2)                      (13)
C AFN=.40                                                 (13.1)
T AFMT=1/.7/.4/.1/0/0                                     (13.2)
NOTE   AVERAGE FERTILITY (1/WK)
A FFD.K=FSM.K*BCIM.K*IIM.K*LUIM.K/AL.K                    (14)
NOTE   FRACTION FLIES DYING (1/WK)
A AL.K=ALN*TABLE(ALMT,TP.K/TPN,0,100,10)                  (14.2)
C ALN=10                                                  (14.1)
T ALMT=1/1/.98/.95/.90/.83/.73/.60/.44/.24/0              (14.3)
NOTE   AVER LIFETIME (WK)
A FSM.K=TABHL(FSMT,HOSTPR.K,0,2,.25)                      (15)
T FSMT=8/4/2/1.3/1/.8/.7/.63/.6                           (15.1)
NOTE   FOOD SCARCITY MULT (DIM)
A TFIH.K=TFIHN*TFIHM.K                                    (16)
C TFIHN=5.0                                               (16.1)
A TFIHM.K=TABLE(TFIHMT,FHI.K,0,1,.1)                      (17)
T TFIHMT=40/20/10/5/2/1/.5/.25/.13/.06/.04                (17.1)
NOTE   TIME TO FIND INFECTED HOST (WK)
R TLD.KL=TL.K*FLD.K                                       (18)
NOTE   TSETSE LARVA DEATHS (FLIES/WK)
A FLD.K=FLDN*DIM.K                                        (19)
C FLDN=.2                                                 (19.1)
NOTE   FRACT LARVA DYING (DIM)
A DIM.K=1.0-SIN(6.283*TIME.K/52)*AMPLI                    (20)
C AMPLI=0                                                 (20.1)
NOTE   DROUGHT INTENSITY MULT (DIM)
A FPSM.K=SMT.K/TP.K                                       (21)
NOTE   FRACTION POPULATION STERILIZED MALES (DIM)
L SMT.K=SMT.J+(DT)(SMPR.JK-SMD.JK)                        (22)
N SMT=SMTN                                                (22.1)
C SMTN=0                                                  (22.2)
NOTE   STERILIZED MALE TSETSE (FLIES)
R SMD.KL=SMT.K/LM                                         (23)
NOTE   STERILIZED MALE DEATHS (FLIES/WK)
C LM=7                                                    (23.1)
NOTE   LIFETIME MALES (WK)
R SMPR.KL=LTCS.K*SMPF                                     (24)
NOTE   STERILIZED MALE PRODUCTION RATE (FLIES/WK)
C SMPF=.2                                                 (24.1)
NOTE   STERILIZED MALE PRODUCTION FRACTION (FLIES/FLY-WK)
A LTCS.K=CLIP(LTCSN,0,TIME.K,PIT)                         (25)
C LTCSN=0                                                 (25.1)
C PIT=10
NOTE   LAB TSETSE COLONY SIZE (FLIES)
A BCIM.K=TABLE(BCIMT,FIACPW.K,0,1,.2)                     (26)
T BCIMT=1/1/1/1/1/1                                       (26.1)
NOTE   BUSH CLEARING INTENSITY MULT (DIM)
A FIACPW.K=CLIP(FIACN,0,TIME.K,40)-CLIP(FIACN,0,TIME.K,49)  (27)
C FIACN=0                                                 (27.1)
NOTE   FRACT INFESTED AREA CLEARED PER WK (DIM)
A IIM.K=TABLE(IIMT,FIASPA.K,0,1,.2)                       (28)
T IIMT=1/1/1/1/1/1                                        (28.1)
NOTE   INSECTICIDE INTENSITY MULT (DIM)
A FIASPA.K=PULSE(FIAS,52,4)                               (29)
C FIAS=0                                                  (29.1)
NOTE   FRACT INFESTED AREA SPRAYED PER APPLICATION (DIM)
A LUIM.K=TABLE(LUIMT,PDEN.K,0,100,100)                    (30)
T LUIMT=1/6                                               (30.1)
NOTE   LAND USE INTENSITY MULT (DIM)
NOTE   GAME SECTOR
A HOSTPR.K=(GP.K+GP.K)/(GPN+PCN)                          (31)
NOTE   HOST POPULATION RATIO
L GP.K=GP.J+(DT)(GPGR.JK-GPRR.JK)                         (32)
N GP=GPN                                                  (32.1)
C GPN=12000                                               (32.2)
NOTE   GAME POPULATION (ANIMALS)
R GPGR.KL=(GPCC.K-GP.K)*GPGF                              (33)
NOTE   SAME POP GROWTH RATE (ANIMALS/WK)
C GPGF=.001                                               (33.1)
NOTE   GAME POP GROWTH FRACTION (1/WK)
A GPCC.K=MAX(1200,(100000-(POP.K*.2)-(GP.K*2))*.12)       (34)
NOTE   GAME POP CARRYING CAPACITY (ANIMALS)
R GPRR.KL=CLIP(GPRRS,0,TIME.K,10)-CLIP(GPRRS,0,TIME.K,35)  (35)
C GPRRS=0                                                 (35.1)
NOTE   HUMAN POPULATION SECTOR
L POP.K=POP.J+(DT)(NPGR.JK+IPCR.JK)                       (36)
N POP=POPN                                                (36.1)
C POPN=1000                                               (32.6)
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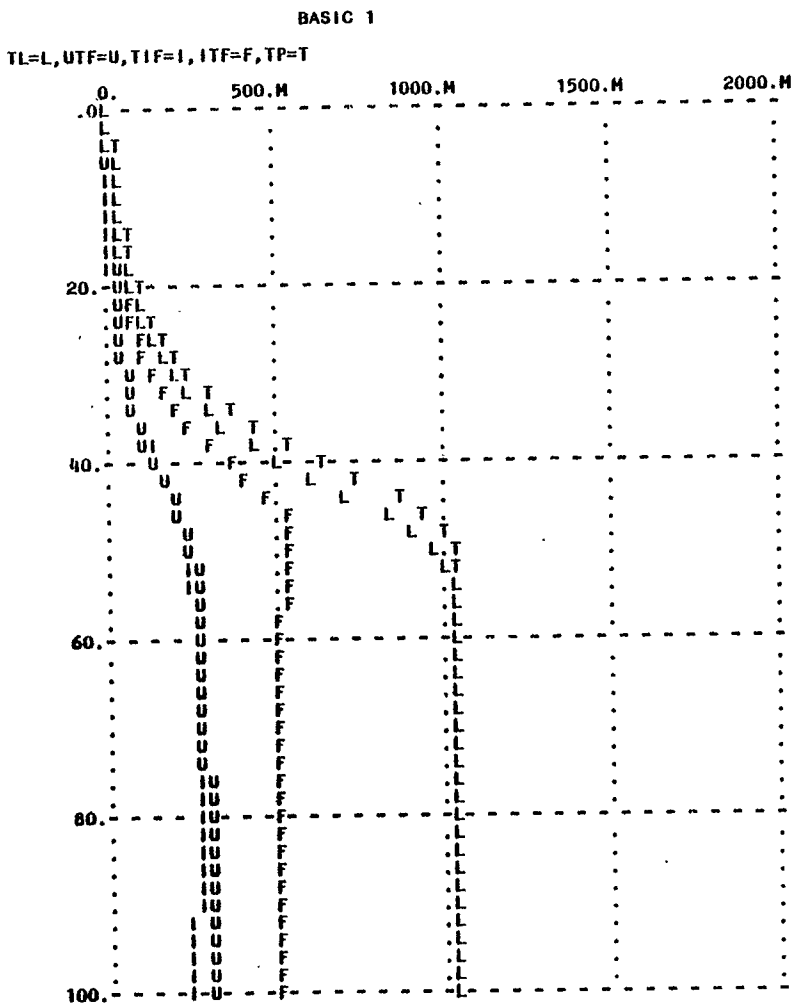
NOTE   POPULATION (PERSONS)
R NPGR.KL=POP.K*NPGF                                                    (37)
NOTE   NORMAL POP GROWTH RATE (PERSONS/WK)
C NPGF=.0005                                                            (37.1)
NOTE   NORMAL POP GROWTH FRACT (1/WK)
R IPGR.KL=MAX(0,(POPCC.K-POP.K)*IPCF)                                   (38)
NOTE   INDUCED POP CHANGE RATE (PERSONS/EK)
C IPCF=.05                                                              (38.1)
NOTE   INDUCED POP CHANGE FRACT (1/WK)
NOTE
NOTE
NOTE   CATTLE SECTOR
L UC.K=UC.J+(DT)(CBR.JK-CVR.JK-CER.JK-UCSR.JK+PER.JK+NCI.JK)            (39)
N UC=UCN                                                                (39.1)
C UCN=3000                                                              (39.2)
NOTE   UNINFECTED CATTLE (CATTLE)
R   NCI.KL=MAX(0,(CPCC.K-CP.K)*NCIF)                                    (40)
C NCIF=.01                                                              (40.1)
NOTE   NEW CATTLE INTRODUCED (CATTLE/WK)
NOTE   CATTLE POP CARRYING CAPACITY (CATTLE)
R CVR.KL=UC.K*FUV.K                                                     (41)
NOTE   CATTLE VACCINATION RATE (CATTLE/WK)
A FUV.K=PULSE(FV,VT,50)                                                 (42)
C VT=400                                                                (42.1)
C FV=.5                                                                 (42.2)
NOTE   FRACT UNINFECTED VACCINATED (1/WK)
R UCSR.KL=UC.K/LNC                                                      (43)
NOTE   UNINFECTED CATTLE SLAUGHTER RATE (CATTLE/WK)
C LNC=250                                                               (43.1)
NOTE   LIFETIME NORMAL CATTLE
R PER.KL=PC.K/PPER                                                      (44)
NOTE   PROPHYLACTIC EXPIRATION RATE (CATTLE/WK)
C PPER=25                                                               (44.1)
NOTE   PROPHYLACTIC PERIOD (WK)
R CBR.KL=CP.K*NCF*CFM.K                                                 (45)
NOTE   CATTLE BIRTH RATE (CATTLE/WK)
C NCF=.006                                                              (45.1)
NOTE   NORMAL CATTLE FERTILITY (1/WK)
R CER.KL=UC.K/TFIF.K                                                    (46)
NOTE   CATTLE EXPOSURE RATE (CATTLE/WK)
A TFIF.K=TFIFN*TFIFM.K*MIM.K                                            (47)
C TFIFN=20                                                              (47.1)
A TFIFM.K=TABLE(TFIFMT,FFI.K*TPR.K,0,3,.25)                             (48)
T TFIFMT=40/5/1/.7/.5/.4/.32/.26/.22/.19/.17/.16/.15                    (48.1)
NOTE   TIME TO FIND INFECTED FLY (WK)
A FFI.K=TABHL(FFIT,TIME.K,0,50,50)                                      (49)
T FFIT=.5/.5                                                            (49.1)
NOTE   FRACTION FLIES INFECTED (DIM)
A MIM.K=TABLE(MIMT,NDC.K/CP.K,0,1,.2)                                   (50)
T MIMT=1/1/1/1/1/1                                                      (50.1)
NOTE   MECHANICAL INFECTION MULT (DIM)
A   CPCC.K=RL.K/LRPC                                                    (51)
C   LRPC=2.0                                                            (51.1)
NOTE   CATTLE POPULATION CARRYING CAPACITY (CATTLE)
NOTE   LAND REQUIRED PER CATTLE (HA/CATTLE)
L TIC.K=TIC.J+(DT)(CER.JK-CIR.JK)                                       (52)
N TIC=TICN                                                              (52.1)
C TICN=300                                                              (52.2)
NOTE   TRYPANOSOME INCUBATING CATTLE (CATTLE)
R CIR.KL=TIC.K/CIT                                                      (53)
NOTE   CATTLE INFECTION RATE (CATTLE/WK)
C CIT=2                                                                 (53.1)
NOTE   CATTLE INCUBATION TIME (WK)
L NDC.K=NDC.J+(DT)(CIR.JK-DCSR.JK-CRR.JK)                               (54)
N NDC=NDCN                                                              (54.1)
C NDCN=6000                                                             (54.2)
NOTE   NAGANA-DISEASED CATTLE (CATTLE)
R DCSR.KL=NDC.K/LDC                                                     (55)
NOTE   DISEASED CATTLE SALUGHTER RATE (CATTLE/WK)
C LDC=200                                                               (55.1)
NOTE   LIFETIME DISEASED CATTLE (WK)
A CP.K=UC.K+TIC.K+NDC.K+PC.K                                            (56)
NOTE   CATTLE POPULATION (CATTLE)
A   FHI.K=(GP.K*FGI+NDC.K)/(GP.K+CP.K)                                  (57)
C   FGI=.5                                                              (57.1)
NOTE   FRACTION HOSTS INFECTED (DIM)
A CFM.K=TABLE(CFMT,NDC.K/CP.K,0,1,1)                                    (57)
T CFMT=1/.5                                                             (57.1)
NOTE   CATTLE FERTILITY MULT (DIM)
R CRR.KL=NDC.K/CRT                                                      (58)
NOTE   CATTLE RECOVERY RATE (CATTLE/WK)
C CRT=50                                                                (58.1)
NOTE   CATTLE RECOVERY TIME (WK)
L PC.K=PC.J+(DT)(CRR.JK+CVR.JK-PER.JK-PCDR.JK)                          (59)
N PC=PCN                                                                (59.1)
C PCN=2700                                                              (59.2)
NOTE   PREMUNIZED CATTLE (CATTLE)
R PCDR.KL=PC.K/LNC                                                      (60)
A TPR.K=TP.K/TPN                                                        (71)
NOTE   TSETSE POP RATIO (DIM)
NOTE   PREMUNIZED CATTLE DEATH RATE (CATTLE/WK)
A PDEN.K=POP.K/AREA                                                     (62)
C AREA=1000000                                                          (62.1)
NOTE   POPULATION DENSITY
A POPCC.K=(CP.K/CRPP)+(FL.K/LRPP)                                       (63)
NOTE   POPULATION (HUMAN) CARRYING CAPACITY (PERSONS)
C CRPP=10                                                               (63.1)
NOTE   CATTLE REQUIRED PER PERSON (CATTLE/PERSON)
C LRPP=0.4                                                              (63.2)
L FL.K=FL.J+(DT)(RFCR.JK)                                               (64)
N FL=FLN                                                                (64.1)
C FLN=100000                                                            (64.2)
NOTE   FARMLAND (HA)
R RFCR.KL=MAX(0,(POP.K-POPCC.K)*LRPP/CONVT)                             (65)
NOTE   RANGE-TO-FARM LAND CONVERSION RATE
C CONVT=20                                                              (65.1)
L RL.K=RL.J+(DT)(RFCR.JK)                                               (66)
N RL=RLN                                                                (66.1)
C RLN=700000                                                            (66.2)
NOTE   RANGELAND (HA)
L FLF.K=FLF.J-(DT)(FLFD.JK)                                             (67)
N FLF=FLFN                                                              (67.1)
C FLFN=600                                                              (67.2)
NOTE   FARMLAND FERTILITY (VEG-EQUIV-KG / HA-YR)
L RLF.K=RLF.J-(DT)(RFD.JK)                                              (68)
N RLF=RLFN                                                              (68.1)
C RLFN=600                                                              (68.2)
NOTE   RANGELAND FERTILITY (VEG-EQUIV-KG / HA-YR)
R FLFD.KL=FLF.K*POP.K/(POPCC.K*LFTC.K)                                  (69)
NOTE   FARMLAND FERTILITY DEGRADATION (VEG-EQUIV-KG/HA-YR-WK)
R RFD.KL=RLF.K*CP.K/(CPCC.K*LFTC.K)                                     (70)
NOTE   RANGELAND FERTILITY DEGRADATION (VEG-EQUIV-KG/HA-YR-WK)
A LFTC.K=LFTCN*POLM.K*EROM.K*DIM.K                                      (71)
C LFTCN=200                                                             (71.1)
NOTE   LAND FERTILITY TIME CONSTANT
A POLM.K=TABLE(POLMT,FIASPA.K,0,1,.5)                                   (72)
T POLMT=1/.9/.5                                                         (72.1)
NOTE   POLLUTION MULTIPLIER (DIM)
A EROM.K=TABLE(EROMT,FIACPW.K,0,1,.5)                                   (73)
T EROMT=1/.9/.5                                                         (73.1)
NOTE   EROSION MULTIPLIER (DIM)
```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1500.M
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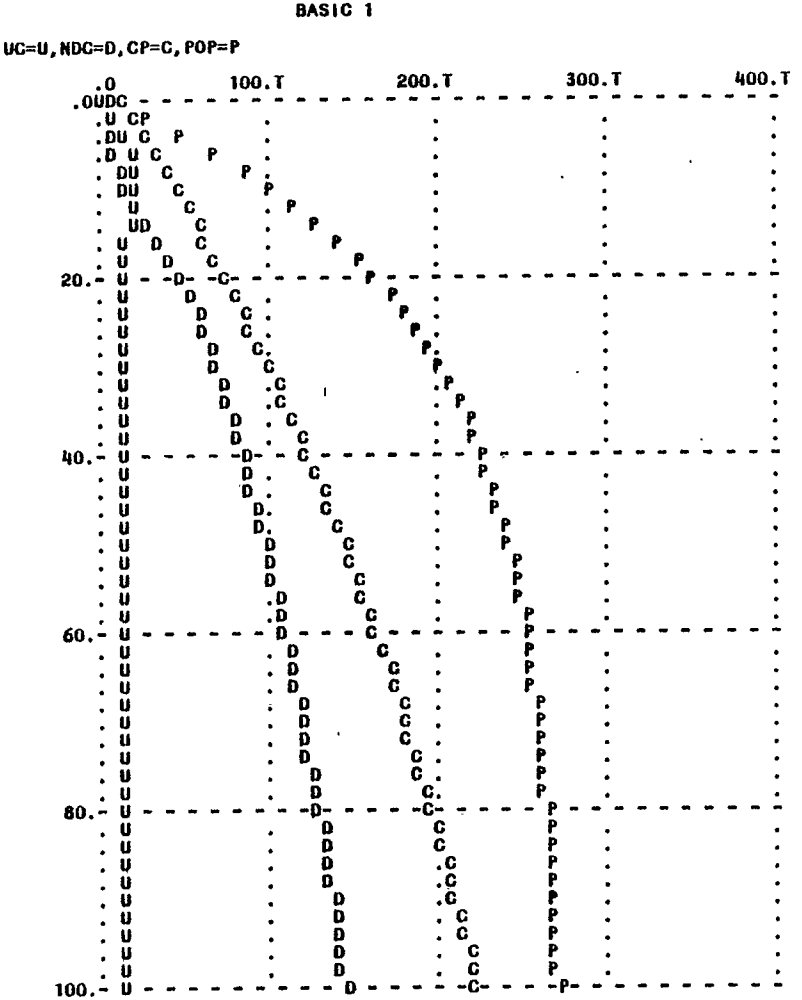
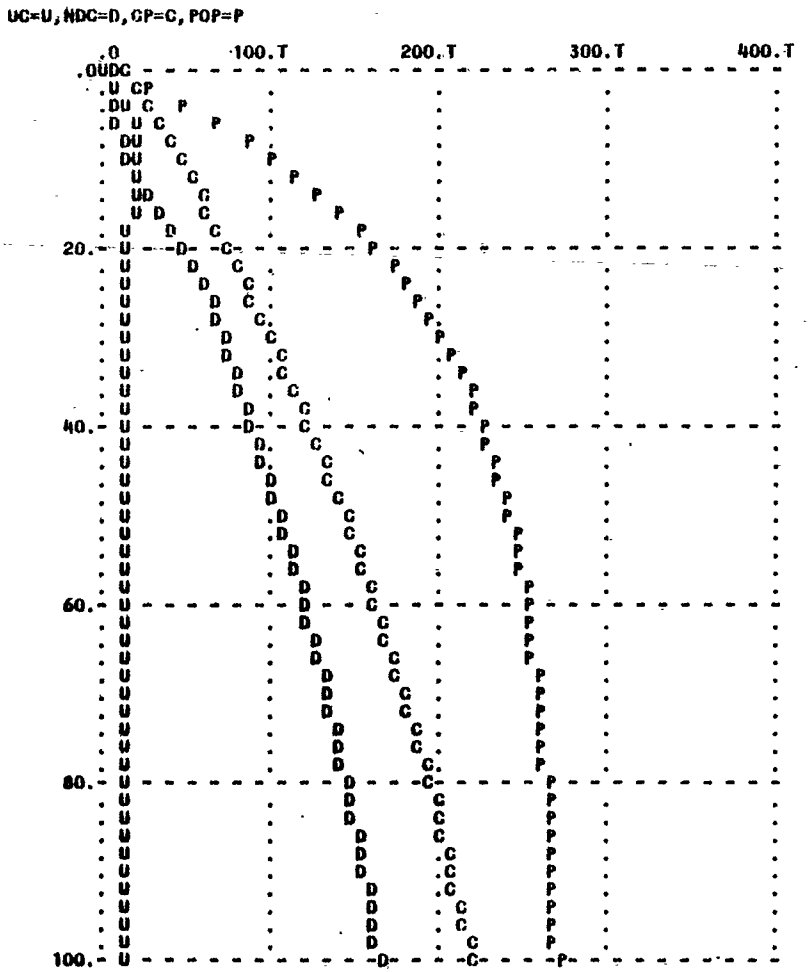
20.
40.
60.
80.
100.


BASIC 1
UC=U,NDC=D,CP=C,POP=P
.0
100.T
200.T
300.T
400.T
20.
40.
60.
80.
100.

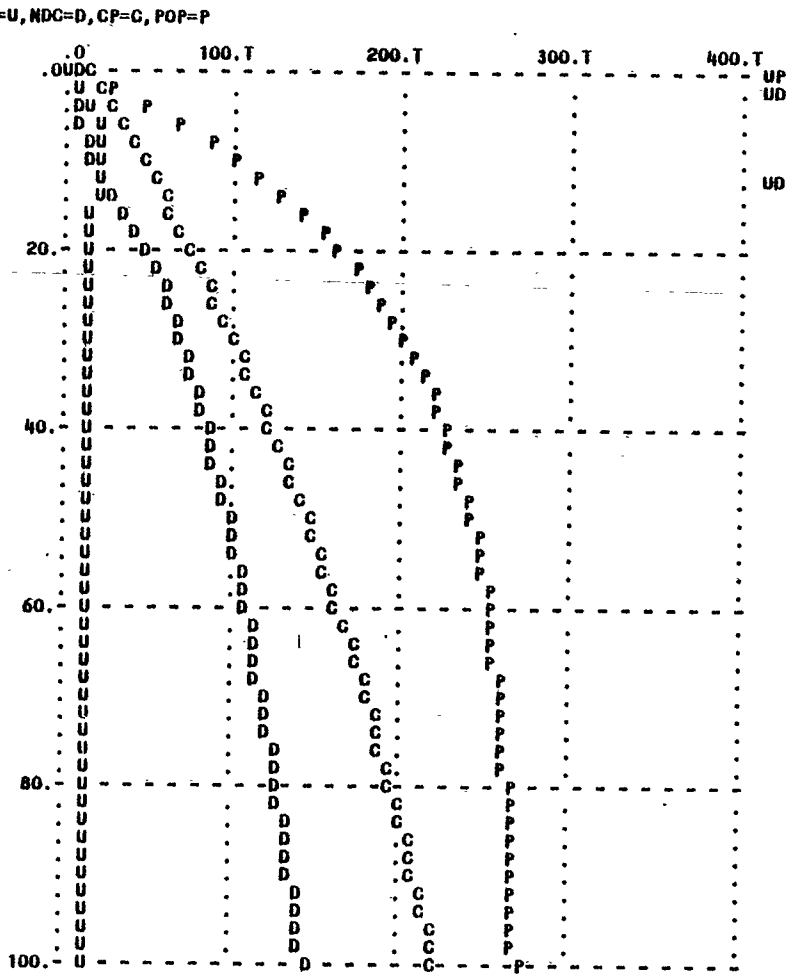
STRATEGY 1. CURATIVE DRUGS



C PPER=10
C FV=.3
C VT=50
RUN STRATEGY 1. CURATIVE DRUGS

	PPER	FV	VT
PRESENT	10.00	.3000	50.00
ORIGINAL	25.00	.5000	400.0

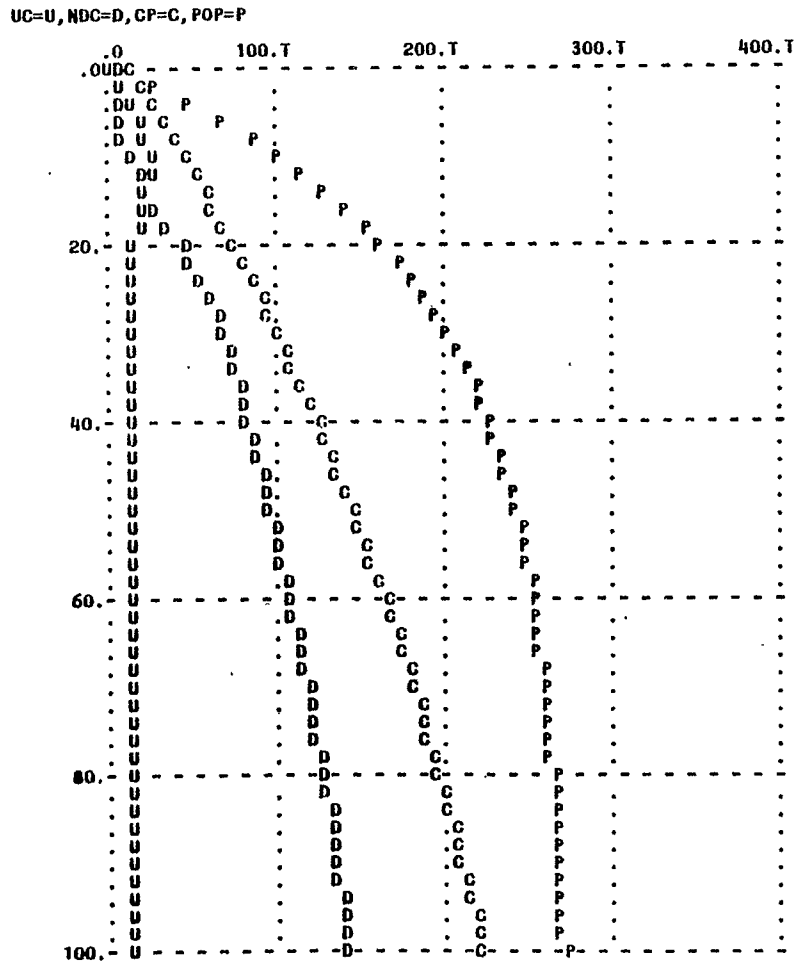
STRATEGY 2. IMMUNIZATION



C VT=50
RUN STRATEGY 2. IMMUNIZATION

	VT
PRESENT	50.00
ORIGINAL	400.0

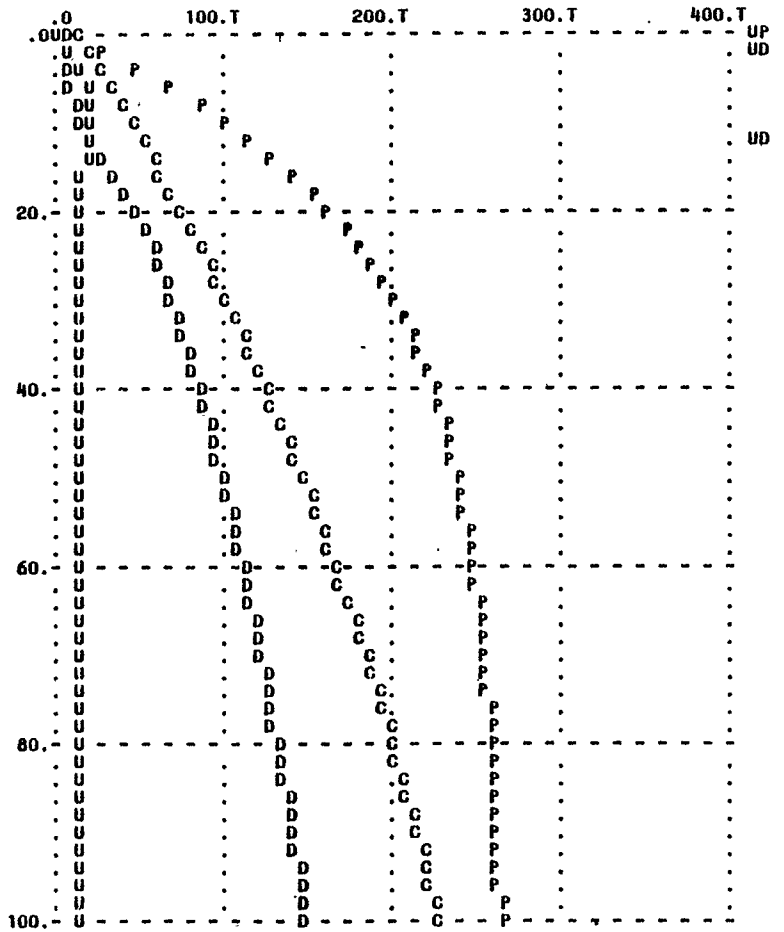
STRATEGY 3. AVOIDANCE



C TFIFN=25
RUN STRATEGY 3. AVOIDANCE

	TFIFN
PRESENT	25.00
ORIGINAL	20.00

STRATEGY 4. SUBSTITUTION

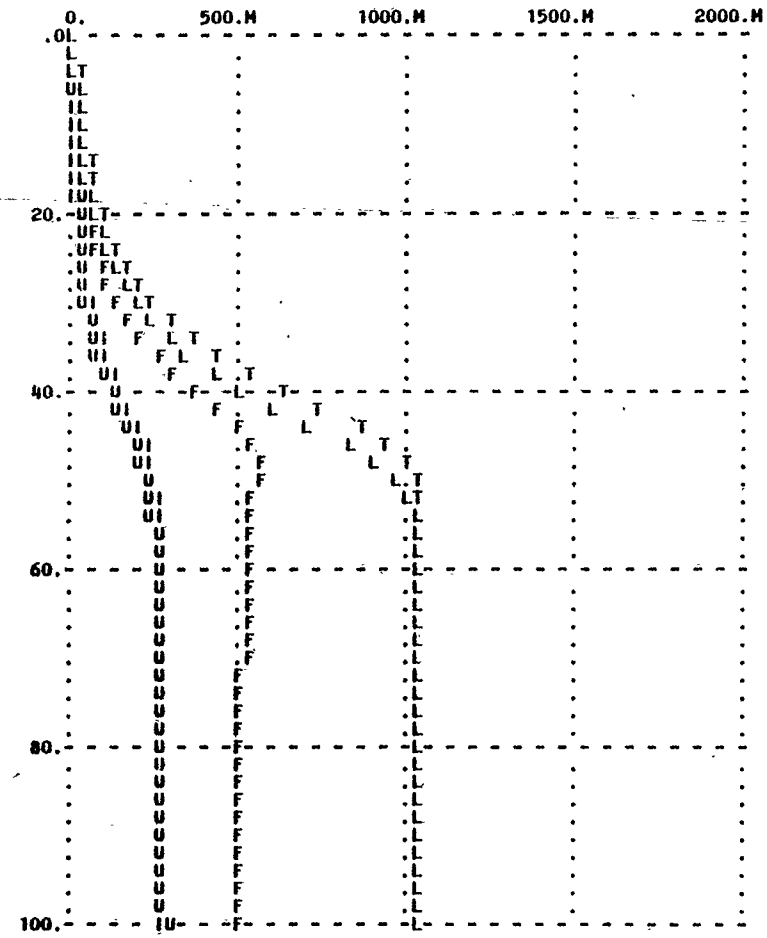


C LDC=250
C CRPP=15
RUN STRATEGY 4. SUBSTITUTION

	LDC	CRPP
PRESENT	250.0	15.00
ORIGINAL	200.0	10.00

STRATEGY 5. GAME REDUCTION

TL=L,UTF=U,TIF=I,ITF=F,TP=T

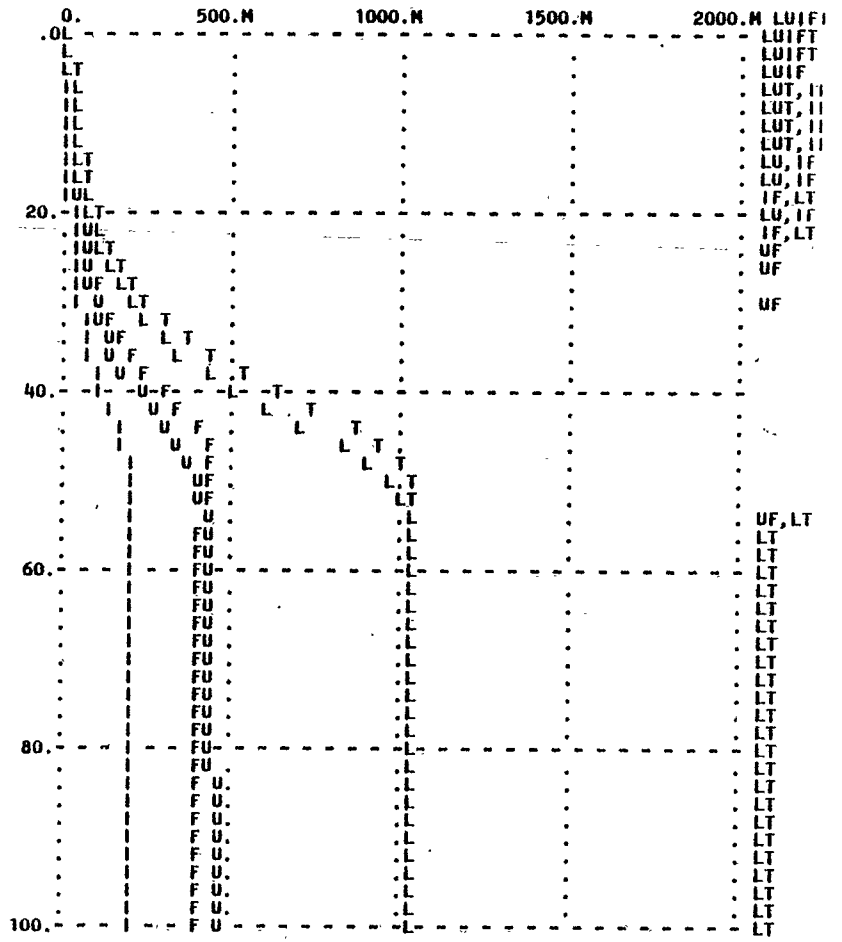


C GPRRS=480
RUN STRATEGY 5. GAME REDUCTION

	GPRRS
PRESENT	480.0
ORIGINAL	0.

STRATEGY 6. WILDLIFE UTILIZATION

TL=L,UTF=U,TIF=I,ITF=F,TP=T

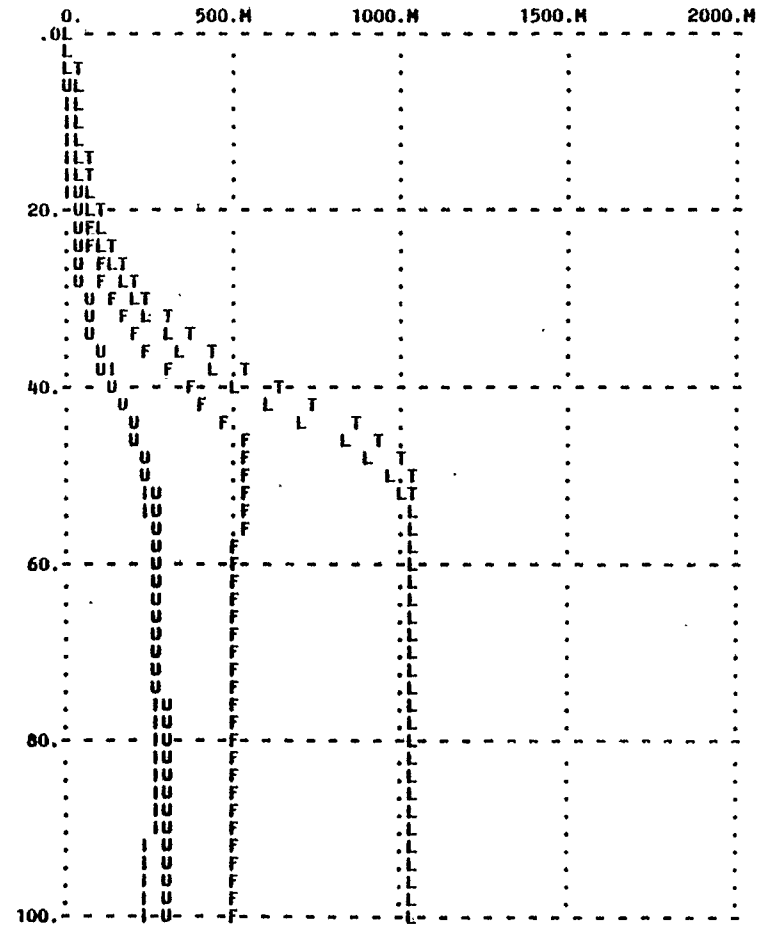


C TFIHN=10
RUN STRATEGY 6. WILDLIFE UTILIZATION

	TFIHN
PRESENT	10.00
ORIGINAL	5.000

STRATEGY 7. MECHANIZED BUSH CLEARING

IL=L,UTF=U,TIF=I,ITF=F,TP=T

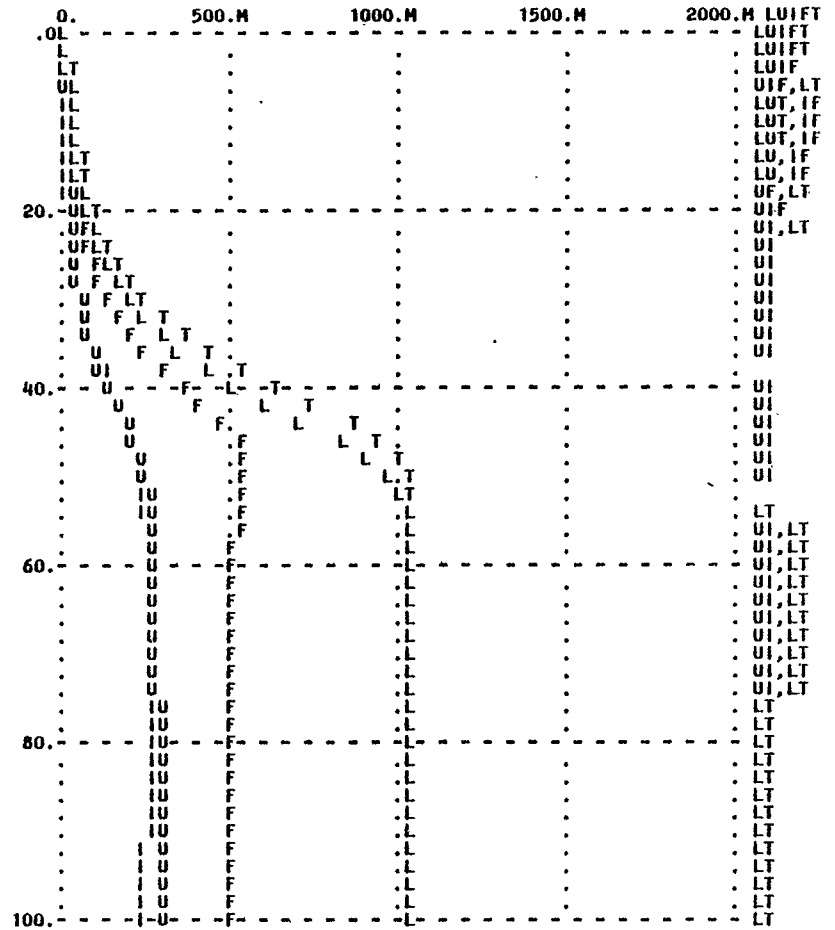


T BCIMT=1/1.2/1.5/2/2.7/3.7
RUN STRATEGY 7. MECHANIZED BUSH CLEARING

	BCIMT					
PRESENT	1.000	1.200	1.500	2.000	2.700	3.700
ORIGINAL	1.000	1.000	1.000	1.000	1.000	1.000

STRATEGY 8. BARRIER CLEARING

TL=L,UTF=U,TIF=I,ITF=F,TP=T

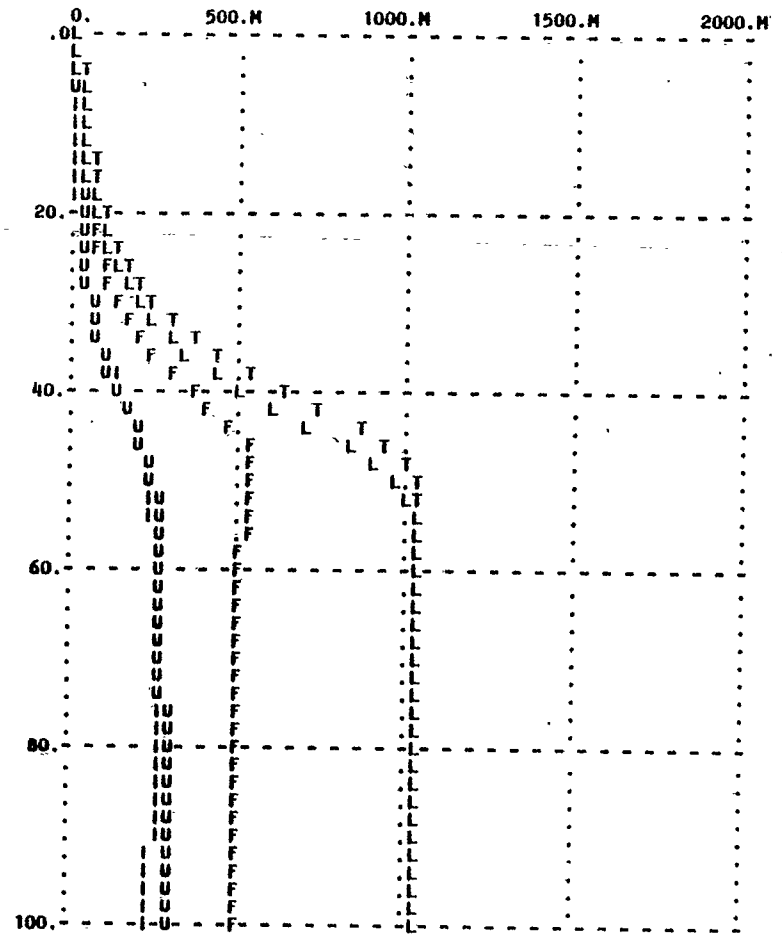


T BCIMT=1/1.1/1.25/1.45/1.7/2
RUN STRATEGY 8. BARRIER CLEARING

	BCIMT					
PRESENT	1.000	1.100	1.250	1.450	1.700	2.000
ORIGINAL	1.000	1.000	1.000	1.000	1.000	1.000

STRATEGY 9. SELECTIVE CLEARING

TL=L,UTF=U,TIF=I,ITF=F,TP=T

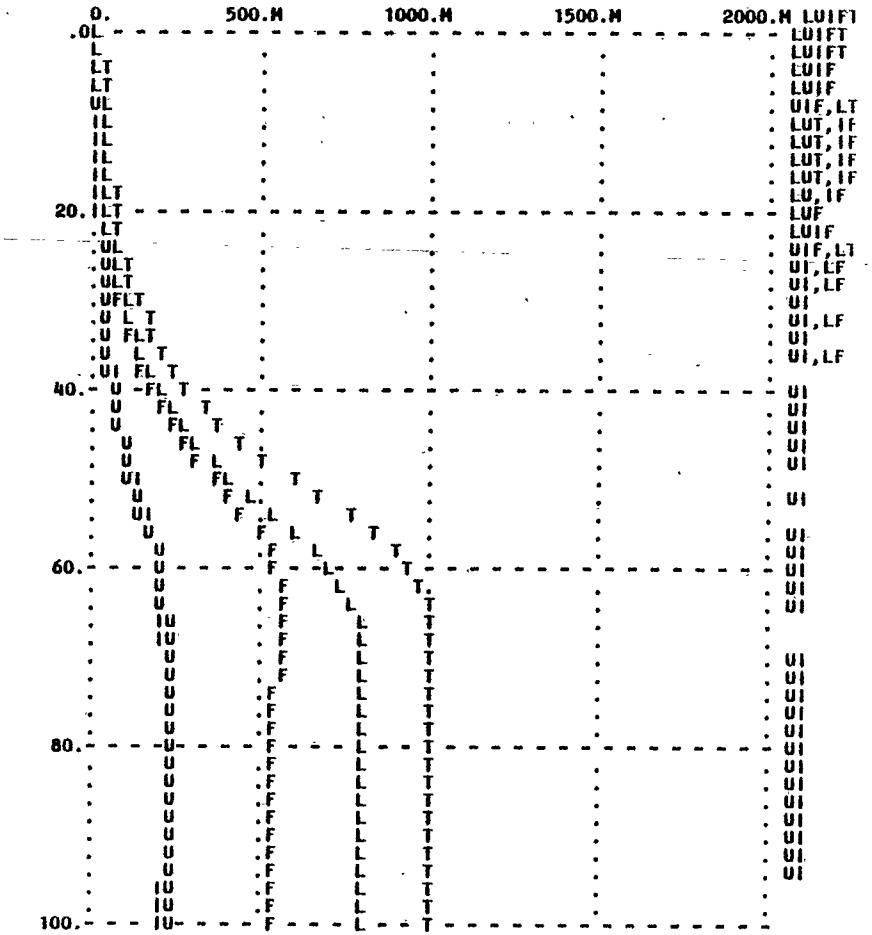


T BCIMT=1/1.05/1.12/1.22/1.35/1.5
RUN STRATEGY 9. SELECTIVE CLEARING

	BCIMT					
PRESENT	1.000	1.050	1.120	1.220	1.350	1.500
ORIGINAL	1.000	1.000	1.000	1.000	1.000	1.000

STRATEGY 10. BIOLOGICAL CONTROL

TL=L,UTF=U,TIF=I,ITF=F,TP=T

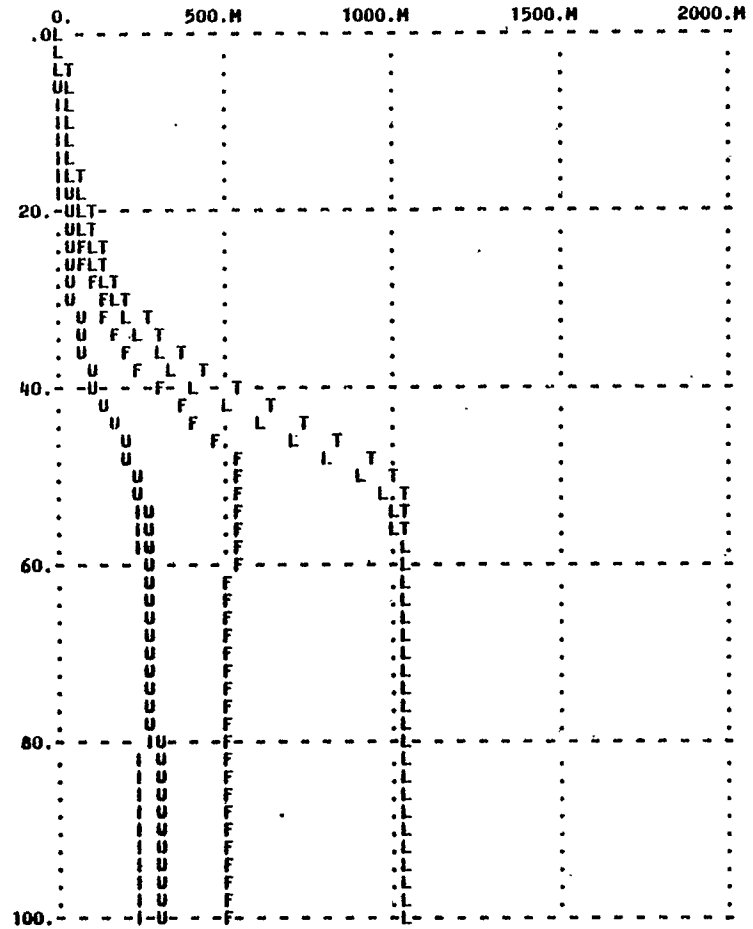


C FLDN=.3
RUN STRATEGY 10. BIOLOGICAL CONTROL

	FLDN
PRESENT	.3000
ORIGINAL	.2000

STRATEGY 11. LABORATORY STERILIZATION

TL=L,UTF=U,TIF=I,ITF=F,TP=T

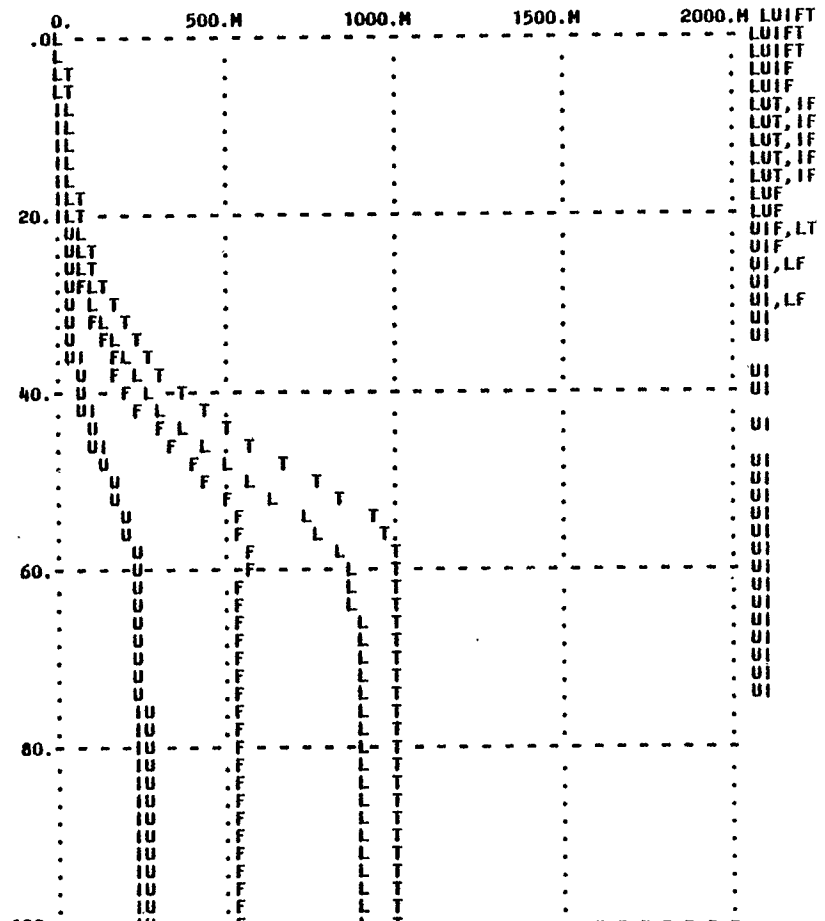


C LTCSN=4.4E6
RUN STRATEGY 11. LABORATORY STERILIZATION

	LTCSN
PRESENT	4400.T
ORIGINAL	0.

STRATEGY 12. FIELD STERILIZATION

TL=L,UTF=U,TIF=I,ITF=F,TP=T



C AFN=.35
RUN STRATEGY 12. FIELD STERILIZATION

	AFN
PRESENT	.3500
ORIGINAL	.4000