

A MATHEMATICAL MODEL OF
THE GEOGRAPHIC SPREAD
OF DISEASE

A Thesis
Presented to
the Faculty of the Department of
Industrial Engineering
University of Houston

In Partial Fulfillment
of the Requirements for the Degree
Master of Science

by
Frank M. Pokladnik
May 1972

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ABSTRACT

This thesis presents an original model of the geographic spread of disease. The model can represent local geographic conditions and can be easily modified. It can be used as an extension of any set of differential equations, either deterministic or stochastic, used to describe a population in an epidemic state. The classical Kermack and McKendrick equations are extended and numerical solutions are generated for various initial conditions. The results indicate an interesting directional effect and suggest the development of a user oriented computer program for the study and simulation of epidemics. Such a program might be used as a communications tool to bring together continuing advances in our knowledge of the causes of epidemic disease and the mathematical theory of epidemics.

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Chapter 1

INTRODUCTION

1.1 General Discussion

The benefits to be derived from the accurate modeling and prediction of contagious disease are numerous. The modeling procedure itself yields valuable insight into the biological processes involved. Public health planning will gain in effectiveness. Both agriculture and commerce may be improved. Our deteriorating environment makes it increasingly important to understand the ramifications of disease control actions such as vector eradication (25, p. 206).

For the above and other reasons (4, p. 237) and (11, p. 30), there is presently a large scale effort (10) under way in this area. While the possible benefits of the effort are substantial, the obstacles are formidable. The gathering and structuring of data (21), mathematical tractability (3, p. 174), and modeling of geographic spread (10, p. 508), and communications between biologists and mathematicians (29, p. 179) are often cited as stumbling blocks. Not only is data often incomplete, but the procedures for identifying a particular disease vary over time. Multiple causes of death and failure to detect mild cases of infection also complicate interpreting records. It is seldom practical to

solve the sets of simultaneous deterministic differential equations involved, though theoretically solvable, except by numerical analysis. In the case of most sets of simultaneous stochastic differential equations, Monte Carlo simulation is the only known means of solution. The problems of data structuring and mathematical tractability combine with the complexity of human populations (29) to make the modeling of spacial dispersion difficult. A communications problem, similar to the one between biologists and mathematicians, also exists between other highly specialized parties such as geneticists, population modelers, and operations researchers. Technical jargon and differences in fundamental approaches to problems are partly responsible. With these general problems in mind, the following research objectives were developed.

1.2 Research Objectives

- A. To develop a mathematical model of the spacial spread of contagious disease which:
 - 1 can represent local geographic conditions
 - 2 has a flexible data structure
 - 3 is compatible with as many of the current mathematical models as possible.
- B. To write a Fortran program for the numerical solution of the model which:

- 1 can be used to investigate the feasibility of a user oriented computer language for the modeling and prediction of epidemic disease.
- 2 can readily be expanded to include such features as Monte Carlo simulation and graphic display.

1.3 Epidemiological Principles

This section is intended to aid those without a background in epidemiological mathematics. The terminology used is mathematical in nature and should not be confused with the terms used in epidemiological field work. Though condensed, hopefully it will be of help to some in reading this thesis. For a more complete account see (21) and (3).

At least two populations (species) are involved in any infectious disease; the host population, which is invaded, and the population of causative organisms. In some cases, a third population acts as a carrier or vector. In malaria, for example, the *Anopheles* mosquito, the vector, carries *Plasmodium* protozoa, the causative organism, from man to man, the host. More complicated relationships, with multiple host species and secondary invaders, are known but are beyond the scope of this discussion. What is important to note is that conditions affecting any one of the populations involved in a particular disease cycle will indirectly

affect all the other populations.

When mathematically modeling the spread of contagious disease, some of the involved populations are not represented explicitly as variables. They are hidden in constants such as the infection rate (see figure 2.4.1). There are manifold pitfalls in such simplifications, but lack of data and mathematical tractability frequently necessitate them. Often the host population is the only population considered.

Given that there are only two populations, host and causative agent, it can be assumed that infective members of the host population are the only sources of infection. Given that the latent period, the time between infection of an individual host and when that individual becomes infectious to others, is approximately zero, the host population can be divided into three groups. When mentioned in this paragraph, diseased individuals include both clinically manifest and laboratory confirmed cases of infection. Susceptibles are free of the disease and do not have sufficient immunity to repel the invaders. Active infectives have the disease and, provided there is adequate contact with susceptibles, will pass on the disease. The remaining population is neither susceptible nor infective due to previously acquired immunity or isolation.

The assumptions and definitions in the above

paragraph are used in the classical deterministic model of infection and removal known as the Kermack and McKendrick equations (18). This model is presented in Chapter 2 and is extended to a geographic model in Chapter 3.

In this thesis, the epidemic curve is defined as the rate at which new infectives are created. The incubation period, the time between infection and the first visible sign of disease, does not modify the shape of the epidemic curve as defined. In field work the epidemic curve is the rate at which new clinical cases are reported and is modified by the incubation period. Typically, a field curve (see figure 1.3.1) is bell shaped and slightly skewed to the right (3, p. 28) and (26, p. 149).

The mathematical description of the population interactions involved in infectious disease is called the mathematical theory of epidemics. In deterministic theory the populations and their interaction rates are considered as real numbers. In stochastic theory they are considered random variables with either discrete or continuous probability distributions. The current trend is to view deterministic models as meaningful approximations to the more complex stochastic models. The thesis model is deterministic, but is compatible with stochastic formulation.

1.4 Chapter Summaries

At the beginning of Chapter 2 the reader may choose

THE EPIDEMIC CURVE

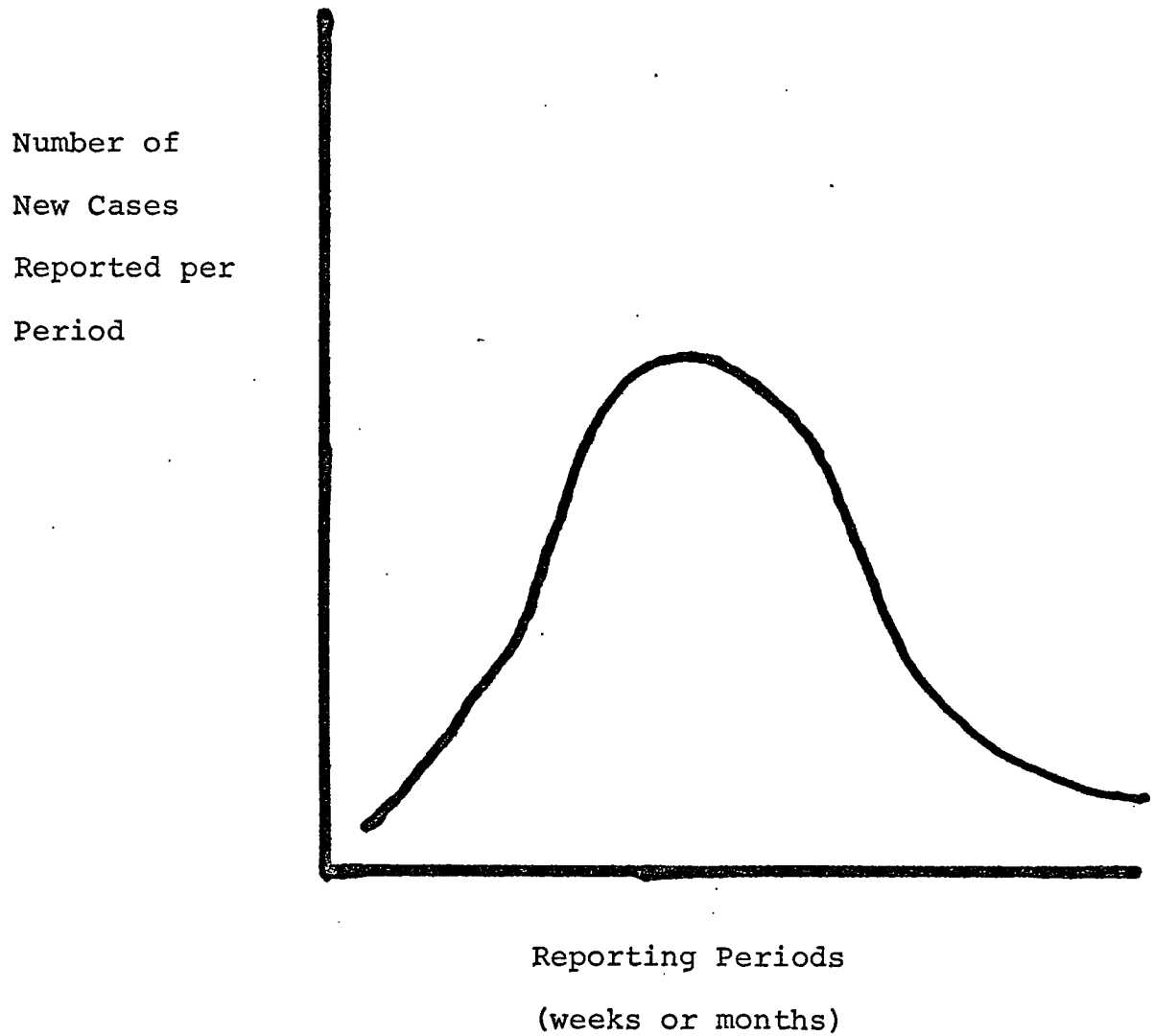


Figure 1.3.1

in which of two ways he wishes to read this thesis. He may choose to read it in the order in which it is arranged. In this case, he will find that Chapter 2 places the mathematical modeling of epidemic disease in an historical framework. Chapter 3 develops the thesis model of the geographic spread of disease in first an informal and then a formal manner. Then Chapter 4 explains the computer program used to numerically solve the thesis model and the significance of the solutions of the ten problems found in appendix C. Chapter 5 summarizes the work done. It then draws conclusions and makes recommendations for further work.

An alternate path is to make a first reading of only those passages which are necessary to comprehend the thesis model. For this purpose, section 2.4, 3.1, 3.2, 4.2 and 5.1 are needed.

Chapter 2

HISTORICAL OVERVIEW

2.1 Introduction

The study of epidemic disease can be divided into three idealized states or eras. The first and longest era was qualitative and empirical in nature. The second era, in which we are still actively engaged, is also qualitative in nature but is based on the scientific principles set forth by men like Koch and Pasteur. The third era, which is just beginning, is earmarked by the mathematical modeling and prediction of the phenomena. The three eras overlap to some degree.

2.2 Empirical Era

During the first era, some practical and effective measures were developed to fight infectious disease. Since there was an absence of modern scientific methodology, there was a heavy reliance on empirical data collection.

Millenniums before the birth of Christ, the Chinese were attempting to inoculate children against smallpox. Although not always successful, they tried to induce mild attacks of small pox as protection against severe attacks. This was done by placing the scabs from infectives in the nostrils of children or by placing the clothes from

infectives on children.

In the fifth century B.C., the Greek historian Thucydides wrote about immunity to the plague, "A person who transmits the disease is already out of danger. For the disease does not recur twice, at least not with a severity sufficient to cause death (11, p. 8)." ((The ancient practice of allowing only those who had recovered from plague to care for plague victims is akin to the present practice of allowing only those medical personnel who show a positive test for *Mycobacterium tuberculosis* to work in tubercular wards.))

Like the concept of inoculation, the concept of isolation of infectives was recognized centuries ago. The Biblical passages dealing with the isolation and persecution of lepers are well known.

During the Smallpox epidemic of 1776, the English physician Jenner observed that milkmaids who had contracted cowpox were not susceptible to Smallpox. He used this observation as the basis for the first effective immunization program. ((Pasteur later explained this phenomena in terms of the Germ Theory of Disease and named it vaccination (Latin *vaccinus*: pertaining to a cow *vacca*: cow) in recognition of Jenner's work.))

As can be seen from the above examples, a lack of knowledge of the causative agents of disease did not

exclude the possibility of limited prophylaxis.

2.3 Microbiological Era

Man entered the second era of the study of epidemic disease with a startling explosion of knowledge in the field of microbiology. The last fifty years of the nineteenth century saw proof of the germ theory of disease and the identification of the causative agents of all the major infectious diseases (11, p. 14). The work of men like Robert Koch and Luis Pasteur proved the everyday importance of the animacules, bacteria and protozoa, described by Antony von Leeuwenhoek (1623-1723).

Robert Koch (1843-1910) was the first to apply his own postulates which set the criteria for demonstrating the etiology of a disease. In 1882, he isolated *Bacillus anthrax* from a sick animal, grew it in a pure culture, infected a healthy animal with it, and then reisolated the infective organism. Within a year, he had done the same thing for the organisms which caused tuberculosis and cholera.

Pasteur, considered less theoretical than Koch, made scientific contributions of a practical nature over a wide range of areas. Among his important discoveries relating to the control of infectious disease is the use of attenuated pathogens to produce immunity against more virulent pathogens of an antigenically related strain. In addition to this procedure, termed vaccination, he developed

sterilization and pasteurization.

These advances lead to an immediate improvement in public health. Among the diseases brought to bay were Black Death which destroyed one-fourth of Europe's population in the fourteenth century and smallpox which ravaged millions of central Americans in the sixteenth century (3, p. 1). In addition, these advances opened the door for the mathematical theory of epidemics.

2.4 Quantitative Era

The transition from the Qualitative Era to the Quantitative Era is a slow process which has already begun. There are no indications that it will suddenly burst forth with a surge of knowledge as did the Microbiological Era. Instead, it is growing slowly on huge banks of data and sophisticated statistical techniques.

The successful modeling of any contagious disease requires an understanding of the underlying biological processes. We presently have a clear picture of how many bacterial infections and some viral infections are transmitted. Yet for reasons stated in the section entitled General Discussion, no disease has been accurately modeled and predicted.

Despite the intricacy and variability of epidemic disease real progress has been made in the mathematical theory of epidemics. Representative examples of some of

the earlier steps taken are the works of Hammer in 1906 (15), Ross in 1911 (26), Kermack and McKendrick in 1927 (18), and Reed and Frost in 1928 (34). Bartlett's 1957 model of measles periodicity (5) is representative of modern efforts which use a truly stochastic approach solved by computer techniques. Comprehensive historical reviews are found in (3) and (28). While (10) is an excellent survey of the work currently underway.

In 1906, Hammer proposed a proportionality which is fundamental to all subsequent deterministic models (3, p. 8). This proportionality is reformulated for use in probabilistic models as well. He assumed the epidemic curve to be a function of the number of susceptibles, the number of infectives, and the contact rate between infectives and susceptibles. By allowing the gradual introduction of new susceptibles, this hypothesis generates periodic bell shaped curves which are left skewed (28, p. 149).

Ross developed a difference equation for the portion of a local human population affected by malaria in 1911. The work was based on his own field observations. It included established epidemiological characteristics of the host and vector populations involved. His rough estimates of the parameters involved enabled him to make practical application of the equation. Even though the model involved some probability concepts, it is deterministic in nature.

Given the population levels in one period, the theoretical levels for the following period can be calculated exactly.

In 1927, Kermack and McKendrick (K & K) expressed the important threshold theorem of their deterministic model (see p. 4). The theorem is based on a set of three differential equations found in figure 2.4.1. The threshold theorem is explained in terms of R , the relative removal rate over the infection rate. When the number of susceptibles in a population, X , is less than or equal to R , an epidemic outbreak is not possible. When X is greater than R , the intensity of the epidemic or the percentage of susceptibles eventually affected, is a non-linear function of the ratio of X over R . A table of this relationship is available in (3, p. 28). The table is based on the exact solution of the K & K equations by Kendall in 1956 (17). K & K used an approximate solution in their work.

In 1928, Lowell J. Reed and Wade Hampton Frost were discussing and lecturing on a truly stochastic model of the spread of disease in closed populations subject to random mixing. These men used a mechanical model for teaching purposes at John Hopkins Medical School in Baltimore, Maryland. The mechanical Reed-Frost model consisted of four sets of balls, each of a different color, a large bowl, and a narrow trough. Each set of balls represented infectives, susceptibles, immunes, or a nonspecified blocking agent. An initial

KERMACK AND MC KENDRICK MODEL
OF INFECTION AND REMOVAL

$$\frac{dx}{dt} = Bxy$$

$$\frac{dy}{dt} = Bxy - Gy$$

$$\frac{dz}{dt} = Gy$$

Where:

$$x + y + z = n$$

x Susceptibles

y Infectives in circulation

z Isolated, dead, or immune

n Community size

B Infection rate

G Removal rate

R = G/B relative removal rate

Figure 2.4.1

population was specified and counted out into the bowl, was stirred, and then was poured into a trough where they formed a long single line. The susceptibles having adequate contact with one or more infectives were replaced by infectives. A susceptible was said to have adequate contact with an infective if there were no intervening blocks in the line. Infectives were replaced by immunes and the mixing was performed again. The procedure was repeated until there were no infectives remaining.

While the problem of multiple exposures was ignored by Hammer, Frost recognized the situation and it was taken into account in the Reed-Frost model. Their definition of adequate contact created only one new case when a single susceptible was exposed to several infectives. Another property of the Reed-Frost model is that given a small number of original infectives in a specified population, the portion of susceptibles eventually affected on different runs may vary from zero to one hundred percent. The Reed-Frost model assumes a constant incubation period and an infectious period so short that it can be represented by a single point in the disease cycle. The original infective generation is spontaneous and produces new infectives in stages at set periods of time equal to the incubation period. For certain common diseases such as measles, chickenpox, and mumps, a point infection model may be more appropriate

than a continuous infection model like the K & K equations (3, p. 75). Chain binomial theory is applicable to the mechanical Reed-Frost model and specifies the probabilities of all the possible states the population can pass through but cannot predict the states through which it will pass or the final state of the population.

Bartlett's 1957 model of measles periodicity and community size is mentioned here so that comparison can be made with it when the thesis model is presented. Bartlett constructed a six by six grid of square cells or wards which represented a city. The grid was used to idealize the spatial dispersion of a city. The total population of a city was divided equally among the thirty-six wards. The population in each cell was controlled by a set of stochastic differential equations derived from the deterministic Hammer-Soper model (30). Susceptibles were not allowed to move from ward to ward. Infectives were allowed to disperse or migrate from an infected ward to contiguous wards at a prescribed migration rate.

Bartlett applied this grid model to nineteen towns in England and Wales in an attempt to explain the relationship of the average period between measles epidemics and community size. In doing so, two observations of importance to this thesis were made. First, Bartlett noted that a town geographically near one or more other towns behaved as if its population was larger than towns of equal size which

were geographically isolated. Secondly, she found that small towns near urban centers tended to mimic the epidemic behavior' of the larger population mass.

The current trend in the mathematical modeling of contagious disease is to view deterministic models as useful approximations. With the increasing use of electronic computers, the more realistic stochastic models are becoming comprehensive and more complicated.

Chapter 3

MODEL PRESENTATION

3.1 Development of Model Concept

The thesis model for the geographic spread of disease is presented in this section. The thesis model is referred to as "NET" for convenience and because of the similarity between its structure and the structure used in network theory. First, there is a discussion of closed populations and random mixing as they relate to the topological factor in the spread of disease. Then, NET is presented in an informal manner. Finally, NET is described formally.

There is presently a great deal of epidemic theory dealing with closed populations, especially those which undergo random or homogeneous mixing. A closed population allows neither immigration or emigration of individuals. Homogeneous mixing provides each individual equal contact or an equal probability of contact with all other members of the population. Obviously, there are no closed systems nor is homogeneous mixing possible over long periods of time. Nevertheless, theorems such as Kermack and McKendrick's threshold theorem based on these assumptions have valuable predictive power when dealing with actual epidemic phenomena. The nineteen towns analyzed by Bartlett are examples of

approximately closed systems. The political boundaries of a city or the mountain ranges surrounding a valley are typical boundaries used to define approximately closed populations. Modeling the spacial dispersion of contagious disease can be viewed as slackening the restrictions of closed populations and random mixing. The presence of a relatively large population center near a community being studied undermines the assumption of a closed population. Both physical and social barriers weaken the assumption of random mixing.

The thesis model, NET, is a procedure for linking approximately closed populations together. The linking further reduces the closed property of the individual communities, but does not invalidate the techniques used to analyze closed populations. The geographic division under study is broken into AREAs with each containing one community. The size of an AREA varies depending on the size of the geographic division studied and the amount of detail desired. An AREA can represent a section of a city, an urban center in a nation, or a coastal region of a continent. An informal explanation of NET is given below.

The example of a group of cities is used because of the interest in human populations and the increasing urbanization of the world. For simplicity, only the human population is considered. The population of each city is estimated from records or assigned in the case of theoretical

work. Individuals are then separated into suitable categories such as infectives, susceptibles, and immunes. If a second population was of interest, say mosquitoes, it could be classified into carriers and non-carriers. These categories are the variables used in any of the available models of epidemic disease in closed populations with random mixing.

Before linking the cities together, we must define the term effective population. The tendencies of geographically associated communities to mimic the epidemic behavior of one another and to behave as if they had larger populations than they actually do, have already been cited. These tendencies motivate the model definition of effective population.

The effective population of a city is based on its actual population, the actual population of its neighbor cities, and the degree of interaction between the city and its neighbors. Neighbor cities are those which have significant interaction (8) and (24) with the city in question. Each city has its own set of neighbors. The degree to which city A is affected by city B is called the permeability of A from B. Permeabilities are real numbers with a range from zero to one. The effective population of a city is its actual population plus a portion of each of its neighbor populations. This portion of a neighbor population is the product of the actual neighbor population times the permeability

of the city from its neighbor. An effective population category is defined in a like manner. An effective population category of a city is the sum of its actual population category plus a portion of the same population category from each of its neighbor cities.

The manner in which NET links the cities together is simple and flexible. Effective population variables are substituted for the actual population variables used to model the cities as closed populations. The actual variables are maintained but are updated by effective information. The proper substitution of effective variables for actual variables depends on the underlying model used for each city. A specific example is given in the next section. NET is directly applicable to any group of closed populations for which each individual population can be modeled by a set of difference or differential equations.

The remainder of this section formally defines NET. The formal presentation progresses in much the same manner as the informal presentation did. The terminology developed is general in nature and is the same as that used in the computer program in appendix A.

NET is a generalized procedure and it requires an underlying epidemiological model and an idealized geographic division before it can be applied. Each NET application may be thought of as an independent model. The reader may choose

to skip over to the next section and use the remaining material in this section as a reference only.

NET models the spacial spread of contagious disease as the interaction of approximately closed epidemiological communities. The geographic division under study is broken down into AREAs which contain one community. The populations (species) to be studied are broken down into categories of epidemiological interest and are the variables in the sets of differential equations used to describe each AREA. The total number of individuals in any one population segment is referred to as a TPOP. Each so-called total population, TPOP (although a convenient convention), represents only a segment of a population in the strict biological sense. $TPOP_1$ may represent the total number of humans susceptible to measles in the NET. The number of individuals from a particular TPOP in each AREA is referred to as an area population or APOP. $APOP_{2,1}$ may represent the number of humans susceptible to measles in $AREA_2$.

Before linking the AREAs together, we must define the term effective APOP. An effective APOP is based on its actual APOP, the actual APOPs of its neighbors, and the degree of interaction between the APOP and its neighbor APOPs. Neighbor AREAs, NEIBs, are those which have significant interaction with the AREA in question. Each AREA has its own set of NEIBs. All APOPs in an AREA are assumed to interact

with the APOPs of a NEIB to the same extent. For this reason only one measure of the effect of a NEIB on an AREA is required. The degree to which an AREA is affected by a NEIB is called the permeability of the AREA from NEIB. Permeabilities, PERMs, are real numbers with a range from zero to one. An effective APOP is the actual APOP plus a portion of each of its neighbor APOPs. This portion of a neighbor APOP is the product of the actual neighbor APOP times the PERM of the AREA from its NEIB.

Once the proper algorithms or equations to calculate effective APOPs are set up, the manner in which NET links AREAs together is simple and flexible. Effective APOPs are substituted for the actual APOPs used as variables to model the approximately closed AREAs. The actual APOPs are retained, but are updated by effective information. The proper substitution of effective APOPs for actual APOPs depends on the underlying model used for each AREA. The program in appendix A is an example of the application of NET to the K & K equations.

3.2 Application to K & K Equations

In this section, NET is applied to two idealized geographic divisions. In both examples, the AREAs, or approximately closed populations, are controlled by the classical K & K equations. Each AREA creates three expanded K & K equations which must be solved simultaneously with all the

other equations from a NET. Note that NET has become an approximately closed system.

The first geographic division, Linear, is a linear arrangement of six AREAs. Each AREA is represented by a square in a line (see data set 2). Only adjacent AREAs interact. The four interior AREAs interact with two other AREAs. The exterior or terminal AREAs interact with only one other AREA. A schematic of Linear and the resulting simultaneous differential equations are illustrated in figure 3.2.1.

The second geographic division, Cubic, is a cubic arrangement of six AREAs. Each AREA is represented by one side of a three dimensional cube (see data set 8). Only AREAs with a common edge interact. Each AREA interacts with four other AREAs. A schematic of Cubic and the resulting differential equations are illustrated in figure 3.2.2.

NET EXTENSION OF K & K EQUATIONS
FOR A LINEAR GEOGRAPHIC
DIVISION

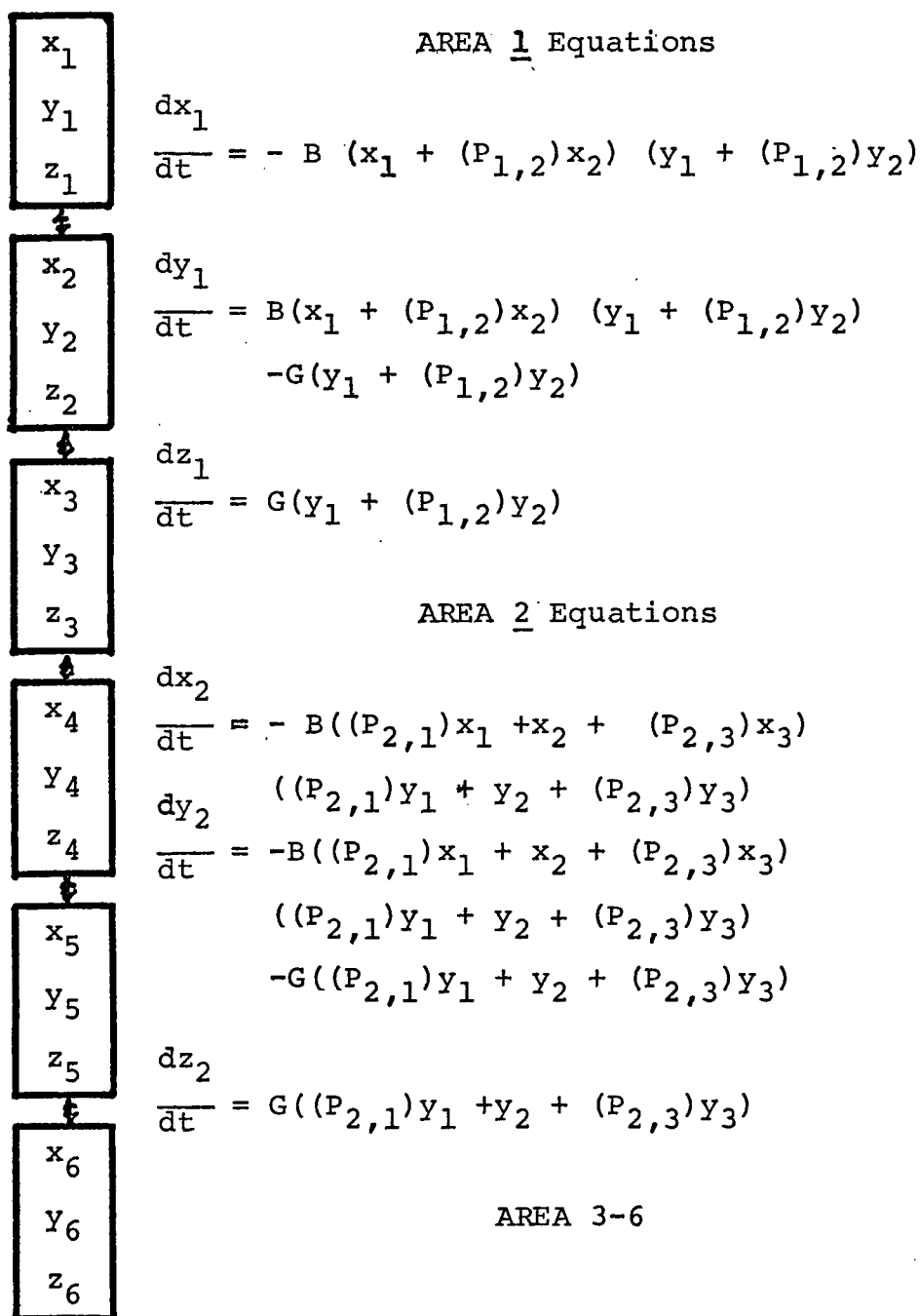
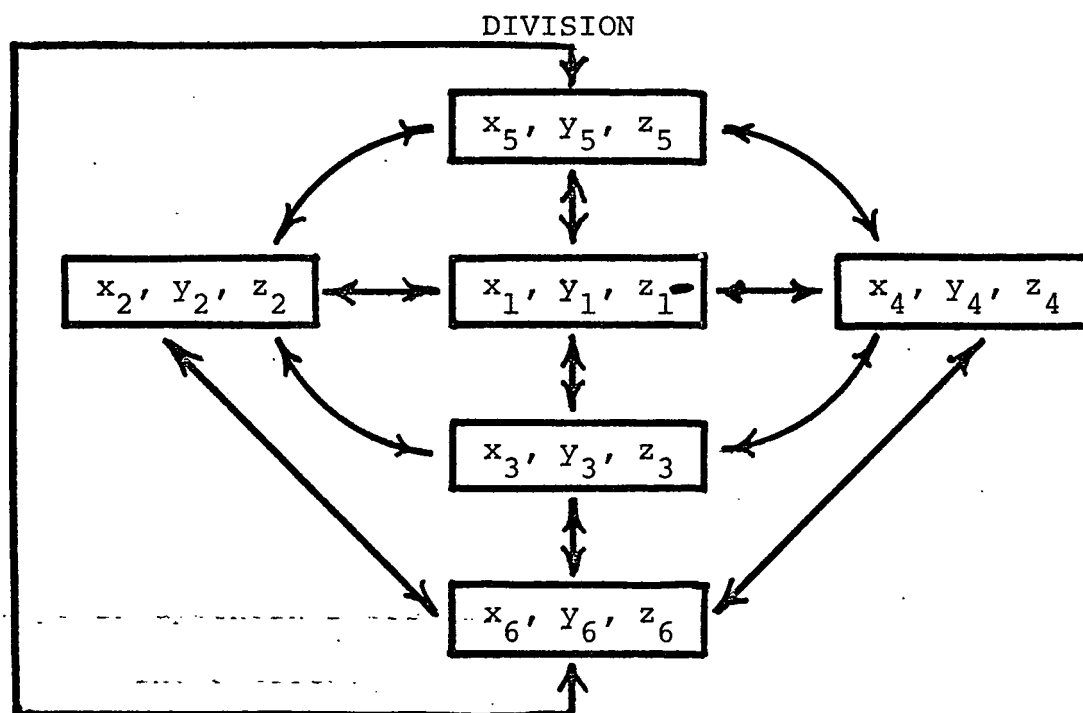


Figure 3.2.1

NET EXTENSION OF K & K EQUATIONS
FOR A CUBIC GEOGRAPHIC



$$\frac{dx_1}{dt} = B(x_1 + x_2 + x_3 + x_4 + x_5)(y_1 + y_2 + y_3 + y_4 + y_5)$$

$$\frac{dy_1}{dt} = B(x_1 + x_2 + x_3 + x_4 + x_5)(y_1 + y_2 + y_3 + y_4 + y_5) - G(y_1 + y_2 + y_3 + y_4 + y_5)$$

$$\frac{dz_1}{dt} = G(y_1 + y_2 + y_3 + y_4 + y_5)$$

*The constant permeability factors have been omitted in this illustration.

Figure 3.2.2

Chapter 4

MODEL EVALUATION

4.1 Programming for numerical analysis

The advantages of a numerical analysis of NET are stated in the beginning of this section. Then, the compatibility of NET to a user oriented program is mentioned. Finally, the Fortran program in appendix A for the numerical solution of NET is discussed. At this point, I would like to thank Mr. Marvin B. Smith of the University of Houston for the use of his plot routines which made the graphic displays in appendix B possible.

The application of NET to the Linear and the Cubic geographic divisions produced two sets of simultaneous differential equations. Both sets are theoretically solvable in closed form by the method used in (17). However, it is not practical to solve them in this manner. The eventual solution would be unyieldy. A minor change in the structure of either geographic region would require extensive changes in the closed form solution.

The extreme difficulty of closed form solution made numerical analysis desirable. Numerical analysis on an electronic computer had the additional advantages of relative flexibility and speed. With this method, intermediate

solutions for tabulation or graphic display are readily available.

A discussion of the Fortran IV program for the numerical solution of NET is now given. NICOLE is used to identify the program and stands for NET Interaction and Computer Operated Linear Evaluation. The term "linear" refers to the use of constant permeabilities in the model. Much of the mathematical manipulation needed to apply NET to a geographic region is coded into the program. NICOLE consists of a main program, Drive, and nine subprograms: Input, Update, Snap, Output, Savepl, Plot, Scal, Ptlr, and Block Data. The main program and the first four subprograms are the basic program and are capable of generating numerical solutions for NET. The next four subprograms are a sophisticated plotting package. The epidemic curves generated by NICOLE are graphically displayed. The last subprogram, Block Data, is used solely to initialize memory storage areas. Due to their auxiliary nature no further mention is made of the plotting package or the Block Data subprogram. NICOLE was written for and executed on an IBM 360/40.

The main program, Drive, makes very few calculations. None of NICOLE's read statements are found in Drive and few of the overall program's write statements are found in Drive. Drive's primary duty, as its name implies, is to drive subprograms. We will continue examining the main program after

looking at the subprograms upon which it is heavily dependent.

Subroutine Input reads and stores a mathematical description of the geographic division under study as well as the parameters used by the underlying epidemiological model. The first data card contains three integer values: NUMA, the number of AREAs; NUMP, the number of population segments; and NUMN, the maximum number of neighbors any AREA has. The second data card contains the infection rate and the removal rate for the modified K & K equations. Then a set of three cards is read in for each AREA.

Before preparing these cards, each AREA is arbitrarily and consecutively numbered starting with one. The first set of three data cards is associated with AREA number one and so on. All of the information on one set of cards pertains to one AREA. There are NUMA times three plus two data cards required to define an idealized geographic division.

The first of the three data cards pertaining to an AREA contains NUMP AREA populations which are stored in the vector $APOP_{I,J}$. The subscript I varies from one to NUMA and the subscript J varies from one to NUMP. The second card specifies the neighbors of an AREA. NUMN integers representing neighbor AREAs are read in and stored in vector $NEIB_{I,K}$. Again I varies from one to NUMA, while K varies from one to NUMN. The third card specifies the permeabilities of the AREA from each of its neighbor AREAs. The permeabilities are

stored in $PERM_{I,K}$. The subscripts I and K vary in the same manner as those on the preceeding card. After processing the two initial cards and three cards for each AREA, Input prints an echo check and returns control to the point where it was called.

Subroutine Update contains the differential equations which update the AREA populations, $APOPs_{I,J}$. These equations are based on effective populations which are supplied by the function $SNAP(I,J)$. Every time Update is called, the APOPs are advanced through one period of time. In the case of the modified K & K equations, B (the infection rate) and G (the removal rate) implicitly define that period of time. B and G are the step sizes in this first order Taylor series solution. A testing and storing procedure prohibits any AREA population from becoming negative.

The function $SNAP(I,J)$ calculates an effective AREA population and stores it in SNAP. $SNAP(I,J)$ s become the effective variables upon which the NET equations are based.

Subroutine Output prints out the present AREA populations and present total populations at the end of any period. Output also prints out the change in these populations since the last such report. The sum of the absolute value of all AREA population changes is calculated and stored in variable TC. TC is common to Output and Drive and is used by Drive to control the frequency of reporting.

With an understanding of Input, Update, and Output, the main program, Drive, can be intelligently read. All other subroutines called by Drive create additional output and do not affect the solution procedure. Input is called once at the beginning of Drive in order to define the NET and supply it with initial conditions. Then Update is called in order to increment the numerical solution. Output is not called as frequently as Update because of the tremendous number of pages of print-out this would generate. Otherwise, up to 10,000 reports would have been generated in a single run with very little difference from report to report. Drive is merely a scheme, derived by trial and error, for controlling the frequency of updates with respect to the reports.

4.2 Results of Numerical Analysis

In appendix C, the results of applying the computer program NICOLE to ten different data sets are summarized. There were no changes made to the program between runs--only the data cards were different. In the first data set all permeabilities were set equal to zero creating a special case. The next six data sets are examples of Linear. The last three data sets are examples of cubic. The Linear and Cubic geographic divisions are explained in section 3.2.

When all permeabilities are zero, the NET model based on the K & K equations reduces to several classical

K & K populations. Data set one reduced to six such populations. The numerical (appendix C) and deterministic (3, p. 28) solutions for the resulting epidemic intensities (also see figure 4.2.1) in these populations are in close agreement. This is encouraging and substantiates the results in the other nine data sets.

However, it is only fair to point out the problems of truncated error (27, p. 80) and instability (22, p. 336) involved with numerical solutions. A quick estimate of the maximum possible truncated error in the NICOLE solution is available because all of the functions involved have second derivatives less than one. Taking the step size, the maximum of B or G in this case, times the number of steps or periods gives us such an estimate. Substitution yields .01 time 10,000 or 100. Since the starting populations are 1,000 or greater in all cases, this gives us a maximum possible error in high intensity epidemics of ten percent. This figure could be reduced by decreasing the step size or applying a more sophisticated solution such as a fourth order Runge-Kutta method. Reducing the step size would markedly increase computing time. A Runge-Kutta method is desirable for some NET applications but would complicate formulating the NET equations. It is important to note that the maximum possible truncated error is not likely to be approached in this case because the curves involved are

COMPARISON OF NUMERICAL AND DETERMINISTIC
SOLUTIONS OF K & K EQUATIONS

| AREA | NUMERICAL | DETERMINISTIC |
|------|-----------|---------------|
| 1 | .04 | .00 |
| 2 | .21 | .20 |
| 3 | .40 | .40 |
| 4 | .60 | .60 |
| 5 | .80 | .80 |
| 6 | .98 | .98 |

Figure 4.2.1

smooth and many of the solutions were close to their final values long before the computer stopped iterating. Either truncated error or (more likely) instability could be responsible for the small difference between the numerical and deterministic solutions in AREA one of data set one.

Examination of the remaining data sets revealed two interesting effects. One, a directional effect, is best demonstrated in the Linear model. The other effect, a modified K & K threshold theorem for effective population, is best demonstrated in the cubic model.

Data sets four and five have the same Linear structure. The permeabilities are identical in both cases. The initial number of susceptibles is the same in all AREAs. The only difference in the two cases is the AREA in which the initial infection was started. However, note that in case four, when the infection was started in AREA one, the final intensity in AREA one was 10.90 percent. In case five, when the infection was started in AREA five, the final intensity in AREA one was 7.25 percent. By comparing data sets four and five to six and seven we can tentatively conclude that increasing the permeabilities decreased the directional effect.

In data set nine, the K & K threshold theorem is seen to apply to the effective population of each AREA. The numerical intensity of 80 percent is the same as the

deterministic K & K threshold theorem when applied to the effective AREA of the symmetrical populations. In data set ten, a combined effective threshold theorem and directional effect seemed to be present. Both effects have interesting biological and political implications.

Chapter 5

SUMMARY, CONCLUSION, AND RECOMMENDATIONS

5.1 Summary

A mathematical modeling procedure entitled NET was developed for linking approximately closed epidemiological communities together. One of the numerous differential models for closed populations was necessary for the NET method to work. A group of cities and their geographic and social relationships were easily represented by a NET model. The closed form solution of the set of simultaneous equations generated by NET was impractical and for this reason numerical analysis was employed. A computer program, NICLOE, was developed to generate the desired solutions and to investigate the feasibility of a user oriented computer program for the modeling and prediction of epidemic disease. The subsequent numerical analysis indicated two interesting effects: a directional effect and a modified Kermack and McKendrick threshold theorem based on effective population levels.

5.2 Conclusions

The NET model is highly flexible and has several realistic properties such as those observed by Bartlett. The directional effect and effective threshold effect

demonstrated in NET are intuitively appealing. These properties and effects are encouraging, however, the model used to produce them is highly simplified and applicable only to diseases like influenza which have a short latent period and are highly contagious. While closed form solution is not practical, the numerical solution of NET models are easily generated with the help of the computer program developed. A user oriented program for the modeling and prediction of the spread of epidemic diseases is a practical and useful objective. While data collection would be a major obstacle to the use of such a program, built in data manipulation capabilities might make better use of the existing data.

5.3 Recommendations

A better numerical method is desirable for less well behaved models than the K & K equations. Monte Carlo simulation has proven to be a valuable tool in epidemiological modeling and could be included in an expanded program. Finally, the model could be taken as is and applied to real world data, making modifications to the procedure as necessary.

APPENDIX A

A Listing of NICOLE, the Computer Program
Used to Solve the Thesis Model

```
//POKL      JOB '2874POKL','FRANK  PCKL',MSGLEVEL=1
// EXEC  FORTGCG
//FCRT.SYSIN DD *
C
```

```
      COMMON /BLK 1/ APOP(100,10),NEIB(100,10),PERM(100,10),
1      FAPCP(100,10),CAPOP(10),NUMA,NUMP,NUMN,P
      COMMON /BLK 2/ B,G,BP,GP,TC,TD,APOPI(100,10)
      INTEGER P
      CALL INPUT
      DO 30  M = 1 , 10
      DO 20  L = 1 , 10
      DO 10  K = 1 , 10
      CALL UPDATE
10  CONTINUE
      CALL SAVEPL
20  CONTINUE
      CALL OUTPUT
      IF ( TC .LT. 0.2 ) GO TO 50
30  CONTINUE
      DO 31  MM = 1 , 18
      DO 21  LL = 1 , 50
      DO 11  KK = 1 , 10
      CALL UPDATE
11  CONTINUE
      IF ( TD .GT. 0.5 ) CALL SAVEPL
21  CONTINUE
      CALL OUTPUT
      IF ( TC .LT. 0.2 ) GO TO 50
31  CONTINUE
50  CONTINUE
      TPOPI = 0.0
      TPOPF = 0.0
200  FORMAT(1H1/,30X,'CALCULATIONS ')
      WRITE(6,200)
      J = 1
100  FORMAT(/// 7X,' APCP ',I2,',',',I2,' TOTAL PERCENT REDUCTION ',F7.3)
      DO 60  I = 1 , NUMA
      TPOPI = TPOPI + APCPI(I,J)
      TPOPF = TPOPF + APOP(I,J)
      APOP(I,J)=((APOPI(I,J)-APOP(I,J))/APOPI(I,J))*100.0
      WRITE(6,100) I , J , APOP(I,J)
60  CONTINUE
      TPOPC = ((TPOPI-TPOPF) / TPOPI) * 100.0
101  FORMAT(////7X,' PCPULATION ',I2,' TOTAL PERCENT REDUCTION ',F7.3)
      WRITE(6,101) J , TPOPC
      CALL PLOT
      STOP
      END
```

```
C
      SUBROUTINE INPUT
      COMMON /BLK 1/ APOP(100,10),NEIB(100,10),PERM(100,10),
1      FAPCP(100,10),DAPCP(10),NUMA,NUMP,NUMN,P
      COMMON /BLK 2/ B,G,BP,GP,TC,TD,APOPI(100,10)
      COMMON /BLK 3/ APCPL(10,10),CL(10,10),X(500),Y(10,500),NP
      INTEGER P
99  FORMAT( 1H1 , 24X , 'GEOGRAPIC DATA',/)
      WRITE(6,99)
100  FORMAT(8I10)
101  FORMAT(8F10.0)
```

```

103 FORMAT(24X,'B = ',F10.8,10X,'G = ',F10.8)
104 FORMAT(24X,'BP = ',F10.8,10X,'GP = ',F10.8)
105 FORMAT(25X,'PERM ',I2,' ',I2,' IS ',F10.8)
106 FORMAT(25X,'NEIB ',I2,' ',I2,' IS ',I2)
10 READ(5,100) NUMA,NUMP,NUMN
   READ(5,101) B,G,BP,GP
   DO 13 I=1,NUMA
     READ(5,101) (APOP(I,J), J=1,NUMP)
     READ(5,100) (NEIB(I,K), K=1,NUMN)
     READ(5,101) (PERM(I,K), K=1,NUMN)
     WRITE(6,105)((I,K,PERM(I,K)),K=1,NUMN)
     WRITE(6,106)((I,K,NEIB(I,K)),K=1,NUMN)
13  CONTINUE
   WRITE(6,103) B,G
   WRITE(6,104) BP,GP
   DO 20 I=1,NUMA
     DO 20 J=1,NUMP
       APOPI(I,J) = APOP(I,J)
       APOPL(I,J) = APOP(I,J)
       DL(I,J) = APOP(I,J)
20  CONTINUE
   CALL OUTPUT
   RETURN
   END

   SUBROUTINE UPDATE
   COMMON /BLK 1/ APOP(100,10),NEIB(100,10),PERM(100,10),
1     FAPCP(100,10),DAPCP(10),NUMA,NUMP,NUMN,P
   COMMON /BLK 2/ B,G,BP,GP,TC,TD,APOPI(100,10)
   INTEGER P
   P = P + 1
   DO 50 I=1,NUMA
     DAPCP( 1) = -B * SNAP(I,1) * SNAP(I,2)
     DAPCP( 3) = G * SNAP(I,2)
     DAPCP( 2) = -DAPCP( 1) - DAPCP( 3)
     DO 50 J=1,NUMP
       FAPOP(I,J) = APOP(I,J) + DAPCP(J)
50  CONTINUE
     DO 60 I=1,NUMA
       DO 60 J=1,NUMP
         IF ( FAPOP(I,J) .LT. 0.0 ) FAPOP(I,J) = 0.0
         APOP(I,J) = FAPOP(I,J)
60  CONTINUE
     RETURN
     END

   FUNCTION SNAP(I,J)
   COMMON /BLK 1/ APOP(100,10),NEIB(100,10),PERM(100,10),
1     FAPCP(100,10),DAPCP(10),NUMA,NUMP,NUMN,P
   INTEGER P
   SNAP = APOP(I,J)
   DO 1 K=1,NUMN
     II = NEIB(I,K)
     SNAP = SNAP + APOP(II,J) * PERM(I ,K)
1  CONTINUE
   RETURN
   END

   SUBROUTINE OUTPUT

```

```

COMMON /BLK 1/ APCP(100,10),NEIP(100,10),PERM(100,10),
1      FAPCP(100,10),DAPCP(10),NUMA,NUMP,NUMN,P
COMMON /BLK 2/ B,G,RP,GP,TC,TD,APOPI(100,10)
COMMON /BLK 3/ APCPL(10,10),DL(10,10),X(500),Y(10,500),NP
DIMENSION CHANGE(10),TPOP(10),TPOPC(10)
DATA CHANGE,TPOP,TPCPC / 30*0.0 /
INTEGER P
100 FORMAT(1H1/,25X,'PERIOD',I10//)
101 FORMAT(30X,'AREA',I4)
102 FORMAT(7X,' POPULATION ',I2,' IS ',F10.2,8X,'IT HAS CHANGED ',
1 F10.2)
TC = 0.0
WRITE(6,100) P
DO 20 I=1,NUMA
WRITE(6,101) I
DO 10 J=1,NUMP
TPOP(J) = APOP(I,J) + TPOP(J)
CHANGE(J) = APOP(I,J) - APOPL(I,J)
APOPL(I,J) = APOP(I,J)
TC = TC + ABS ( CHANGE(J) )
WRITE(6,102) J,APCP(I,J) , CHANGE(J)
TPOPC(J) = TPOPC(J) + CHANGE(J)
10 CONTINUE
20 CONTINUE
103 FORMAT(7X,' TOTAL POP ',I2,' IS ',F10.2,8X,'IT HAS CHANGED ',
1 F10.2)
99 FORMAT(/,29X,'TOTALS')
WRITE(6, 99)
DO 30 J = 1,NUMP
WRITE(6,103) J , TPCP(J) , TPOPC(J)
TPOP(J) = 0.0
TPOPC(J) = 0.0
30 CONTINUE
RETURN
END

SUBROUTINE SAVEPL
COMMON /BLK 1/ APOP(100,10),NEIB(100,10),PERM(100,10),
1      FAPCP(100,10),CAPOP(10),NUMA,NUMP,NUMN,P
COMMON /BLK 2/ B,G,BP,GP,TC,TD,APOPI(100,10)
COMMON /BLK 3/ APCPL(10,10),DL(10,10),X(500),Y(10,500),NP
INTEGER P
TD = 0.0
NP = NP + 1
DO 20 I=1,NUMA
D = APOP(I,1) - DL(I,1)
DL(I,1) = APOP(I,1)
TD = TD + ABS(D)
Y(I,NP) = ABS ( D )
20 CONTINUE
I = I + 1
Y(I,NP) = TD
X(NP) = P
RETURN
END

SUBROUTINE PLOT
COMMON /BLK 1/ APCP(100,10),NEIB(100,10),PERM(100,10),
1      FAPCP(100,10),DAPOP(10),NUMA,NUMP,NUMN,P

```


| | |
|---|-----------|
| IF(XMIN.GT.X(I))XMIN=X(I) | SCAL 3 |
| IF(XMAX.LT.X(I))XMAX=X(I) | 43 SCAL 3 |
| 1 CONTINUE | SCAL 3 |
| *** TEST FOR CONSTANT | SCAL 3 |
| 2 IF(XMIN.EQ.XMAX)GO TO 7 | SCAL 4 |
| *** COMPUTE INITIAL TRIAL VALUE FOR INCREMENT IN PROBLEM UNITS | SCAL 4 |
| 3 J=1 | SCAL 4 |
| K=ALOG10(ABS(XMAX-XMIN)) | SCAL 4 |
| XDEL=10.0**K | SCAL 4 |
| *** COMPUTE TRIAL VALUE FOR MAXIMUM IN PROBLEM UNITS | SCAL 4 |
| 4 K=XMAX/XDEL+1.0 | SCAL 4 |
| ZMAX=K*XDEL | SCAL 4 |
| *** COMPUTE TRIAL VALUE FOR MINIMUM IN PROBLEM UNITS | SCAL 4 |
| K=XMIN/XDEL-1.0 | SCAL 4 |
| ZMIN=K*XDEL | SCAL 5 |
| IF(XMIN.GE.0.0.AND.XMAX.GE.3.0*XMIN.AND.ZMAX.NE.0.0)ZMIN=0.0 | SCAL 5 |
| *** COMPUTE VALUE FOR INCREMENT IN GRAPH UNITS | SCAL 5 |
| LDEL = FLOAT(LMAX-1)*XDEL / (ZMAX-ZMIN) | SCAL 5 |
| *** TEST FOR PROPER INCREMENT | SCAL 5 |
| IF(LDEL.GT.20)GO TO (5,6,8),J | SCAL 5 |
| IF(LDEL.GE.10)GO TO 8 | SCAL 5 |
| IF(J.NE.3)XDEL=2.0*XDEL | SCAL 5 |
| IF(J.EQ.3)XDEL=2.5*XDEL | SCAL 5 |
| J=3 | SCAL 5 |
| GO TO 4 | SCAL 6 |
| *** COMPUTE SECOND TRIAL VALUE FOR INCREMENT IN PROBLEM UNITS | SCAL 6 |
| 5 XDEL=0.5*XDEL | SCAL 6 |
| J=2 | SCAL 6 |
| GO TO 4 | SCAL 6 |
| *** COMPUTE FINAL TRIAL VALUE FOR INCREMENT IN PROBLEM UNITS | SCAL 6 |
| 6 XDEL=0.4*XDEL | SCAL 6 |
| J=3 | SCAL 6 |
| GO TO 4 | SCAL 6 |
| *** COMPUTE INITIAL TRIAL VALUE FOR INCREMENT IN PROBLEM UNITS - | SCAL 6 |
| CONSTANT X | SCAL 7 |
| 7 K=1 | SCAL 7 |
| IF(XMAX.NE.0.0) K=ALOG10(ABS(XMAX)) | SCAL 7 |
| XDEL=10.**K | SCAL 7 |
| XMAX=XMAX+0.5*XDEL | SCAL 7 |
| XMIN=XMIN-0.5*XDEL | SCAL 7 |
| XDEL=0.2*XDEL | SCAL 7 |
| J=3 | SCAL 7 |
| GO TO 4 | SCAL 7 |
| *** SET NEW MAXIMUM AND MINIMUM FOR DATA | SCAL 7 |
| 8 XMAX=ZMAX | SCAL 8 |
| XMIN=ZMIN | SCAL 8 |
| *** TEST OUTPUT SWITCH | SCAL 8 |
| IF(IO.NE.1)GO TO 10 | SCAL 8 |
| WRITE(6,100) | SCAL 8 |
| DO 9 I=1,N | SCAL 8 |
| WRITE(6,101) I, X(I) | SCAL 8 |
| 9 CONTINUE | SCAL 8 |
| WRITE(6,102) N, XDEL, XMAX, XMIN, LDEL, LMAX | SCAL 8 |
| 10 RETURN | SCAL 8 |
| 100 FORMAT('1SCAL - SCALING FOR PRINTER PLOT') | SCAL 9 |
| 101 FORMAT(5X,'X(',I3,')=',E14.6) | |
| 102 FORMAT(5X,'N',5X,'=',I14/5X,'XDEL',2X,'=',E14.6/5X,'XMAX',2X, | SCAL 9 |
| 1 '=' ,E14.6/5X,'XMIN',2X,'=',E14.6/5X,'LDEL',2X,'=',I14/ | SCAL 9 |
| 2 5X,'LMAX',2X,'=',I14) | |

END

SCAL 5

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SUBROUTINE PLTR(X,Y,N,XMAX,XMIN,XDEL,LX,YMAX,YMIN,YDEL,LY,

PLTR

PURPOSE

PLTR

PRINTS A PLOT FOR A SINGLE CURVE

PLTR

USAGE

PLTR

CALL PLTR(X,Y,N,XMAX,XMIN,XDEL,LX,YMAX,YMIN,YDEL,LY,LABX,
LABY)

PLTR

PLTR

PLTR

PLTR

DESCRIPTION OF PARAMETERS

PLTR 1

X -VECTOR CONTAINING X VALUES TO BE PLOTTED

PLTR 1

Y -VECTOR CONTAINING Y VALUES TO BE PLOTTED

PLTR 1

N -NUMBER OF VALUES FOR X AND Y

PLTR 1

XMAX -MAXIMUM SCALE MARK FOR X

PLTR 1

XMIN -MINIMUM SCALE MARK FOR X

PLTR 1

XDEL -INCREMENT FOR X SCALE

PLTR 1

LX -NUMBER OF PRINT POSITIONS FOR X-AXIS

PLTR 1

YMAX -MAXIMUM SCALE MARK FOR Y

PLTR 1

YMIN -MINIMUM SCALE MARK FOR Y

PLTR 2

YDEL -INCREMENT FOR Y SCALE

PLTR 2

LY -NUMBER OF LINES FOR Y-AXIS

PLTR 2

LABX -LABEL FOR X-AXIS

PLTR 2

LABY -LABEL FOR Y-AXIS

PLTR 2

REMARKS

PLTR 2

NONE

PLTR 2

METHOD

PLTR 2

DIRECT CALCULATION

PLTR 2

SUBPROGRAMS CALLED

PLTR 2

NONE

PLTR 2

PLTR 3

PLTR 3

PLTR 3

PLTR 3

1 LABX,LABY)

PLTR 3

DIMENSION X(1), Y(1), LABX(40), LABY(40), LINE(150), XXXX(30)

PLTR 3

DATA NXAX/1H-/ , NYAX/1H1/ , NMRK/1H+/, NPNT/1H*/ , NBLK/1H /,

PLTR 3

1 NLPN/1H(/ , N1XC/3H1X,/ , NFTP/4HF10./ , NZER/2H0,/ , NTWO/2H2,/ ,

PLTR 3

2 NRPN/4H1X,)/

PLTR 3

**** COMPUTE PRINT POSITIONS PER X SCALE DIVISION

PLTR 3

LXDL=FLOAT(LX-1)*XDEL/(XMAX-XMIN) + 0.001

PLTR 3

**** COMPUTE LINES PER Y SCALE DIVISION

PLTR 4

LYDL=FLOAT(LY-1)*YDEL/(YMAX-YMIN) +0.001

PLTR 4

**** COMPUTE X SCALING FACTOR

PLTR 4

XSCL=FLOAT(LXDL)/XDEL

PLTR 4

**** COMPUTE Y SCALING FACTOR

PLTR 4

YSCL=FLOAT(LYDL)/YDEL

PLTR 4

**** INITIALIZE POINTER FOR Y-AXIS LABEL

PLTR 4

L=(40-LY)/2

PLTR 4

**** INITIALIZE LINE COUNT

PLTR 4

I = LY-1

PLTR 5

**** INITIALIZE SCALE VALUE FOR Y-AXIS

PLTR 5

YS=YMAX

PLTR 5

YP=ABS(YMAX)

PLTR 5

**** PREPARE TOP LINE OR BOTTOM LINE

PLTR 5

1 DO 2 J=1,LX

PLTR 5

LINE(J)=NXAX

PLTR 5

2 CONTINUE

PLTR 5

| | |
|--|--------|
| DO 3 J=1,LX,LXDL | PLTR 5 |
| LINE(J)=NMRK | PLTR 5 |
| 3 CONTINUE | PLTR 6 |
| LINE(LX)=NMRK | PLTR 6 |
| IS=1 | PLTR 6 |
| *** TEST FOR SCALE DIVISION | PLTR 6 |
| IF(LYDL*(I/LYDL).EQ.I)GO TO 5 | PLTR 6 |
| GO TO 6 | PLTR 6 |
| *** TEST FOR SCALE DIVISION | PLTR 6 |
| 4 IF(LYDL*(I/LYDL).EQ.I)GO TO 5 | PLTR 6 |
| *** PREPARE LINE FOR NO SCALE VALUE | PLTR 6 |
| IS=1 | PLTR 6 |
| IF(I.EQ.LY) GO TO 6 | PLTR 7 |
| LINE(1)=NYAX | PLTR 7 |
| LINE(LX)=NYAX | PLTR 7 |
| GO TO 6 | PLTR 7 |
| *** PREPARE LINE FOR SCALE VALUE | PLTR 7 |
| 5 IS=3 | PLTR 7 |
| LINE(1)=NMRK | PLTR 7 |
| LINE(LX)=NMRK | PLTR 7 |
| *** TEST FOR POINTS | PLTR 7 |
| 6 DO 7 J=1,N | PLTR 7 |
| JY=(Y(J)-YMIN)*YSCL+0.5 | PLTR 8 |
| IF(JY.NE.I)GO TO 7 | PLTR 8 |
| *** PLOT POINT | PLTR 8 |
| JX=(X(J)-XMIN)*XSCL+1.5 | PLTR 8 |
| *** TEST FOR POINT IN X-AXIS RANGE | PLTR 8 |
| IF(JX.LE.LX.AND.JX.GT.0.AND.LINE(JX).NE.NMRK)LINE(JX)=NPNT | PLTR 8 |
| 7 CONTINUE | PLTR 8 |
| *** TEST FOR Y-AXIS LABEL | PLTR 8 |
| IF(L.GT.C.AND.L.LE.40)IS=IS+1 | PLTR 8 |
| GO TO(8,9,10,11),IS | PLTR 8 |
| *** NO SCALE - NO LABEL | PLTR 9 |
| 8 WRITE(6,100) (LINE(J),J=1,LX) | PLTR 9 |
| GO TO 12 | PLTR 9 |
| *** NO SCALE - LABEL | PLTR 9 |
| 9 WRITE(6,101) LABY(L), (LINE(J),J=1,LX) | PLTR 9 |
| GO TO 12 | PLTR 9 |
| *** SCALE - NO LABEL | PLTR 9 |
| 10 IF(YP.GT. 10.0)WRITE(6,102) YS, (LINE(J),J=1,LX) | PLTR 9 |
| IF(YP.LE. 10.0)WRITE(6,103) YS, (LINE(J),J=1,LX) | PLTR 9 |
| YS=YS-YDEL | PLTR 9 |
| GO TO 12 | PLTR10 |
| *** SCALE - LABEL | PLTR10 |
| 11 IF(YP.GT. 10.0)WRITE(6,104) LABY(L), YS, (LINE(J),J=1,LX) | PLTR10 |
| IF(YP.LE. 10.0)WRITE(6,105) LABY(L), YS, (LINE(J),J=1,LX) | PLTR10 |
| YS=YS-YDEL | PLTR10 |
| *** UPDATE LABEL POINTER | PLTR10 |
| 12 L=L+1 | PLTR10 |
| *** TEST FOR END OF GRAPH | PLTR10 |
| IF(I.EQ.0)GO TO 14 | PLTR10 |
| *** TEST FOR LAST LINE OF GRAPH | PLTR10 |
| I=I-1 | PLTR11 |
| IF(I.EQ.0)GO TO 1 | PLTR11 |
| *** BLANK OUT LINE | PLTR11 |
| DO 13 J=1,LX | PLTR11 |
| LINE(J)=NBLK | PLTR11 |
| 13 CONTINUE | PLTR11 |
| GO TO 4 | PLTR11 |

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PLTR18
PLTR18
PLTR18
PLTR18
PLTR18
PLTR19

```
*
/GC.SYSIN DD *
```

| 00001 | 6 | 3 | 2 | | | | | |
|-------|-----|-----|-----|-----|-----|-----|-----|--|
| 1000 | .01 | | | | | | | |
| 2 | | 6 | | | | | | |
| .05 | | | .05 | .05 | .05 | .05 | .05 | |
| 1116 | | | | | | | | |
| 1 | | 3 | | | | | | |
| .05 | | .05 | .05 | .05 | .05 | .05 | .05 | |
| 1277 | | | | | | | | |
| 2 | | 4 | | | | | | |
| .05 | | .05 | .05 | .05 | .05 | .05 | .05 | |
| 1527 | | | | | | | | |
| 3 | | 5 | | | | | | |
| .05 | | .05 | .05 | .05 | .05 | .05 | .05 | |
| 2012 | | | | | | | | |
| 4 | | 6 | | | | | | |
| .05 | | .05 | .05 | .05 | .05 | .05 | .05 | |
| 3992 | | | | | | | | |
| 5 | | 2 | | | | | | |
| .05 | | 1 | .05 | .05 | .05 | .05 | .05 | |

APPENDIX B

Selected Output from NICOLE

Using Data Set 2

GEOGRAPHIC DATA

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| | | | | |
|------|---|---|----|------------|
| PERM | 1 | 1 | IS | 0.05000000 |
| PERM | 1 | 2 | IS | 0.0 |
| NEIB | 1 | 1 | IS | 2 |
| NEIB | 1 | 2 | IS | 6 |
| PERM | 2 | 1 | IS | 0.05000000 |
| PERM | 2 | 2 | IS | 0.05000000 |
| NEIB | 2 | 1 | IS | 1 |
| NEIB | 2 | 2 | IS | 3 |
| PERM | 3 | 1 | IS | 0.05000000 |
| PERM | 3 | 2 | IS | 0.05000000 |
| NEIB | 3 | 1 | IS | 2 |
| NEIB | 3 | 2 | IS | 4 |
| PERM | 4 | 1 | IS | 0.05000000 |
| PERM | 4 | 2 | IS | 0.05000000 |
| NEIB | 4 | 1 | IS | 3 |
| NEIB | 4 | 2 | IS | 5 |
| PERM | 5 | 1 | IS | 0.05000000 |
| PERM | 5 | 2 | IS | 0.05000000 |
| NEIB | 5 | 1 | IS | 4 |
| NEIB | 5 | 2 | IS | 6 |
| PERM | 6 | 1 | IS | 0.05000000 |
| PERM | 6 | 2 | IS | 0.0 |
| NEIB | 6 | 1 | IS | 5 |
| NEIB | 6 | 2 | IS | 1 |

B = 0.00001000 G = 0.01000000
BP = 0.0 GP = 0.0

| | | | | | | |
|------------|---|----|----------|---|----------------|-----|
| | | | AREA | 1 | | |
| POPULATION | 1 | IS | 1000.00 | | IT HAS CHANGED | 0.0 |
| POPULATION | 2 | IS | 1.00 | | IT HAS CHANGED | 0.0 |
| POPULATION | 3 | IS | 0.0 | | IT HAS CHANGED | 0.0 |
| | | | AREA | 2 | | |
| POPULATION | 1 | IS | 1116.00 | | IT HAS CHANGED | 0.0 |
| POPULATION | 2 | IS | 0.0 | | IT HAS CHANGED | 0.0 |
| POPULATION | 3 | IS | 0.0 | | IT HAS CHANGED | 0.0 |
| | | | AREA | 3 | | |
| POPULATION | 1 | IS | 1277.00 | | IT HAS CHANGED | 0.0 |
| POPULATION | 2 | IS | 0.0 | | IT HAS CHANGED | 0.0 |
| POPULATION | 3 | IS | 0.0 | | IT HAS CHANGED | 0.0 |
| | | | AREA | 4 | | |
| POPULATION | 1 | IS | 1527.00 | | IT HAS CHANGED | 0.0 |
| POPULATION | 2 | IS | 0.0 | | IT HAS CHANGED | 0.0 |
| POPULATION | 3 | IS | 0.0 | | IT HAS CHANGED | 0.0 |
| | | | AREA | 5 | | |
| POPULATION | 1 | IS | 2012.00 | | IT HAS CHANGED | 0.0 |
| POPULATION | 2 | IS | 0.0 | | IT HAS CHANGED | 0.0 |
| POPULATION | 3 | IS | 0.0 | | IT HAS CHANGED | 0.0 |
| | | | AREA | 6 | | |
| POPULATION | 1 | IS | 3992.00 | | IT HAS CHANGED | 0.0 |
| POPULATION | 2 | IS | 0.0 | | IT HAS CHANGED | 0.0 |
| POPULATION | 3 | IS | 0.0 | | IT HAS CHANGED | 0.0 |
| | | | TOTALS | | | |
| TOTAL POP | 1 | IS | 10924.00 | | IT HAS CHANGED | 0.0 |
| TOTAL POP | 2 | IS | 1.00 | | IT HAS CHANGED | 0.0 |
| TOTAL POP | 3 | IS | 0.0 | | IT HAS CHANGED | 0.0 |

| | | | | | |
|------------|------|----------|---|----------------|-------|
| | | AREA | 1 | | |
| POPULATION | 1 IS | 998.90 | | IT HAS CHANGED | -1.10 |
| POPULATION | 2 IS | 1.06 | | IT HAS CHANGED | 0.06 |
| POPULATION | 3 IS | 1.03 | | IT HAS CHANGED | 1.03 |
| | | AREA | 2 | | |
| POPULATION | 1 IS | 1115.92 | | IT HAS CHANGED | -0.08 |
| POPULATION | 2 IS | 0.01 | | IT HAS CHANGED | 0.01 |
| POPULATION | 3 IS | 0.06 | | IT HAS CHANGED | 0.06 |
| | | AREA | 3 | | |
| POPULATION | 1 IS | 1277.00 | | IT HAS CHANGED | 0.0 |
| POPULATION | 2 IS | 0.00 | | IT HAS CHANGED | 0.00 |
| POPULATION | 3 IS | 0.00 | | IT HAS CHANGED | 0.00 |
| | | AREA | 4 | | |
| POPULATION | 1 IS | 1527.00 | | IT HAS CHANGED | 0.0 |
| POPULATION | 2 IS | 0.00 | | IT HAS CHANGED | 0.00 |
| POPULATION | 3 IS | 0.00 | | IT HAS CHANGED | 0.00 |
| | | AREA | 5 | | |
| POPULATION | 1 IS | 2012.00 | | IT HAS CHANGED | 0.0 |
| POPULATION | 2 IS | 0.00 | | IT HAS CHANGED | 0.00 |
| POPULATION | 3 IS | 0.00 | | IT HAS CHANGED | 0.00 |
| | | AREA | 6 | | |
| POPULATION | 1 IS | 3992.00 | | IT HAS CHANGED | 0.0 |
| POPULATION | 2 IS | 0.00 | | IT HAS CHANGED | 0.00 |
| POPULATION | 3 IS | 0.00 | | IT HAS CHANGED | 0.00 |
| | | TOTALS | | | |
| TOTAL POP | 1 IS | 10922.82 | | IT HAS CHANGED | -1.18 |
| TOTAL POP | 2 IS | 1.07 | | IT HAS CHANGED | 0.07 |
| TOTAL POP | 3 IS | 1.09 | | IT HAS CHANGED | 1.09 |

| | | | | | |
|------------|------|---------|---|----------------|----------|
| | | AREA | 1 | | |
| POPULATION | 1 IS | 986.08 | | IT HAS CHANGED | -1.71 |
| POPULATION | 2 IS | 1.64 | | IT HAS CHANGED | 0.07 |
| POPULATION | 3 IS | 13.17 | | IT HAS CHANGED | 1.63 |
| | | AREA | 2 | | |
| POPULATION | 1 IS | 1113.03 | | IT HAS CHANGED | -0.69 |
| POPULATION | 2 IS | 0.53 | | IT HAS CHANGED | 0.13 |
| POPULATION | 3 IS | 2.33 | | IT HAS CHANGED | 0.55 |
| | | AREA | 3 | | |
| POPULATION | 1 IS | 1276.22 | | IT HAS CHANGED | -0.35 |
| POPULATION | 2 IS | 0.19 | | IT HAS CHANGED | 0.10 |
| POPULATION | 3 IS | 0.47 | | IT HAS CHANGED | 0.24 |
| | | AREA | 4 | | |
| POPULATION | 1 IS | 1517.05 | | IT HAS CHANGED | -9.01 |
| POPULATION | 2 IS | 3.99 | | IT HAS CHANGED | 3.63 |
| POPULATION | 3 IS | 5.89 | | IT HAS CHANGED | 5.37 |
| | | AREA | 5 | | |
| POPULATION | 1 IS | 1701.56 | | IT HAS CHANGED | -272.79 |
| POPULATION | 2 IS | 157.30 | | IT HAS CHANGED | 136.40 |
| POPULATION | 3 IS | 153.09 | | IT HAS CHANGED | 136.38 |
| | | AREA | 6 | | |
| POPULATION | 1 IS | 897.40 | | IT HAS CHANGED | -2385.77 |
| POPULATION | 2 IS | 1684.92 | | IT HAS CHANGED | 1166.12 |
| POPULATION | 3 IS | 1409.58 | | IT HAS CHANGED | 1219.61 |
| | | TOTALS | | | |
| TOTAL POP | 1 IS | 7491.34 | | IT HAS CHANGED | -2670.31 |
| TOTAL POP | 2 IS | 1848.57 | | IT HAS CHANGED | 1306.44 |
| TOTAL POP | 3 IS | 1584.53 | | IT HAS CHANGED | 1363.78 |

| | | | | | |
|------------|------|---------|---|----------------|--------|
| | | AREA | 1 | | |
| POPULATION | 1 IS | 899.56 | | IT HAS CHANGED | -4.23 |
| POPULATION | 2 IS | 0.67 | | IT HAS CHANGED | -0.27 |
| POPULATION | 3 IS | 100.20 | | IT HAS CHANGED | 4.45 |
| | | AREA | 2 | | |
| POPULATION | 1 IS | 745.07 | | IT HAS CHANGED | -7.58 |
| POPULATION | 2 IS | 1.10 | | IT HAS CHANGED | -1.59 |
| POPULATION | 3 IS | 369.06 | | IT HAS CHANGED | 9.06 |
| | | AREA | 3 | | |
| POPULATION | 1 IS | 631.37 | | IT HAS CHANGED | -0.36 |
| POPULATION | 2 IS | 0.0 | | IT HAS CHANGED | 0.0 |
| POPULATION | 3 IS | 645.06 | | IT HAS CHANGED | 0.39 |
| | | AREA | 4 | | |
| POPULATION | 1 IS | 492.99 | | IT HAS CHANGED | 0.0 |
| POPULATION | 2 IS | 0.0 | | IT HAS CHANGED | 0.0 |
| POPULATION | 3 IS | 1034.71 | | IT HAS CHANGED | 0.0 |
| | | AREA | 5 | | |
| POPULATION | 1 IS | 306.84 | | IT HAS CHANGED | 0.0 |
| POPULATION | 2 IS | 0.0 | | IT HAS CHANGED | 0.0 |
| POPULATION | 3 IS | 1707.96 | | IT HAS CHANGED | 0.0 |
| | | AREA | 6 | | |
| POPULATION | 1 IS | 24.97 | | IT HAS CHANGED | 0.0 |
| POPULATION | 2 IS | 0.0 | | IT HAS CHANGED | 0.0 |
| POPULATION | 3 IS | 3967.24 | | IT HAS CHANGED | 0.0 |
| | | TOTALS | | | |
| TOTAL POP | 1 IS | 3100.80 | | IT HAS CHANGED | -12.18 |
| TOTAL POP | 2 IS | 1.77 | | IT HAS CHANGED | -1.86 |
| TOTAL POP | 3 IS | 7824.23 | | IT HAS CHANGED | 13.89 |

| | | | | | | |
|------------|---|----|---------|---|----------------|-------|
| | | | AREA | 1 | | |
| POPULATION | 1 | IS | 890.09 | | IT HAS CHANGED | -0.12 |
| POPULATION | 2 | IS | 0.02 | | IT HAS CHANGED | -0.01 |
| POPULATION | 3 | IS | 109.76 | | IT HAS CHANGED | 0.11 |
| | | | AREA | 2 | | |
| POPULATION | 1 | IS | 739.47 | | IT HAS CHANGED | 0.0 |
| POPULATION | 2 | IS | 0.0 | | IT HAS CHANGED | 0.0 |
| POPULATION | 3 | IS | 374.79 | | IT HAS CHANGED | 0.0 |
| | | | AREA | 3 | | |
| POPULATION | 1 | IS | 630.93 | | IT HAS CHANGED | 0.0 |
| POPULATION | 2 | IS | 0.0 | | IT HAS CHANGED | 0.0 |
| POPULATION | 3 | IS | 645.18 | | IT HAS CHANGED | 0.0 |
| | | | AREA | 4 | | |
| POPULATION | 1 | IS | 492.99 | | IT HAS CHANGED | 0.0 |
| POPULATION | 2 | IS | 0.0 | | IT HAS CHANGED | 0.0 |
| POPULATION | 3 | IS | 1034.71 | | IT HAS CHANGED | 0.0 |
| | | | AREA | 5 | | |
| POPULATION | 1 | IS | 306.84 | | IT HAS CHANGED | 0.0 |
| POPULATION | 2 | IS | 0.0 | | IT HAS CHANGED | 0.0 |
| POPULATION | 3 | IS | 1707.96 | | IT HAS CHANGED | 0.0 |
| | | | AREA | 6 | | |
| POPULATION | 1 | IS | 24.97 | | IT HAS CHANGED | 0.0 |
| POPULATION | 2 | IS | 0.0 | | IT HAS CHANGED | 0.0 |
| POPULATION | 3 | IS | 3967.24 | | IT HAS CHANGED | 0.0 |
| | | | TOTALS | | | |
| TOTAL POP | 1 | IS | 3085.28 | | IT HAS CHANGED | -0.12 |
| TOTAL POP | 2 | IS | 0.02 | | IT HAS CHANGED | -0.01 |
| TOTAL POP | 3 | IS | 7839.65 | | IT HAS CHANGED | 0.11 |

CALCULATIONS

55

APOP 1, 1 TOTAL PERCENT REDUCTION 10.991

APOP 2, 1 TOTAL PERCENT REDUCTION 33.739

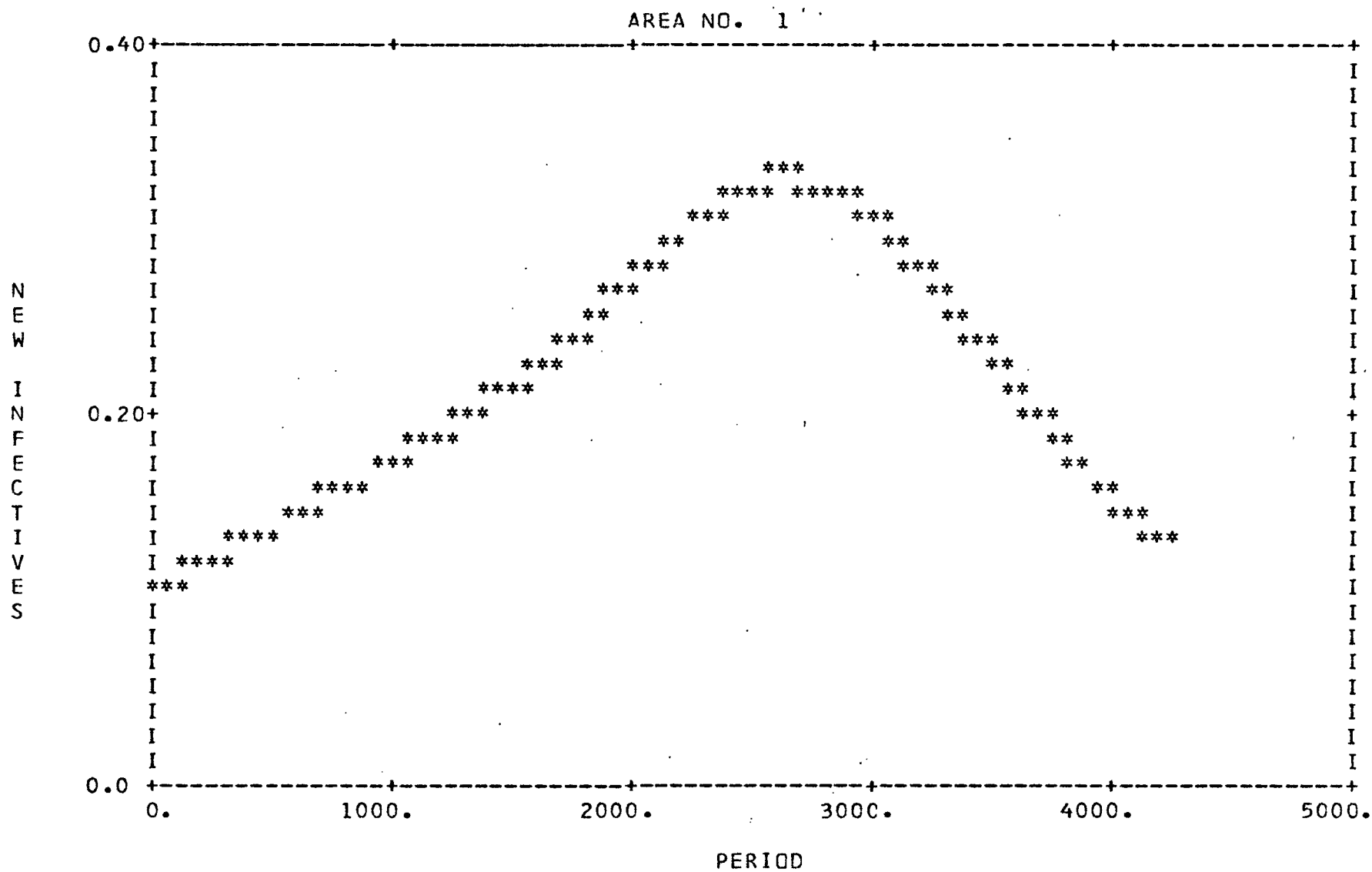
APOP 3, 1 TOTAL PERCENT REDUCTION 50.593

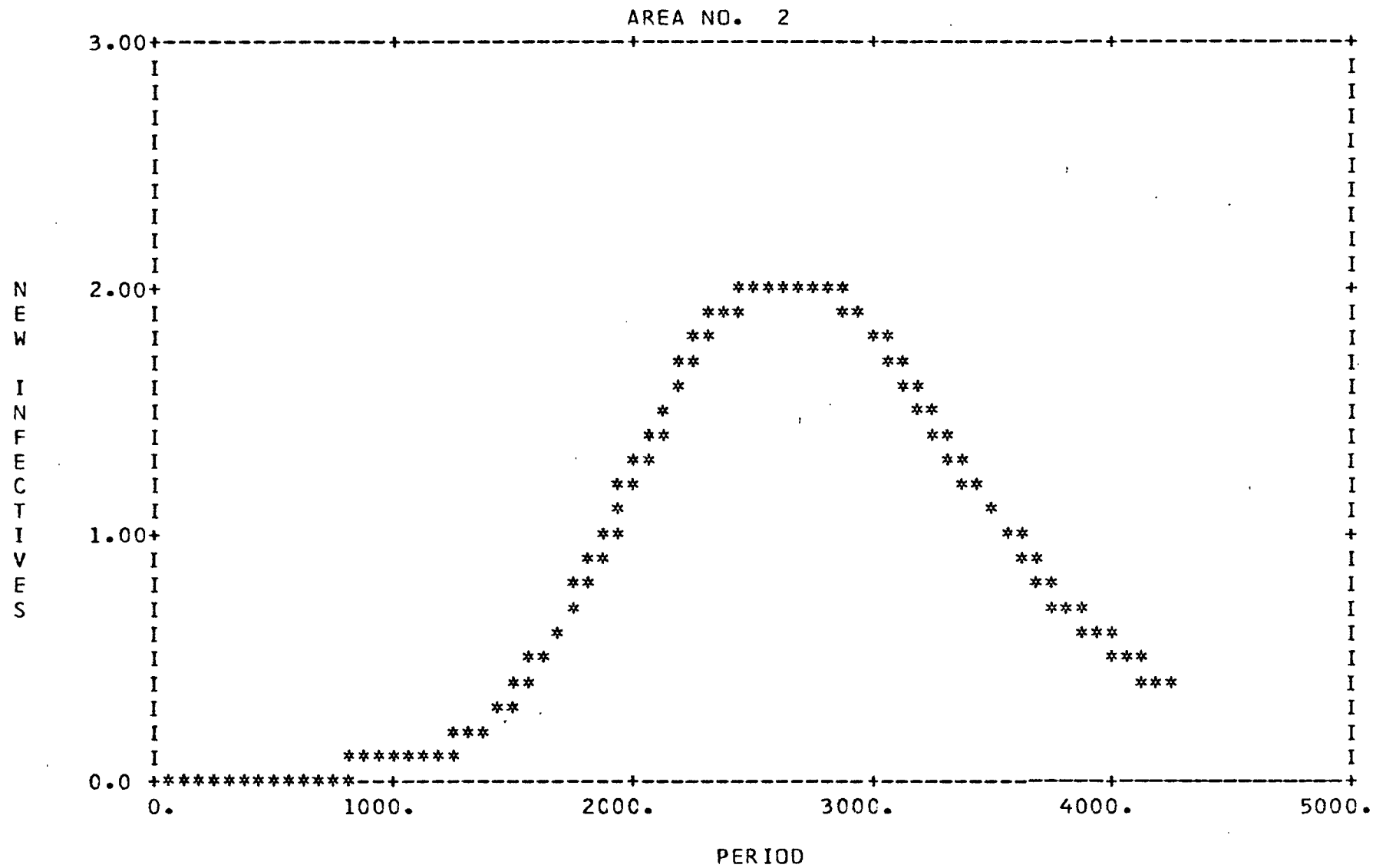
APOP 4, 1 TOTAL PERCENT REDUCTION 67.715

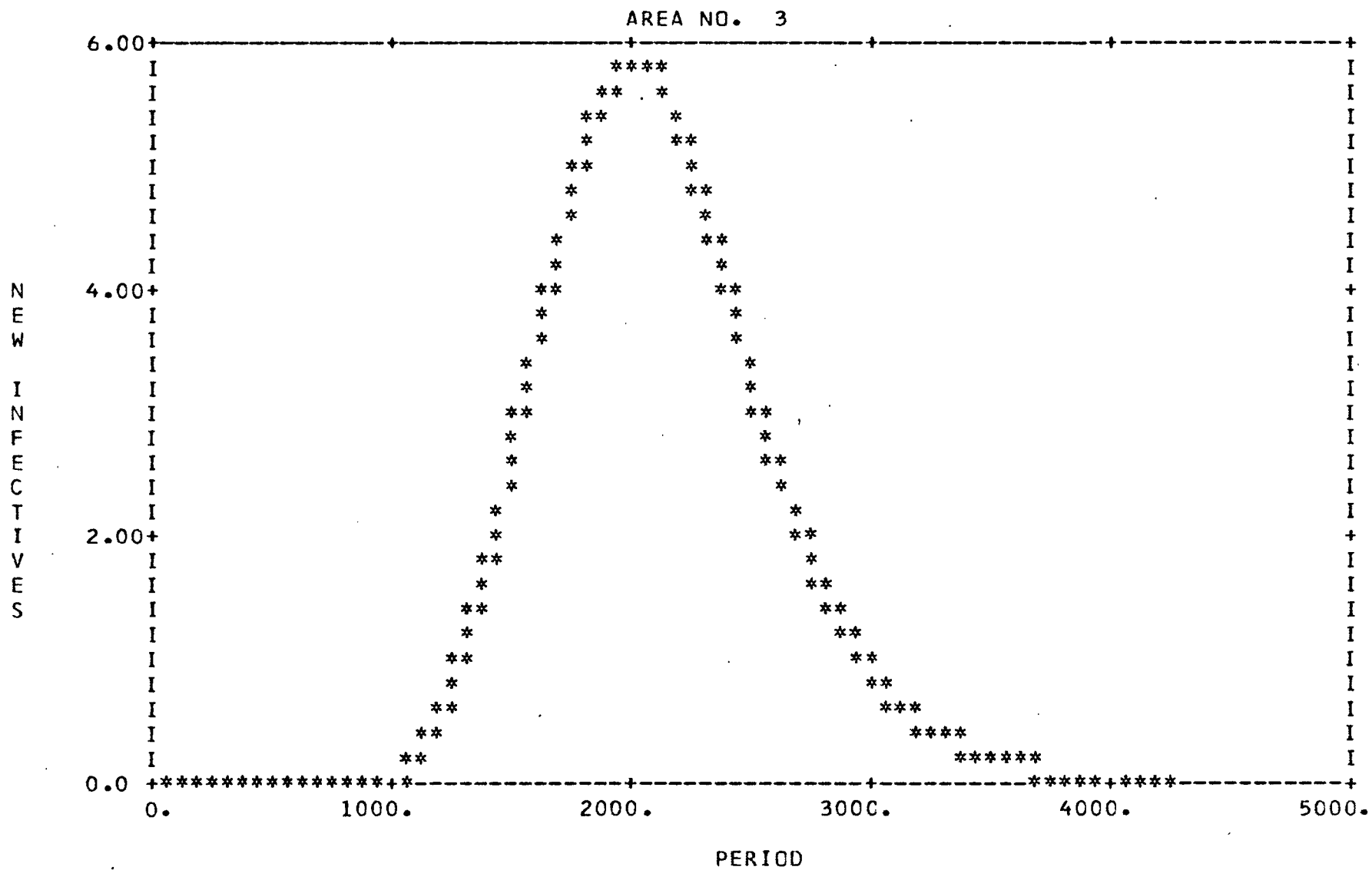
APOP 5, 1 TOTAL PERCENT REDUCTION 84.750

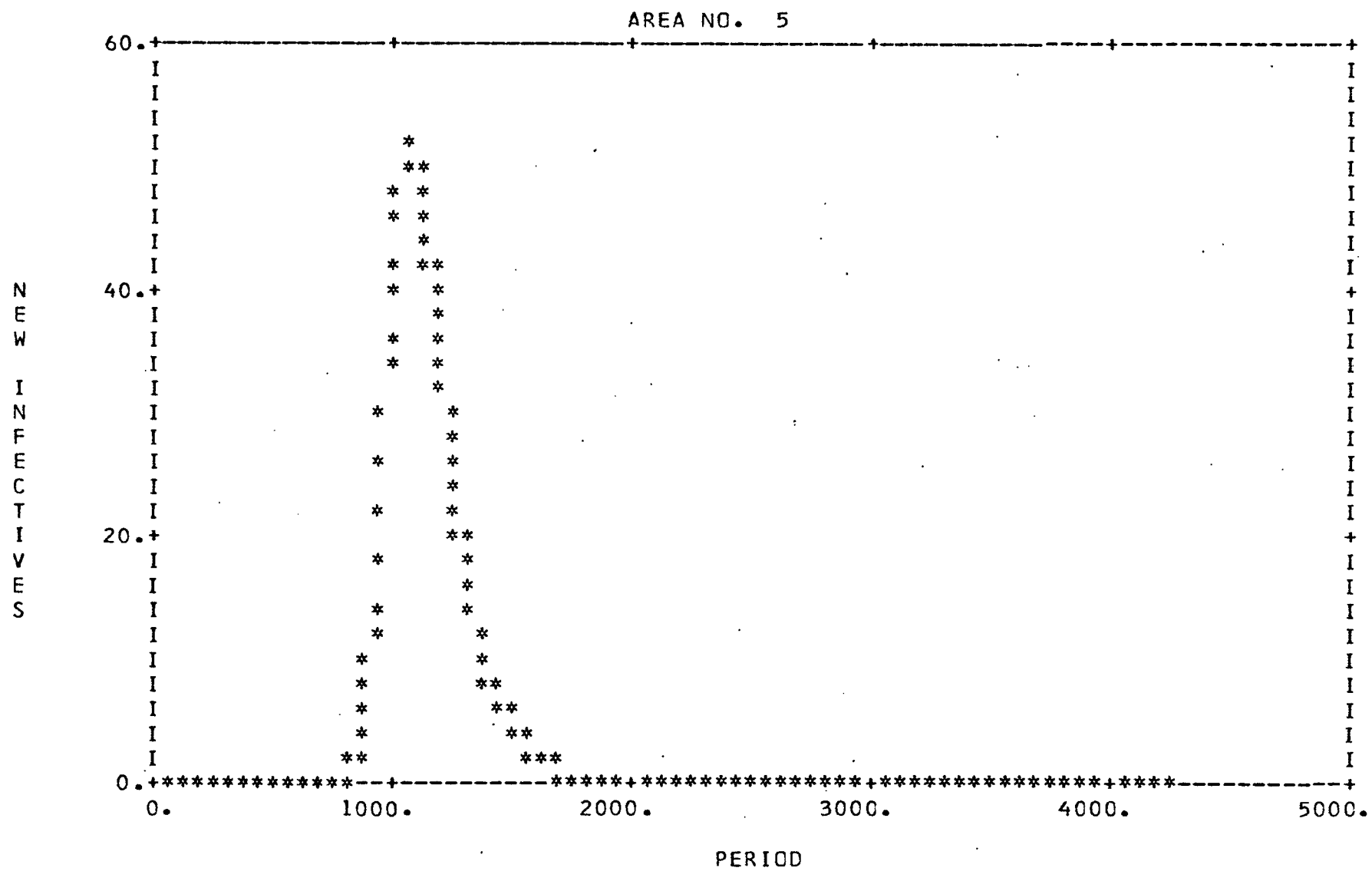
APOP 6, 1 TOTAL PERCENT REDUCTION 99.374

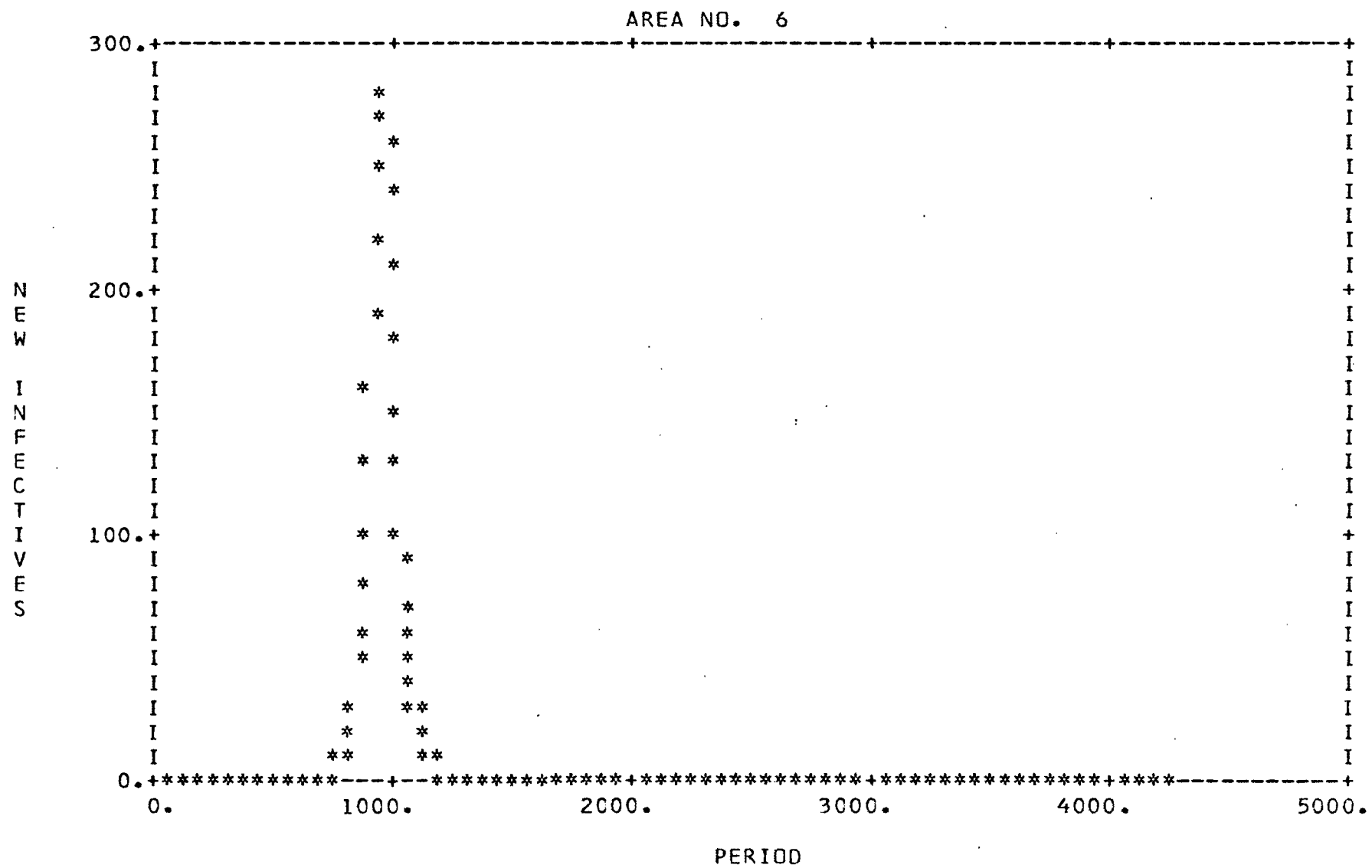
POPULATION 1 TOTAL PERCENT REDUCTION 71.757











APPENDIX C

Summary of the Solution of Ten NET Models

CONVENTIONS USED TO SUMMARIZE
THE 10 NET DATA SETS PRESENTED IN APPENDIX C

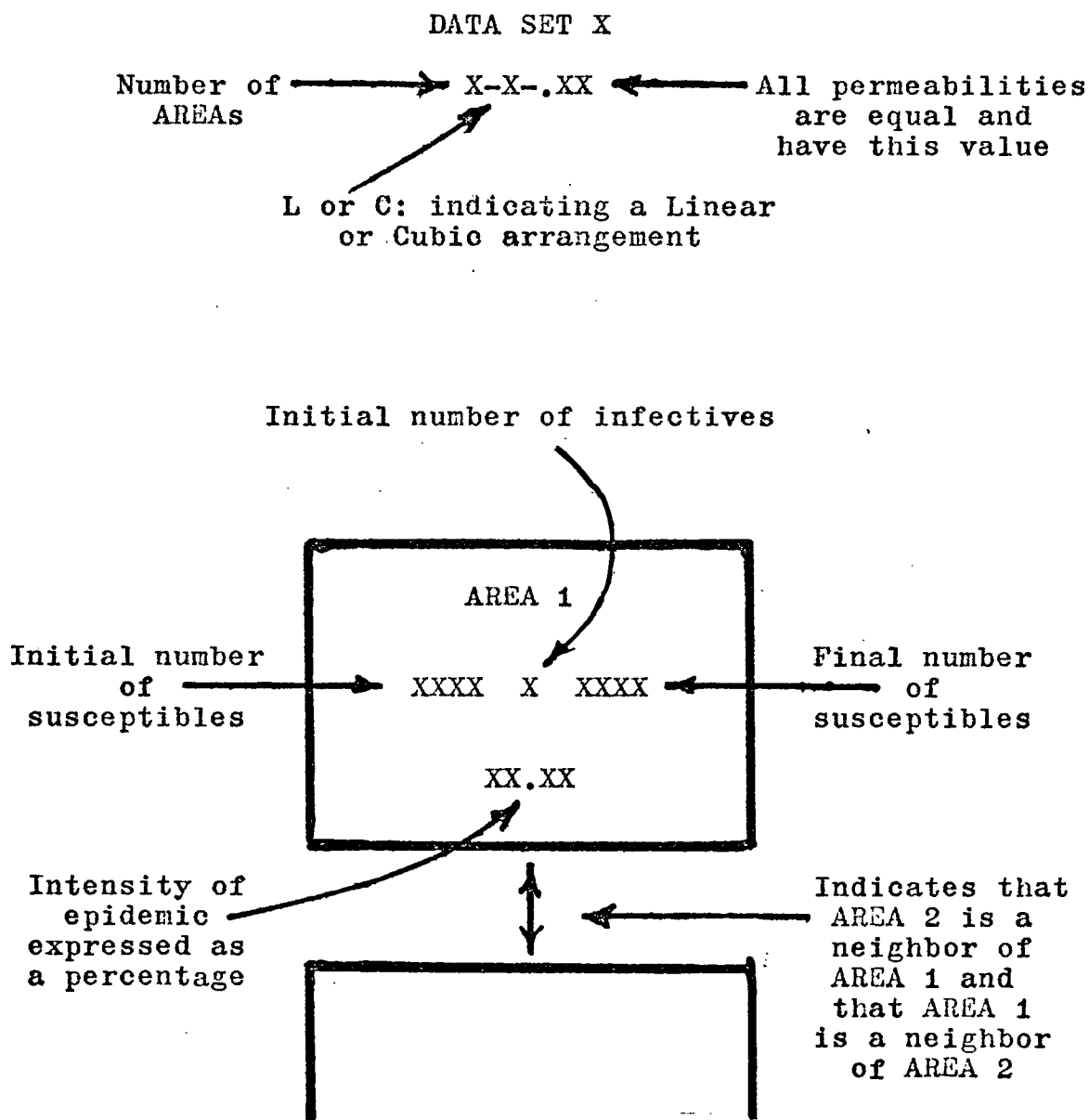


Figure C.1

DATA SET 1

6-L-0

AREA 2

1116 1 884

20.80

AREA 1

1000 1 956

4.38

AREA 3

1277 1 763

40.29

AREA

AREA 4

1527 1 609

60.14

AREA 5

2012 2 400

80.14

AREA 6

3992 2 78

98.05

AREA TOTAL

10924 8 3689

66.23

RUN TIME

9.15

MAXIMUM PERIOD

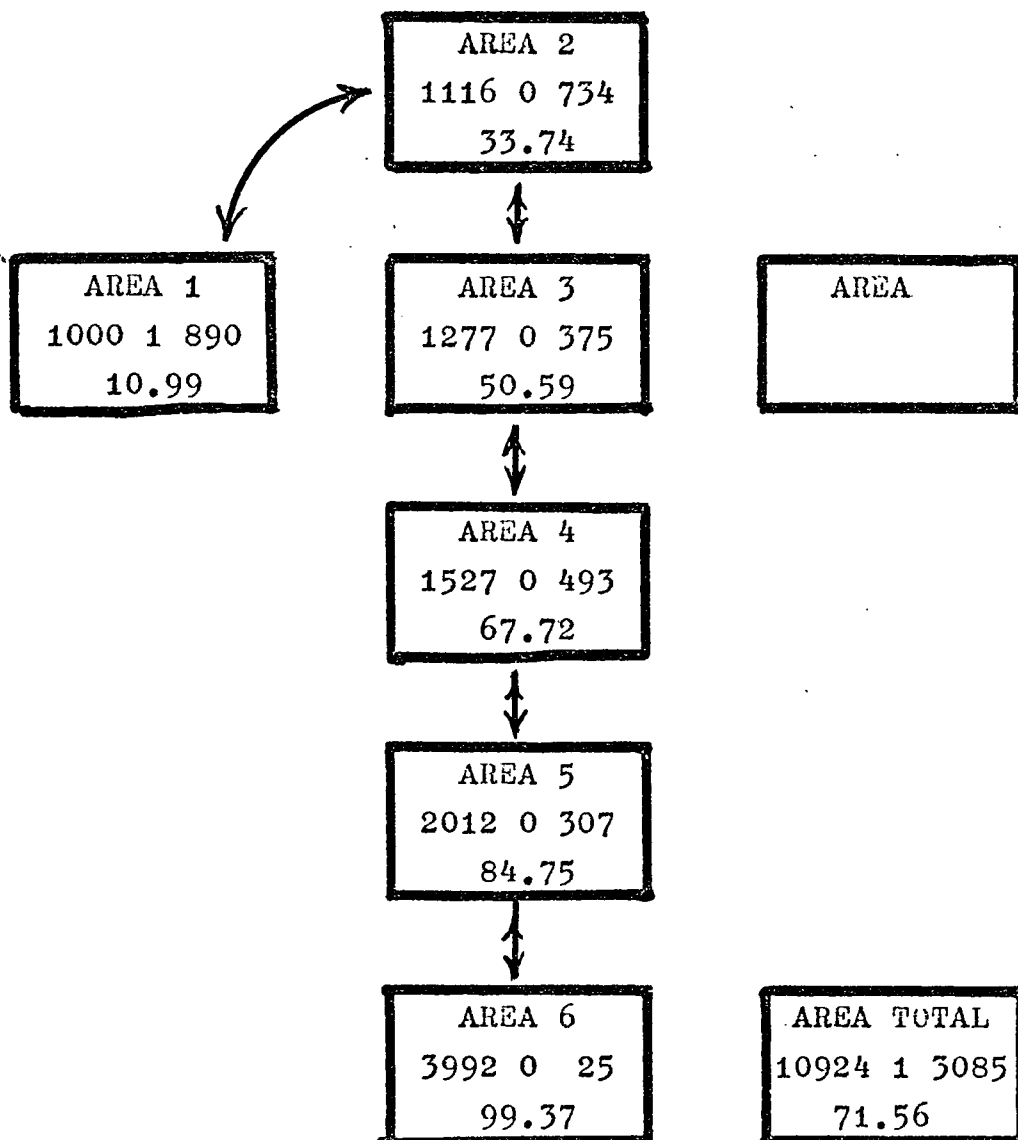
10,000

RELATIVE REMOVAL

RATE 1,000

DATA SET 2

6-L-.05



RUN TIME

8.33

MAXIMUM PERIOD

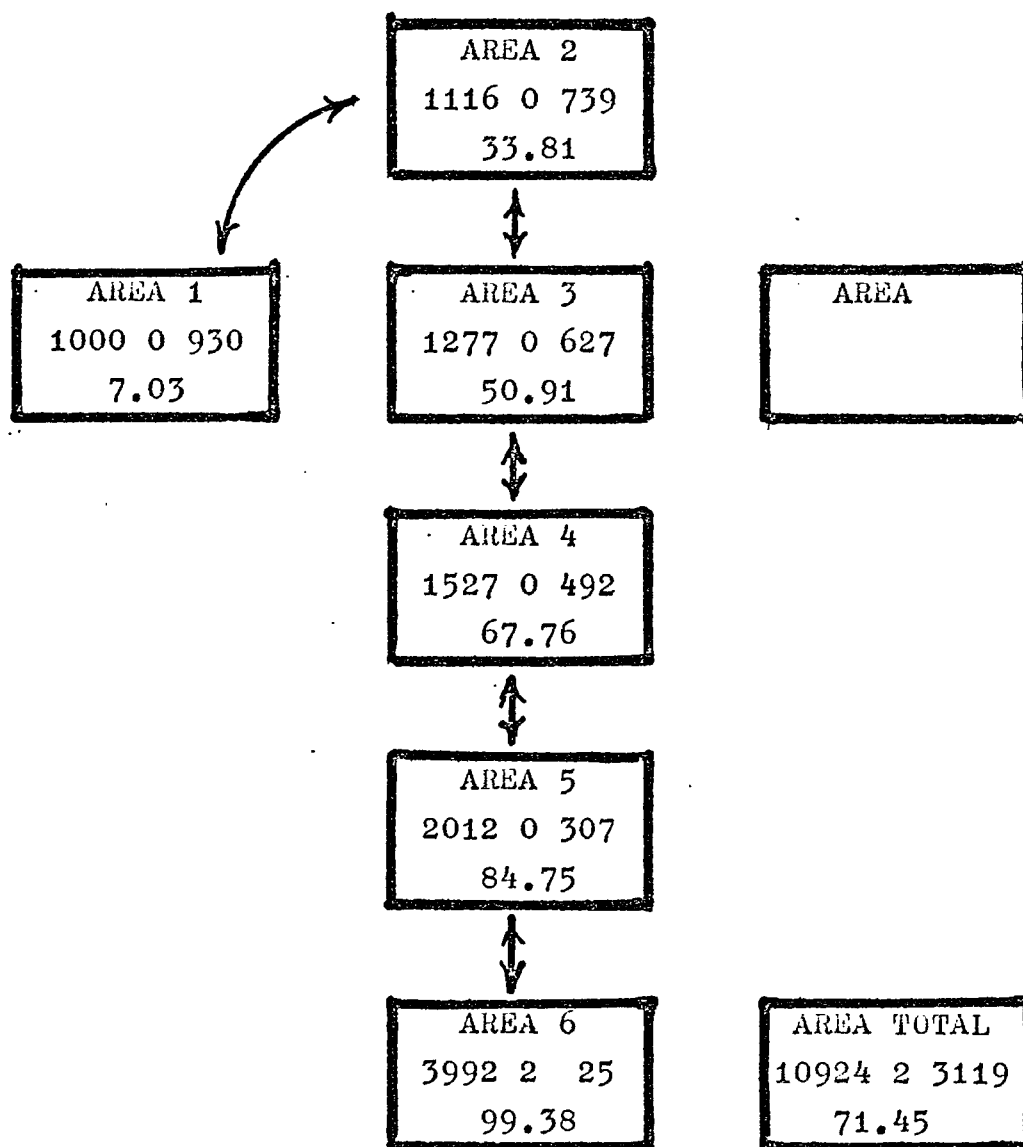
10,000

RELATIVE REMOVAL

RATE 1,000

DATA SET 3

6-L-.25



RUN TIME

9.33

MAXIMUM PERIOD

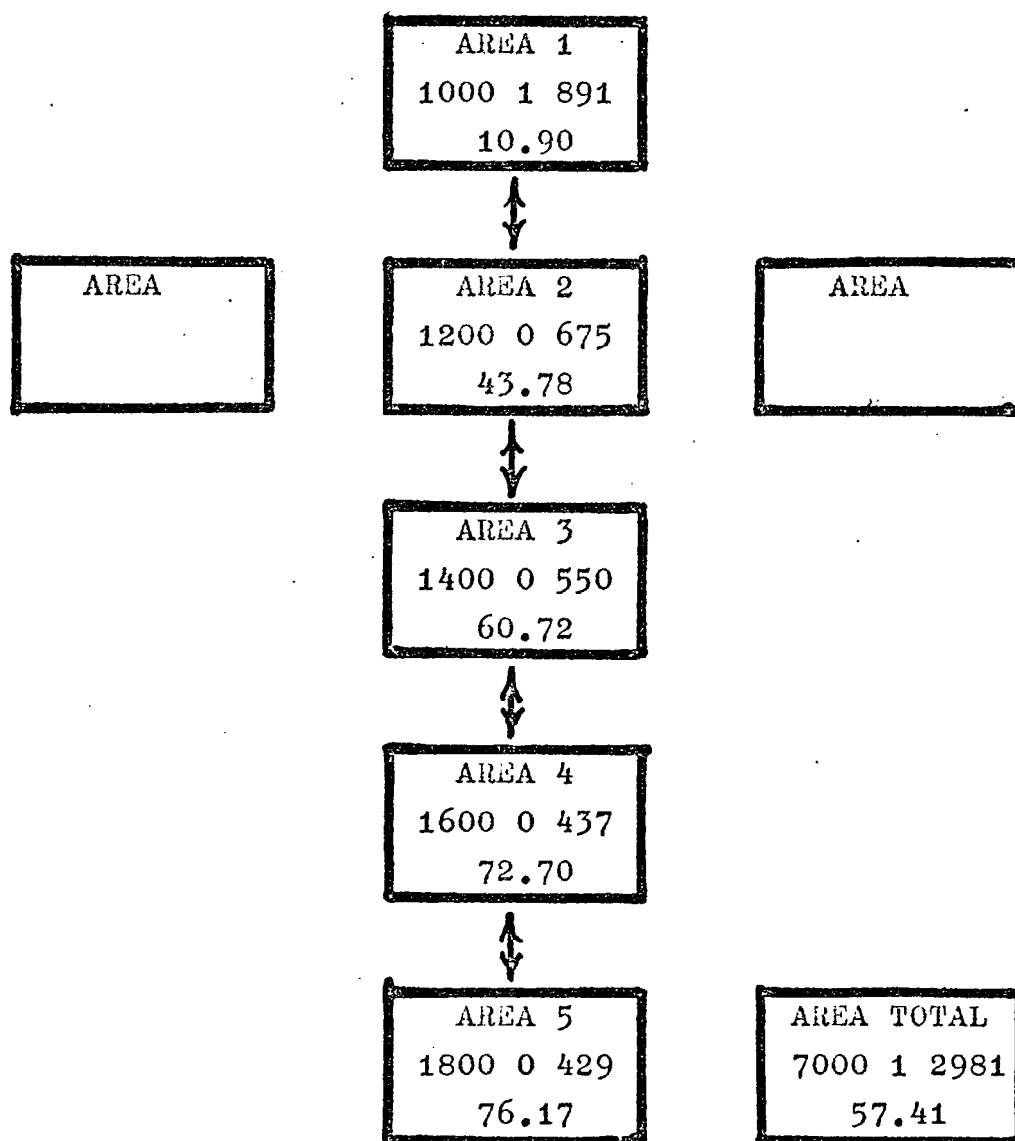
10,000

RELATIVE REMOVAL

RATE 1,000

DATA SET 4

5-L-.05



RUN TIME

7.44

MAXIMUM PERIOD

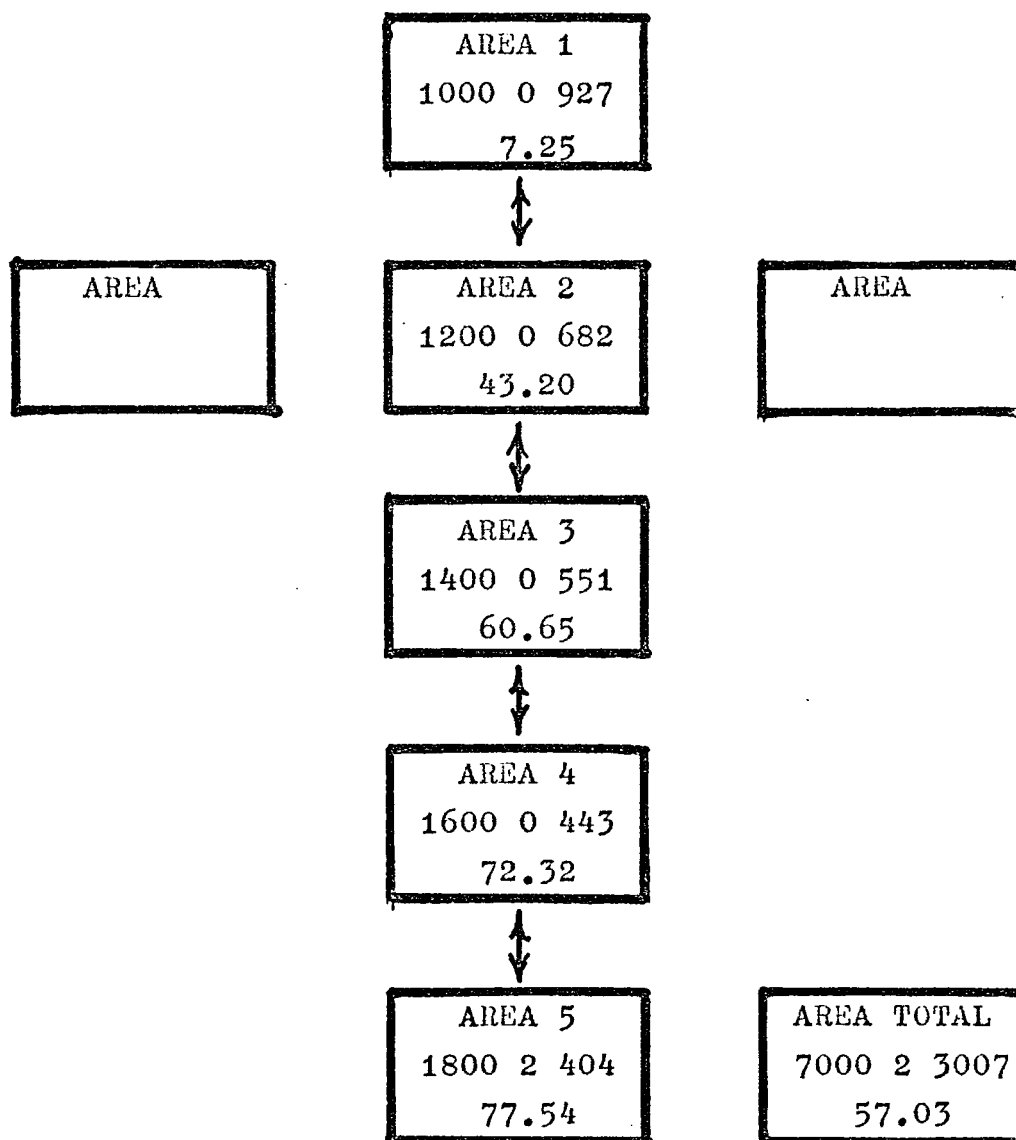
10,000

RELATIVE REMOVAL

RATE 1,000

DATA SET 5

5-L-.05



RUN TIME

7.43

MAXIMUM PERIOD

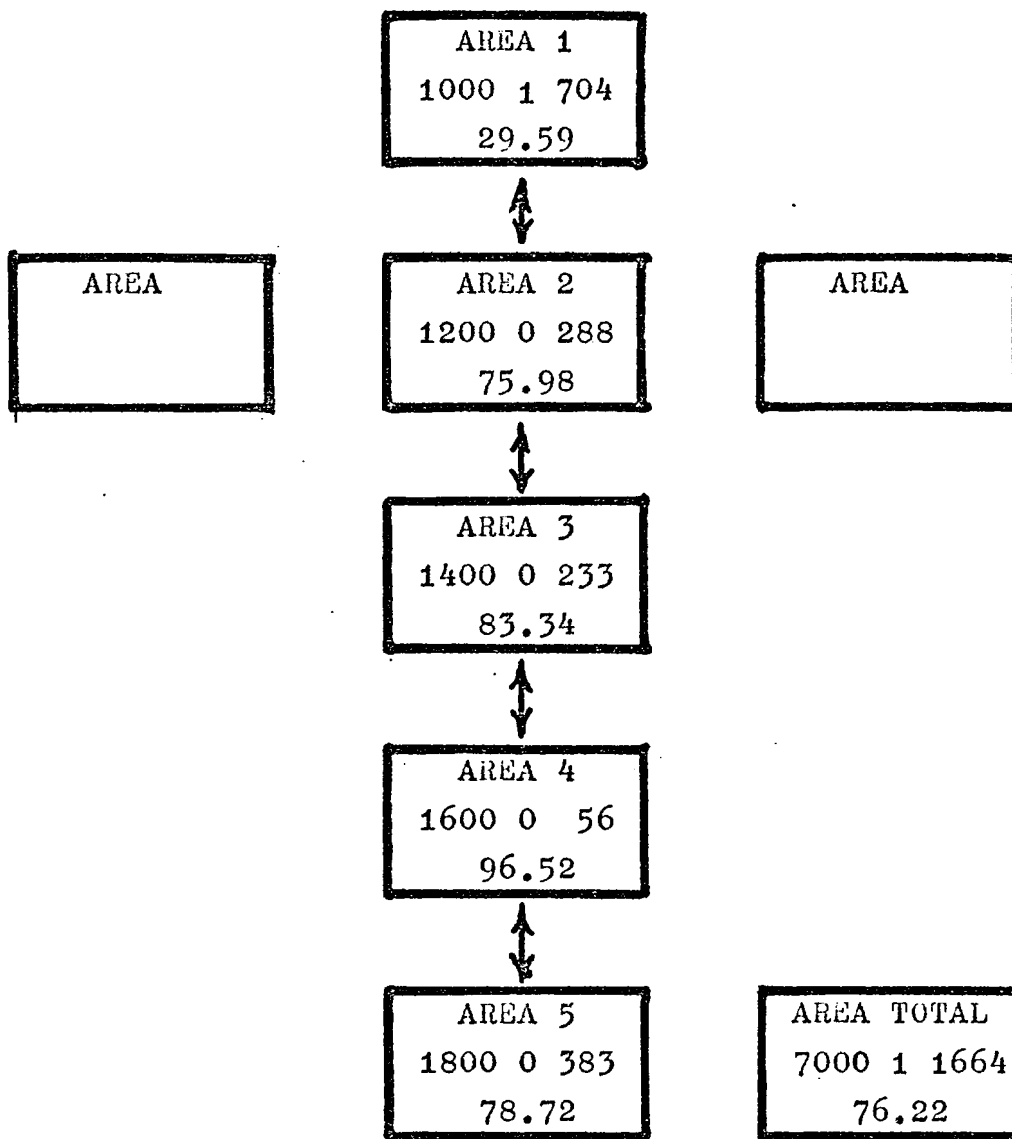
10,000

RELATIVE REMOVAL

RATE 1,000

DATA SET 6

5-L-.25



RUN TIME

5.24

MAXIMUM PERIOD

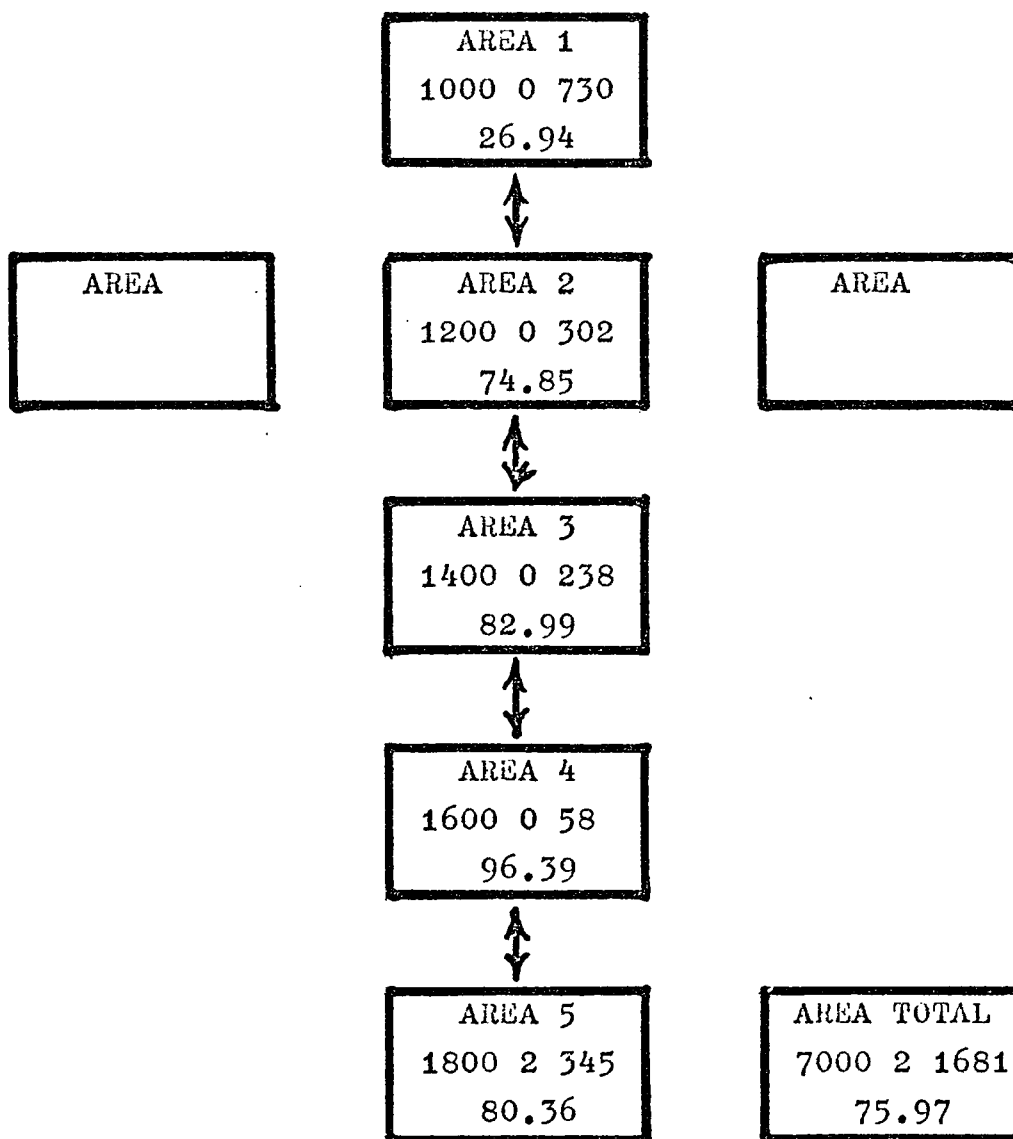
3,500

RELATIVE REMOVAL

RATE 1,000

DATA SET 7

5-L-.25



RUN TIME

5.45

MAXIMUM PERIOD

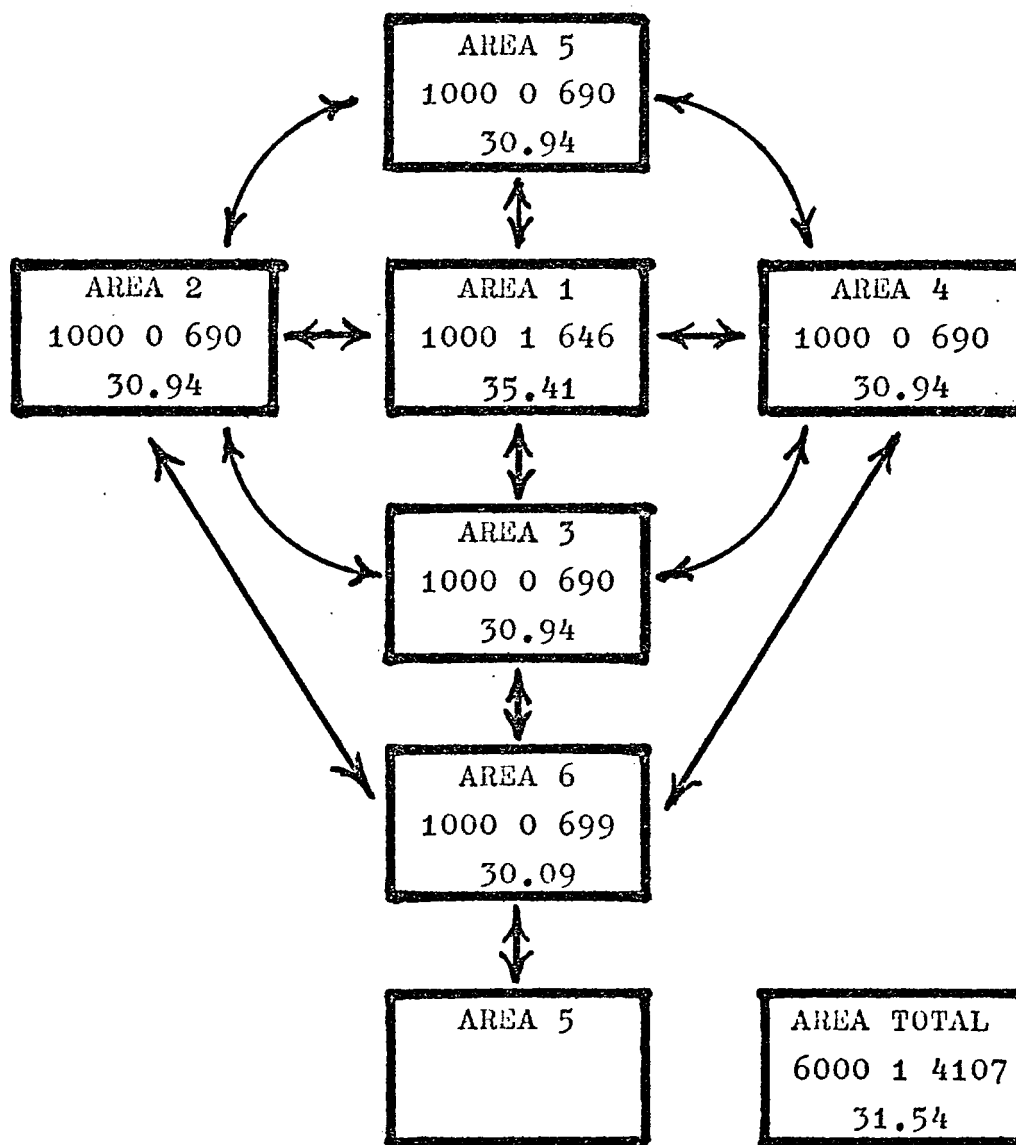
4,500

RELATIVE REMOVAL

RATE 1,000

DATA SET 8

6-C-.05



RUN TIME

8.17

MAXIMUM PERIOD

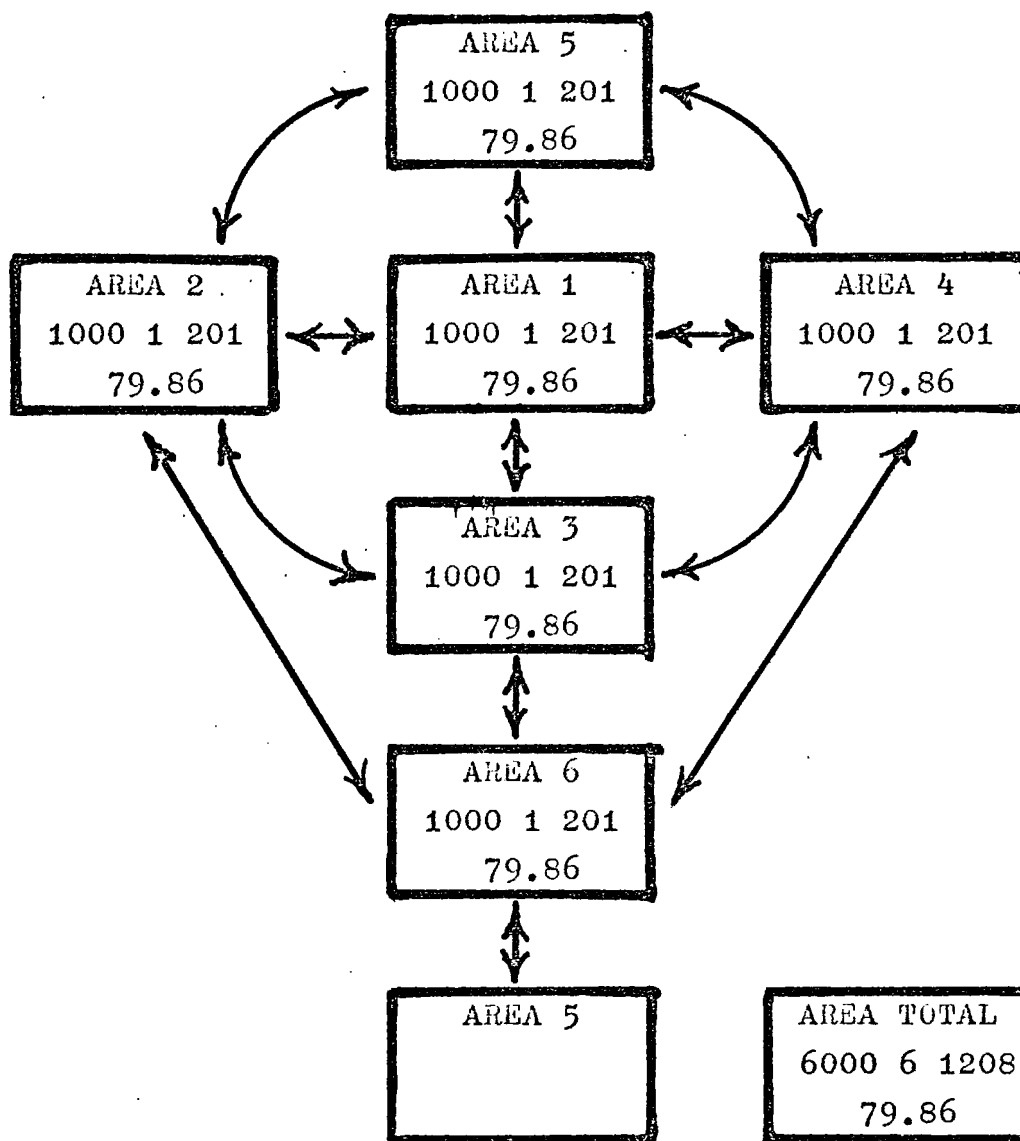
8,000

RELATIVE REMOVAL

RATE 1,000

DATA SET 9

6-C-.25



RUN TIME

5.47

MAXIMUM PERIOD

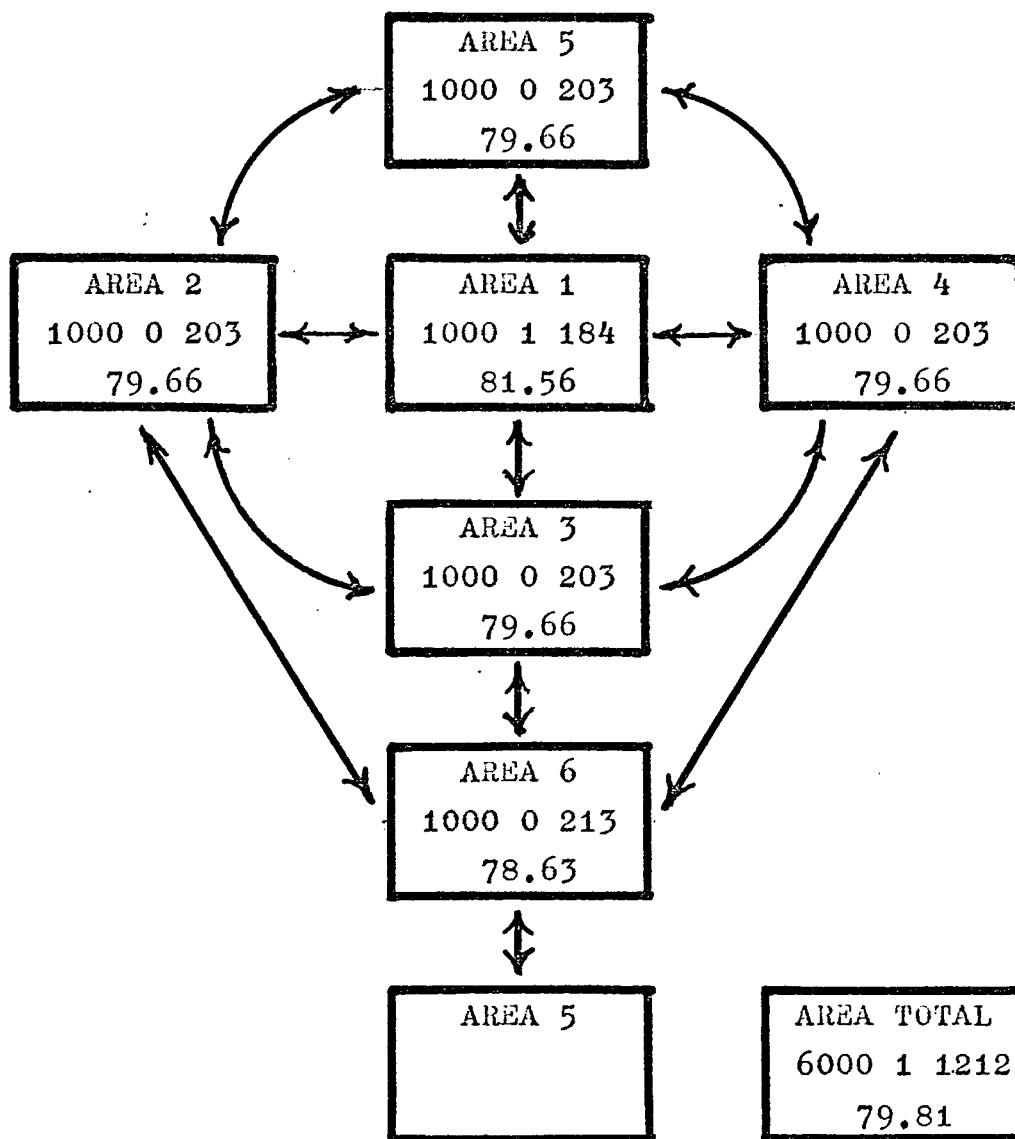
2,000

RELATIVE REMOVAL

RATE 1,000

DATA SET 10

6-C-.25



RUN TIME

5.37

MAXIMUM PERIOD

2,000

RELATIVE REMOVAL

RATE 1,000

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