

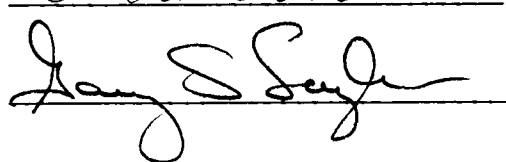
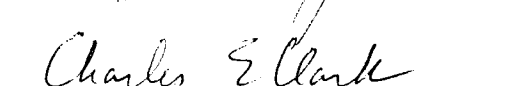
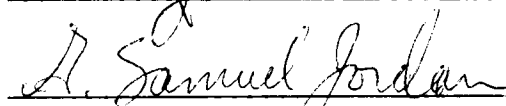
To the Graduate Council:

I am submitting herewith a dissertation written by Jose T. de Luna entitled "Analysis of Mathematical Models of Resource-Consumer-Toxicant Interactions." I have examined the final copy of this dissertation for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Doctor of Philosophy, with a major in Mathematics.



T.G. Hallam, Major Professor

We have read this dissertation  
and recommend its acceptance:



Accepted for the Council:



The Graduate School

ANALYSIS OF MATHEMATICAL MODELS OF  
RESOURCE-CONSUMER-TOXICANT  
INTERACTIONS

A Dissertation  
Presented for the  
Doctor of Philosophy  
Degree  
The University of Tennessee, Knoxville

Jose T. de Luna

August 1983

## DEDICATION

To my family, especially to daughters Joselin and Joylin.

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## ABSTRACT

A model of a toxicant-population interaction with variables population biomass, concentration of toxicant in the environment, and concentration of toxicant in the organism are investigated. First order kinetics are assumed for the toxicant while the population is governed by a modified Smith-type equation. Exposure is allowed through both environmental and food chain pathways. The non-additivity of environmental and food chain toxicant inputs is modelled by assuming that there is a coupling between the uptake rates of the different toxicant pathways. This feedback between the toxicant accumulation sources is assumed to depend reciprocally upon the environmental uptake parameter  $a_1$ . The main theoretical results focus on system level effects involving persistence and extinction of the population. Additionally, an approximation to the original model is shown to be adequate since error propagation is uniform in the toxicant components and when appropriate biological conditions hold, the reduced model yields environmentally safe predictions.

Three models of population-toxicant interactions that include an explicit representation for a dynamic resource have been developed and analyzed. Each model contains components representing population-resource subsystem, concentration of the toxicant in the environment, and concentration of the toxicant in the organism. The first resource-consumer-toxicant system includes a resource-consumer subsystem of the Leslie type; the second model uses a resource-consumer subsystem of the Gallopin type; the third deals with a model of the continuous culture type.

The reduced model is employed to model the concentration of the toxicant components.

Although the three resource-consumer-toxicant systems studied have modelling features that are different, each exhibits an interesting phenomenon related to ultimate population levels under stressed and unstressed conditions. The presence of toxicant in the system results in higher levels of resource needed to support a lower population level, that is, the equilibrium value of the population with toxicant is less than the population equilibrium without toxicant while the inequality is reversed for the resource equilibrium values. Thus, the presence of toxicant in the resource-consumer-toxicant models could be interpreted as resulting in higher maintenance costs and lower growth rates.

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## CHAPTER 1

### INTRODUCTION

#### 1.1 Introduction

We are all affected by pollution; hence, the need to study population-toxicant interactions cannot be overemphasized. Our current civilization and life styles utilize increasing amounts of energy, industrial chemicals, insecticides, and fertilizers. The production and use of these necessities often result in vast amounts of harmful waste products whose disposal leads to a general deterioration of environmental quality. Chemical contamination of air, water, soil, and food sources has become a threat to the existence of a number of plants and animal communities of the biosphere. Because of this potential danger, scientists in universities, in industries, and in government service have engaged in research studies on various aspects of pollution. These studies have resulted in the accumulation of data that need ordering and processing. Mathematical modelling, a tool that can synthesize such information, is employed here to quantify and predict effects of toxicants upon populations and communities.

#### 1.2. Model Types

Most studies dealing with the evaluation of potential hazards of toxic chemicals can be grouped into three model types, each of which is described and briefly discussed below.

(i) Ecosystem Models. Models under this category deal with whole ecosystems such as lakes (Scavia, et al., 1976; Park, et al. (CLEAN), 1975), aquatic communities (Lane and Levins, 1977), forests (Shugart and West (FORET), 1977), etc.. Other examples include a generalized ecosystem model developed by DeAngelis, et al. (1975), that can be used in theoretical studies of ecosystem structure and function. A model that investigates general patterns of ecosystem dynamics has been proposed by R.V. O'Neill (1976).

The available ecosystem models have been primarily designed for purposes other than to predict the effects of toxic substances on ecosystems. For example, the lake ecosystem model CLEAN has been designed as a diagnostic tool to study the effects of nutrient enrichments and other perturbations on the ecosystem. FORET has been designed to simulate both community dynamics and biomass of forest stands. Most ecosystem models, however, "are detailed enough for the effects of toxic substances on organismal physiology to be extrapolated to population and ecosystem effects" (Barnthouse, 1980). Although the process of extrapolating to real systems is a difficult task and ecosystem models are difficult to use because of their sizable number of equations (CLEAN, for example, has 28 coupled ordinary differential equations), they are useful in testing for trends or direction of impact and hence could be useful as an aid in formulating research strategies.

(ii) Fate Models. Models under this category generally "provide information on the extent of partitioning and life time of chemicals in air, water, and soil and on their potential bioaccumulation in the

biota." (Haque, et al., 1980). Examples include EXAMS (Lassiter, Baughman, and Burns, 1978), PEST (Park, et al., 1980), methyl mercury accumulation (Fagerstrom and Assel, 1973), and a fate model of micro-contaminants in a diverse aquatic food chain (Thomann, 1981). In general, fate models can predict the movement and fate of the toxicant and the potential for bioaccumulation, but cannot predict biological effects (Barnthouse, 1980).

(iii) Fate and Effects Models. These are recent "hybrid models" (Barnthouse, 1980) that can predict both fate and biological effects of chemicals. An example is provided by Falco and Mulkey (1976), in which a mathematical model for predicting the fate and transport of malathion in a river has been developed. The resulting equations are coupled with a simple dose-response model that predicts the reductions in standing crops of various fish species. Relatively little work has been done in this important area of ecotoxicology. Recent research in this area is discussed in the next section.

### 1.3. Modelling Perspective

Any modelling strategy that deals with risk assessment of population exposed to harmful chemicals must incorporate both fate and biological effects. Not all aspects of exposure should be modelled. Only those processes believed to be critically important should be studied. We believe that fate and biological effects are the two most important factors that determine the evolution of populations subjected to stress. Our aim, therefore, is to develop and analyze models that are suitable for predicting fate of chemicals and its

effects on exposed populations. Our analysis will be concerned mainly with the qualitative behavior of the model, that is, persistence or extinction of the exposed population and effects of stress on the ultimate population levels when the stressed population survives.

As we indicated earlier, models that can predict both the fate and biological effects of toxic chemicals are practically nonexistent. Recent efforts to develop models in this area are limited to the work done by Hallam, Clark, and Lassiter (1982) and by Hallam, Clark, and Jordan (1983).

We identify three types of fate and effects models, namely (in order of increasing complexity):

- (i) Fate and effects models - environmental toxicant uptake incorporated;
- (ii) Fate and effects models - environmental and food chain toxicant uptake incorporated;
- (iii) Same as in (ii) but with dynamic representation of the resource.

The mathematical models developed and analyzed by Hallam, et al. (1982; 1983) were under the first category. We are not aware of any mathematical models under categories (ii) and (iii). Specifically, the goal of this investigation is to develop and analyze models under the last two categories. The resulting models will be referred to as population-toxicant interaction models (Chapter 2) and resource-consumer-toxicant interaction models (Chapter 3), respectively.

#### 1.4. Modelling Approach

We start from the hypothesis that the dynamics of the population is known in the absence of a toxicant. We will introduce modifications into the population model to account for stress created by the exposure of organisms to a toxic chemical in both the environment and the resource of the population. With a few exceptions (i.e. the logistic dynamics), most population models can be modified (with varying degrees of difficulty) to incorporate toxic effects arising from uptake of toxicant from the environment and food pathways. First order kinetics are employed to describe the dynamics of the body burden  $c_0$ , and the environmental concentration,  $c_E$ . The mass balance equation is the fundamental principle around which the toxicant dynamics are formulated. The coupling between the population dynamics and body burden dynamics is achieved through a linear dose-response function. The dynamics of the resource is dependent on the particular model under consideration.

In Chapter 2, we will develop and analyze a population-toxicant interaction model based on a population dynamics proposed by F.E. Smith in 1963. A reduced model, formulated under the assumption that the population does little or no regulation of toxicant in the environment, is introduced. It is then argued that under natural conditions, the reduced model is an adequate, environmentally sound predictor of threshold values for toxicant concentration as well as the levels of persistence of the population. A numerical study of the interaction of Daphnia magna and dieldrin is presented to illustrate some of our conclusions.

In Chapter 3, we will study several resource-consumer-toxicant interaction models. Each model contains components representing the population-resource dynamics as well as the body burden dynamics and environmental concentration of the toxicant. The reduced model is employed for the concentration of the toxicant components. We generate numerical solutions for each of the resource-consumer-toxicant models considered and a phenomenon predicted by our analytical results and common to all models studied is graphically illustrated.

In Chapter 4, we will study and analyze a Lotka-Volterra type predator-prey system and a comparison of its asymptotic behavior with the resource-consumer-toxicant models will be undertaken.

## CHAPTER 2

### A POPULATION-TOXICANT MODEL AND SIMULATION

#### 1.1. Introduction

The Verhulst-Pearl logistic equation, a frequently employed dynamic model of a population, requires that the per capita growth rate is limited by the proportion of the population size that has not been attained. While recent modifications of the logistic equation include adjustments to account for environmental perturbations (Coleman, 1978; Hallam and Clark, 1981) and for environmental toxicant effects (Hallam, Clark, and Lassiter, 1982; Hallam, Clark, and Jordan, 1983), there is no mechanism present in the logistic model to allow formulation of stress as propagated through the resources of the population.

Not all organisms competing for some common environmental limiting factors behave according to logistic dynamics. F.E. Smith (1963) proposed a model for the dynamics of a population where growth limitations were based upon the proportion of available resources not utilized. This hypothesis allows incorporation of both environmental and food chain effects of stress and will be one of the major concerns of this chapter.

Related to the problem of formulating stress as propagated through the resource of the population is the issue of the ecological significance of toxicant accumulation via the food pathway. Experimental data indicate that for dichlorodiphenyltrichloroethane (DDT) (Carson, 1962; Hunt and Bischoff, 1960; Jarvinen, et al., 1977) endrin (Jarvinen, et al., 1977), hexachlorobenzene (HCB) (Niimi and Cho, 1981), and

Mirex and Kepone (Skaar, et al., 1981), the food chain input is important. However, other chemicals do not apparently share this cumulative effect; chlorinated hydrocarbons (Moriarty, 1975; Rosenberg, 1975), di (2-ethylhexyl) phthalate (DEHP), 1, 2, 4 - trichlorobenzene (TCB) leptophos (Macek, et al.), and dieldrin (Chadwick and Brocksen, 1969; Reinert, 1972), when taken in by the food chain pathway, do not significantly accumulate in the test organisms. Hence, one other goal in this chapter is to have an understanding of the process of uptake of toxicant from the environment and from the food chain pathway.

## 2.2. Toxicant Stress Effects in Smith's Model

Smith's model has the form

$$\frac{dx}{dt} = rx \left[ \frac{K - x}{K + \frac{r}{c} x} \right] \quad (2.2.1)$$

where  $x = x(t)$  denotes the biomass of the population at time  $t$ ,  $r$  is the rate of increase with abundant resource,  $K$  is the value of  $x$  at saturation ( $\frac{dx}{dt}$  is zero), and  $c$  is the rate of replacement of mass in the population at saturation.

It is necessary to modify Smith's model to account for stress effects created by the introduction of a toxic chemical into both the environment and the resource of an organism. To explain our modifications, it is convenient to review the hypothesis imposed by Smith (1963) in his development. An hypothesis that governs the form of the model is that the per capita growth rate of the population is proportional to some limiting factor,  $P$ , not yet utilized. In a resource-limited

system  $P$  can represent the proportion of food supply not momentarily being used by the population; specifically, Smith employed  $P = 1 - F/T$ , where  $F$  is the rate at which a population of size  $x$  uses food and  $T$  is the total rate of food utilization at saturation. Assuming  $F$  has the form

$$F = c_1 x + c_2 \frac{dx}{dt}$$

(the first term represents the rate of food uptake for maintenance and the second term models the rate of food uptake for growth) results in (2.2.1), where  $c = c_1/c_2$ .

Equation (2.2.1) has a per capita growth rate that is concave upward in contrast to the linear growth rate of the logistic equation. In spite of this difference, it suffers from many of the deficiencies of the logistic equation delineated in Hallam and Clark (1981). In particular, modifications are necessary to make (2.2.1) useful when the population is stressed by a perturbation.

For clarity, we make these changes in an incremental manner. First, the model is written as

$$\frac{dx}{dt} = x(r - a \frac{F}{T}) \quad (2.2.2)$$

where the parameter  $a$  is a measure of the population's response to stress effects (Hallam and Clark, 1981). This representation also bypasses modelling difficulties associated with  $r$  appearing in (2.2.1) as a multiplicative factor, where the growth rate can dominate the dynamics of the population.

In general, the population's carrying capacity,  $B$ , and the steady state of the population,  $K$ , need not be the same (Hallam and Clark, 1981). The carrying capacity need not be an equilibrium, so the saturation and equilibrium value,  $K$ , as used in (2.2.1) should be reinterpreted. When  $x = B$ , then  $F = T$  and

$$T = c_1 B + c_2 \left. \frac{dx}{dt} \right|_{x=B} . \quad (2.2.3)$$

Substituting (2.2.3) in (2.2.2), we obtain

$$\frac{dx}{dt} = x \left[ r - \frac{a(c_1 x + c_2 \frac{dx}{dt})}{c_1 B + c_2 \left. \frac{dx}{dt} \right|_{x=B}} \right] \quad (2.2.4)$$

If we let  $\alpha = \left. \frac{dx}{dt} \right|_{x=B}$  and evaluate (2.2.4) at  $x = B$ , we obtain  $\alpha = B(r - a)$ . Consequently,

$$T = c_1 B + c_2 B(r - a) . \quad (2.2.5)$$

From (2.2.4) it follows that

$$\frac{dx}{dt} = x \frac{[rB(c + r - a) - acx]}{B(c + r - a) + ax} \quad (2.2.6)$$

When all parameters are constant and  $a < r + c$ , then (2.2.6) has a feasible equilibrium given by  $x^* = rB(c + r - a)/ac$ . We will assume throughout the remainder of this chapter that the inequality  $a < r + c$  holds. The model (2.2.6) is a reformulation of (2.2.1) that allows the incorporation of effects of stress.

### 2.3. Toxicant Dynamics

The mass balance equation is the foundation of our development of the dynamics of the organismal toxicant concentration,  $c_0(t)$ , and the environmental toxicant concentration  $c_E(t)$ . The exogenous input to the body burden  $c_0$  is assumed to be from the environment at a rate proportional to the environmental concentration:  $G_1 = a_1 c_E$ , where  $a_1$  is the population's rate per unit mass toxicant withdrawal efficiency. An alternative approach would be to assume that a diffusion mechanism governs organismal uptake of toxicant from the environment, that is,  $G_1 = a_1(nc_E - c_0)$ . But this approach leads to the same dynamics for  $c_0$  given in (2.3.1).

To approximate the process of uptake of toxicant through the food chain pathway, we assume that the toxicant concentration  $\theta$  in the resource is a constant. Let  $\beta$  denote the average rate of food intake per unit organismal mass.

If toxicant inputs are additive in the sense that the total organism toxicant concentration is the sum of environmental and food chain inputs, then we should assume that the total toxicant gain,  $G$ , is  $G = a_1 c_E + \theta \beta$ . However, inputs are not, in general, additive (Macek, et al., 1979; Reinert, 1972). Thus, there must be a coupling between the uptake rates of different toxicant pathways. This feedback between the toxicant accumulation sources is assumed here to depend reciprocally upon the environmental uptake parameter  $a_1$ . The formulation for  $G$  is

$$G = a_1 c_E + \frac{d_1}{a_1} \theta \beta .$$

The losses of toxicant from the organism are assumed given by

$$L = \ell_1 c_0(t) + \ell_2 c_0(t)$$

where  $\ell_1$  and  $\ell_2$  are egestion and depuration rates respectively.

Combining the gain and loss formulations results in

$$\frac{dc_0}{dt} = a_1 c_E + \frac{d_1}{a_1} \theta \beta - \ell_1 c_0 - \ell_2 c_0 \quad (2.3.1)$$

The dynamics of  $c_E$  are assumed to be governed by

$$\frac{dc_E}{dt} = g(t) + k_1 \ell_1 c_0^x - k_1 a_1 c_E^x - k_2 c_E. \quad (2.3.2)$$

The first term  $g$  in (2.3.2) represents exogenous input of toxicant into the environment. The second term in (2.3.2) represents total toxicant egested. The third term represents total toxicant uptake from the environment. The last term models losses due to detoxifying process such as photolysis, hydrolysis, volatilization, etc., (Lassiter, et al., 1978).

## 2.4. Toxicant Stress

Exposure to toxicant may lead to change in fecundity and mortality rates of a population. This stress can be modelled by assuming the growth rate of the population is a function of the body burden

$$r = r(c_0) = r_0 - H(c_0) \quad (2.4.1)$$

where  $H$  is a nondecreasing function of  $c_0$  with  $H(0) = 0$  and  $r_0$  is the intrinsic growth rate of the population.  $H(c_0)$  is a dose-response function and has many forms in the open literature. One commonly used form is the linear dose-response function

$$H(c_0) = r_1 c_0 . \quad (2.4.2)$$

The linear dose-response function does occur in population-toxicant interactions and has been used successfully by Hallam et al. (1983). Another common type of dose-response function is a sigmoid dose-response curve (Butler, 1978; Filov et al., 1979). In performing the model analysis, however, for convenience and for reasons discussed in Section 2.10, we will assume  $H(c_0)$  is given by (2.4.2).

Toxicant stress effects may be expressed further through the parameter  $c_1$  of (2.2.3) which measures the resource uptake required for maintenance of the population. Since stressed populations may require more resources to maintain than unstressed populations of the same density,  $c_1$  should be a function of body burden (and perhaps, its derivatives as well). However, in this chapter, we assume that  $c_1$  is constant and treat this aspect of toxic stress in the next chapter which deals with consumer-toxicant interactions.

## 2.5. The Complete Model

We now incorporate in (2.2.4) toxicant stress effects as modelled by (2.4.1) to obtain

$$\frac{dx}{dt} = x \left[ r(c_0) - \frac{a(c_1 x + c_2 \frac{dx}{dt})}{c_1 B + c_2 \frac{dx}{dt}|_{x=B}} \right] .$$

Solving for  $\frac{dx}{dt}$  and using the fact that  $\frac{dx}{dt}|_{x=B} = B(r_0 - a)$ , yields

$$\frac{dx}{dt} = \frac{x[r(c_0) B(r_0 + c - a) - acx]}{B(c + r_0 - a) + ax} \quad (2.5.1)$$

The model (C) consists of equations (2.5.1), (2.3.1), and (2.3.2)

together with the linear dose-response function  $H(c_0) = r_1 c_0$  :

$$\frac{dx}{dt} = \frac{x[r(c_0) B(c + r_0 - a) - acx]}{B(c + r_0 - a) + ax}$$

$$\frac{dc_0}{dt} = a_1 c_E + \frac{d_1}{a_1} \theta \beta - \ell_1 c_0 - \ell_2 c_0$$

$$\frac{dc_E}{dt} = g(t) + k_1 \ell_1 c_0 x - k_1 a_1 c_E x - k_2 c_E$$

where  $r(c_0) = r_0 - r_1 c_0$ . The initial conditions are

$$x(0) = x_0 > 0, \quad c_0(0) = 0, \quad c_E(0) = c_{E0} > 0.$$

## 2.6. Model Analysis

Define  $g_m = \inf_{t \in \mathbb{R}_+} g(t)$  and  $g_M = \sup_{t \in \mathbb{R}_+} g(t)$  and assume  $g_m \geq 0$  and  $g_M < \infty$  where  $\mathbb{R}_+ = [0, \infty)$ . Two properties of the

solutions that are needed in subsequent analysis are contained in the next result.

THEOREM 2.6.1. Each component of the trajectory  $(x(t), c_0(t), c_E(t))$  is positive and bounded on  $\mathbb{R}_+$ .

PROOF: Both of equations (2.3.1) and (2.3.2) are linear in their respective variables; this fact allows integral representations of  $c_0$  and  $c_E$  :

$$c_0(t) = \frac{d_1 \theta \beta}{a_1(\ell_1 + \ell_2)} \{1 - \exp[-(\ell_1 + \ell_2)t]\} \\ + a_1 \exp[-(\ell_1 + \ell_2)t] \int_0^t c_E(s) \exp[(\ell_1 + \ell_2)s] ds \quad (2.6.1)$$

$$c_E(t) = c_{E0} \exp\{-\int_0^t [k_1 a_1 x(s) + k_2] ds\} \\ + \exp\{-\int_0^t [k_1 a_1 x(s) + k_2] ds\} \int_0^t \{g(s) + k_1 \ell_1 c_0(s) x(s)\} \cdot \\ \cdot \exp\{\int_0^s [k_1 a_1 x(s_1) + k_2] ds_1\} ds . \quad (2.6.2)$$

To obtain an integral representation for  $x$ , we note from (2.5.1) that we can write

$$\left(\frac{T_0 + ax}{x}\right) \frac{dx}{dt} + acx = T_0(r_0 - r_1 c_0)$$

where  $T_0 = B(c + r_0 - a)$ . An integration leads to

$$\frac{e^{-ax_0/T_0}}{x_0} x(t) e^{ax(t)/T_0} e^{(ac/T_0) \int_0^t x(s) ds} \quad (2.6.3)$$

$$= e^{\int_0^t [r_0 - r_1 c_0(s)] ds} \quad (2.6.3.) \text{ con't.}$$

The positivity of  $x(t)$  follows from (2.6.3). We demonstrate the positivity of  $c_E(t)$  by contradiction. Hence, suppose  $c_E(t_1) < 0$ . Since  $c_{E0} > 0$  and  $c_E(t)$  is continuous, there exists a  $t_0 > 0$  such that  $c_E(t_0) = 0$  and  $c_E(t) > 0$  for  $0 \leq t < t_0$ . From (2.6.2), it follows that

$$c_{E0} = - \int_0^{t_0} \{g(s) + k_1 \ell_1 c_0(s) x(s)\} \cdot e^{\int_0^s [k_1 a_1 x(s_1) + k_2] ds_1} ds .$$

Hence, in some subinterval  $[t_1, t_2] \subseteq [0, t_0]$ , we must have  $g(t) + k_1 \ell_1 c_0(t) x(t) < 0$ . Since  $g(t) \geq 0$ , then  $c_0(t) x(t) < 0$  for  $t \in [t_1, t_2]$ . But  $x(t) > 0$ . Hence  $c_0(t) < 0$  for  $t \in [t_1, t_2]$ . But from (2.6.1) and from our definition of  $t_0$ , we see that  $c_0(t) \geq 0$  for  $t \in [t_1, t_2]$ , which is a contradiction. Thus,  $c_E(t)$  is positive for all  $t$  in  $\mathbb{R}_+$ . The positivity of  $c_0(t)$  follows immediately from (2.6.1).

The boundedness of the  $x$ -component follows from (2.5.1), since by differential inequality theory (Lakshmikantham and Leela, 1969), this component is bounded by the solution  $y$  of the logistic problem

$$\frac{dy}{dt} = \frac{y[r_0 T_0 - a c y]}{T_0}, \quad y(0) = x_0 .$$

In particular,  $y(t) \leq X_M$  for  $t \geq 0$  (Hallam, Clark, and Jordan, 1983), where

$$X_M = \max\{x_0, \frac{r_0 T_0}{ac}\}.$$

Hence,  $x(t) \leq X_M$  for  $t \geq 0$ .

The boundedness of  $c_0(t)$  and  $c_E(t)$  follows from an application of L'Hopital's rule to (2.6.1) and (2.6.2). In particular, since  $c_0$  and  $c_E$  are concentrations, we must have  $0 \leq c_0(t) \leq 1$  and  $0 \leq c_E(t) \leq 1$ .

### Persistence and Extinction Results

We now present a sufficient condition for persistence and a sufficient condition for extinction of the population.

THEOREM 2.6.2. Let

$$\rho_M = \frac{d_1 \theta \beta}{a_1(\ell_1 + \ell_2)} + \frac{a_1(g_M + k_1 \ell_1 X_M)}{(\ell_1 + \ell_2) k_2}, \text{ and}$$

$$\rho_m = \frac{d_1 \theta \beta}{a_1(\ell_1 + \ell_2)} + \frac{a_1 g_m}{(\ell_1 + \ell_2)(k_1 a_1 X_M + k_2)}.$$

- (i) If  $r_0 > r_1 \rho_M$ , the the population is persistent; that is,  $\limsup_{t \rightarrow \infty} x(t) > 0$  for all solutions of (C).
- (ii) If  $r_0 < r_1 \rho_m$ , then the population goes to extinction, that is,  $\lim_{t \rightarrow \infty} x(t) = 0$  for all solutions of model (C). Moreover, in this situation,  $x \in L^1(\mathbb{R}_+)$ .

PROOF: To prove (i) we first show that whenever  $r_0 > r_1 \rho_M$ , then  $r_0 > r_1 c_0(t)$  for all  $t$  sufficiently large. To accomplish this, we substitute (2.6.2) into (2.6.1) to obtain

$$c_0(t) = I_1(t) + I_2(t) + I_3(t), \quad (2.6.4)$$

where

$$\begin{aligned} I_1(t) &= \frac{d_1 \theta \beta}{a_1(\ell_1 + \ell_2)} [1 - e^{-(\ell_1 + \ell_2)t}] . \\ I_2(t) &= a_1 e^{-(\ell_1 + \ell_2)t} \int_0^t c_{E0} e^{-\int_0^s [k_1 a_1 x(s_1) + k_2] ds_1} \cdot \\ &\quad \cdot e^{(\ell_1 + \ell_2)s} ds , \\ I_3(t) &= a_1 e^{-(\ell_1 + \ell_2)t} \int_0^t e^{-\int_0^s [k_1 a_1 x(s_1) + k_2] ds_1} \cdot \\ &\quad \cdot \left\{ \int_0^s [g(s_1) + k_1 \ell_1 c_0(s_1) x(s_1)] e^{\int_0^{s_1} [k_1 a_1 x(s_2) + k_2] ds_2} \right. \\ &\quad \left. \cdot e^{(\ell_1 + \ell_2)s} ds \right\} . \end{aligned} \quad (2.6.5)$$

From (2.6.5), we can write

$$\begin{aligned} I_3(t) &\leq a_1 e^{-(\ell_1 + \ell_2)t} \int_0^t \left( \int_0^s [g_1 + k_1 \ell_1 x_M] e^{-\int_{s_1}^s k_2 ds_2} \right. \\ &\quad \left. \cdot e^{(\ell_1 + \ell_2)s} ds \right) . \end{aligned}$$

Integrating the right hand side of this inequality results in

$$I_3(t) \leq \frac{a_1[g_M + k_1 \ell_1 X_M]}{k_2} \left[ \frac{1 - e^{-(\ell_1 + \ell_2)t}}{\ell_1 + \ell_2} - \frac{(e^{-k_2 t} - e^{-(\ell_1 + \ell_2)t})}{\ell_1 + \ell_2 - k_2} \right] .$$

Hence, from (2.6.4),

$$c_0(t) \leq \rho_M + \underline{o}(1) ,$$

where  $\underline{o}(1)$  represents a function that approaches zero as  $t$  approaches infinity. In particular,

$$\begin{aligned} \underline{o}(1) = & - \frac{d_1 \theta \beta}{a_1(\ell_1 + \ell_2)} e^{-(\ell_1 + \ell_2)t} + I_2(t) \\ & - \frac{a_1[g_M + k_1 \ell_1 X_M]}{k_2} \left[ \frac{e^{-(\ell_1 + \ell_2)t}}{(\ell_1 + \ell_2)} + \frac{e^{-k_2 t} - e^{-(\ell_1 + \ell_2)t}}{\ell_1 + \ell_2 - k_2} \right] . \end{aligned}$$

Consequently,

$$r_0 - r_1 c_0 \geq r_0 - r_1(\rho_M + \underline{o}(1)) , \quad (2.6.6)$$

and so, for  $t$  sufficiently large,

$$r_0 - r_1 c_0(t) \geq r_0 - r_1 \rho_M > 0 .$$

Hence, taking limits in (2.6.3) yields

$$\lim_{t \rightarrow \infty} K_0 x(t) e^{ax(t)/T_0} \cdot e^{-(ac/T_0) \int_0^t x(s) ds} = \infty,$$

where  $K_0 = \frac{e^{-ax_0/T_0}}{x_0}$ . This implies that  $\limsup_{t \rightarrow \infty} x(t) > 0$ . For if

extinction occurs, then  $\lim_{t \rightarrow \infty} x(t) = 0$ . If  $x(t) \in L^1(\mathbb{R}_+)$ , then we

are led to a contradiction. If  $x(t) \notin L^1(\mathbb{R}_+)$ , then both the numerator and denominator of

$$\psi(t) = x(t)/e^{-\int_0^t x(s) ds}$$

approaches zero as  $t$  approaches infinity. An application of L'Hopital's Rule yields

$$0 \leq \lim_{t \rightarrow \infty} \psi(t) \leq \lim_{t \rightarrow \infty} \left\{ e^{\int_0^t x(s) ds} [-(r_0 - r_1 \rho_M)] + o(1) \right\} = -\infty.$$

This contradiction shows that the population persists.

To prove (ii) a lower bound on  $c_0$  is required. From (2.6.5) we can write

$$I_3(t) \geq a_1 e^{-(\ell_1 + \ell_2)t} \int_0^t \left( \int_0^s g_m e^{-\int_{s_1}^s [k_1 a_1 x_M + k_2] ds_2} ds_1 \right) e^{(\ell_1 + \ell_2)s} ds$$

$$\cdot \int_0^t \left( \int_0^s g_m e^{-\int_{s_1}^s [k_1 a_1 x_M + k_2] ds_2} ds_1 \right) e^{(\ell_1 + \ell_2)s} ds$$

Integrating the right hand side of this inequality results in

$$I_3(t) \geq \frac{a_1 g_m}{k_2 + k_1 a_1 X_M} e^{-(\ell_1 + \ell_2)t} \left[ \frac{e^{(\ell_1 + \ell_2)t} - 1}{\ell_1 + \ell_2} - \frac{(\ell_1 + \ell_2 - k_1 a_1 X_M - k_2)t - 1}{\ell_1 + \ell_2 - k_1 a_1 X_M - k_2} \right].$$

Hence from (2.6.4)

$$c_0(t) \geq \frac{d_1 \theta \beta}{a_1(\ell_1 + \ell_2)} + \frac{a_1 g_m}{(\ell_1 + \ell_2)(k_1 a_1 X_M + k_2)} + o(1),$$

or

$$c_0(t) \geq \rho_m + o(1).$$

Consequently, for  $t$  sufficiently large,

$$r_0 - r_1 c_0(t) \leq r_0 - r_1 \rho_m + o(1) < 0.$$

As in the proof of (i), we use (2.6.3) to obtain

$$\lim_{t \rightarrow \infty} K_0 x(t) e^{ax(t)/T_0} e^{(ac/T_0) \int_0^t x(s) ds} = 0.$$

This implies that  $\lim_{t \rightarrow \infty} x(t) = 0$ .

It remains to demonstrate that  $x \in L^1(\mathbb{R}_+)$ . Suppose again for the purpose of contradiction that  $x \notin L^1(\mathbb{R}_+)$ . Since

$$K_0 x(t) e^{ax(t)/T_0} e^{\frac{ac}{T_0} \int_0^t x(s) ds} \geq K_0 x(t) e^{\frac{ac}{T_0} \int_0^t x(s) ds}$$

an application of L'Hopital's Rule yields

$$0 = \frac{K_0 T_0}{ac} \lim_{t \rightarrow \infty} e^{\frac{ac}{T_0} \int_0^t x(s) ds} \{-[r_0 - (r_1 \rho_M + o(1))]\} = \infty$$

which again is a contradiction. This shows that  $x \in L^1(\mathbb{R}_+)$  and completes the proof of the theorem.

REMARKS. It is not easy to obtain persistence or extinction criteria when the conditions of Theorem 2.6.2 are not satisfied. This difficulty lies in the dependence of  $\rho_m$  and  $\rho_M$  on the fluctuations of  $g$  and on the population bound  $X_M$ . If  $g$  fluctuates widely or if  $X_M$  is large, then  $\rho_m$  and  $\rho_M$  need not be good estimates of  $c_0$ .

## 2.7. A Simplified Model

To obtain more detailed results and to compare strength of the toxicant pathways to the target organisms, it is assumed that  $\lim_{t \rightarrow \infty} g(t) = g_\infty$  exists. We also make an additional simplification of the model (C) based upon an hypothesis that the environmental medium is much larger than that of the organisms so the environmental concentration of toxicant is unaffected by continued uptake and egestion by organisms (Filov, et al., 1979). Thus, an approximation to (2.3.2) could regard exogenous toxicant input,  $g$ , and environmental physical-chemical detoxifying processes,  $k_2 c_E$ , as dominant. This yields

$$\frac{dc_E}{dt} = g(t) - k_2 c_E. \quad (2.7.1)$$

We will refer to the model consisting of (2.5.1), (2.3.1), and (2.7.1), with initial conditions as in model (C), as the reduced model (R).

$$\frac{dx}{dt} = \frac{x[T_0(r_0 - r_1 c_0) - acx]}{T_0 + ax}$$

$$\frac{dc_0}{dt} = a_1 c_E + \frac{d_1 \theta \beta}{a_1} - (\ell_1 + \ell_2) c_0$$

$$\frac{dc_E}{dt} = g(t) - k_2 c_E.$$

#### Asymptotic Behavior of the Reduced Model

The persistence and extinction criteria of Theorem 2.6.2 are valid for the reduced model since (R) is the special case of (C) with  $k_1 = 0$ . In the situation where the exogenous input of toxicant approaches a limit the complete asymptotic behavior of (R) can be determined.

THEOREM 2.7.1. Let  $\lim_{t \rightarrow \infty} g(t) = g_\infty$ . Then all components of (R)

have limits and

$$(i) \quad \lim_{t \rightarrow \infty} c_E(t) = g_\infty / k_2 \equiv c_E(\infty),$$

$$(ii) \quad \lim_{t \rightarrow \infty} c_0(t) = \frac{d_1 \theta \beta}{a_1 (\ell_1 + \ell_2)} + \frac{a_1 g_\infty}{k_2 (\ell_1 + \ell_2)} \equiv c_0(\infty),$$

$$(iii) \quad \lim_{t \rightarrow \infty} x(t) = 0, \text{ provided } r_0 \leq r_1 c_0(\infty),$$

$$(iv) \quad \lim_{t \rightarrow \infty} x(t) = \frac{T_0(r_0 - r_1 c_0(\infty))}{ac}, \text{ provided } r_0 > r_1 c_0(\infty).$$

PROOF. When the input function approaches a limit, then the limits in (i) and (ii) can be computed directly from the integral equation (2.6.1) and (2.6.2) with  $k_1 = 0$ , by an application of L'Hopital's Rule.

We now show that  $\lim_{t \rightarrow \infty} x(t)$  exists. Suppose this limit does not exist. Let  $\underline{x} = \liminf_{t \rightarrow \infty} x(t)$  and  $\bar{x} = \limsup_{t \rightarrow \infty} x(t)$ . Then  $\underline{x} \neq \bar{x}$ .

Select a sequence  $\{t_n^M\}_{n=1}^\infty$  such that

$$\lim_{n \rightarrow \infty} x(t_n^M) = \bar{x} \quad \text{and} \quad x'(t_n^M) = 0$$

for all  $n = 1, 2, \dots$ . Similarly, choose  $\{t_n^m\}_{n=1}^\infty$  such that

$$\lim_{n \rightarrow \infty} x(t_n^m) = \underline{x} \quad \text{and} \quad x'(t_n^m) = 0$$

for all  $n = 1, 2, \dots$ . From (2.5.1), we obtain

$$r(c_0(t_n^M)T_0 - acx(t_n^M)) = r(c_0(t_n^m)T_0 - acx(t_n^m)).$$

Letting  $n \rightarrow \infty$  and using the fact that  $\lim_{t \rightarrow \infty} c_0(t)$  exists, we obtain

$\bar{x} = \underline{x}$ , which is a contradiction. Thus,  $\lim_{t \rightarrow \infty} x(t)$  exists for all

solutions of (R).

We now show that (iv) is true. If  $r_0 > r_1 c_0(\infty)$ , then using an argument similar to the proof of Theorem 2.6.2(i), one can show that  $\lim_{t \rightarrow \infty} x(t) > 0$ . Hence,  $x$  must approach the value claimed by (iv).

To prove (iii), we need to consider two cases.

Case 1.  $r_0 < r_1 c_0(\infty)$ .

Since  $r_0 - r_1 c_0(\infty) < 0$ , then  $\int_0^\infty [r_0 - r_1 c_0(t)] dt = -\infty$ . Using

(2.6.3) and taking limits, we obtain

$$\begin{aligned} \lim_{t \rightarrow \infty} K_0 x(t) e^{\frac{ax(t)}{T_0}} &= e^{\frac{ac}{T_0} \int_0^t x(s) ds} \\ &= \lim_{t \rightarrow \infty} e^{\int_0^t (r_0 - r_1 c_0(s)) ds} \\ &= 0. \end{aligned}$$

Since  $ax(t)/T_0$  and  $(ac/T_0) \int_0^t x(s) ds$  are positive for all  $t$ ,

we must have  $\lim_{t \rightarrow \infty} x(t) = 0$  since  $\lim_{t \rightarrow \infty} x(t) > 0$  leads to

$$\lim_{t \rightarrow \infty} K_0 x(t) e^{\frac{ax(t)}{T_0}} = e^{\frac{ac}{T_0} \int_0^t x(s) ds} > 0, \text{ which is a contradiction.}$$

Case 2.  $r_0 = r_1 c_0(\infty)$ .

We demonstrate the conclusion by contradiction. Assume

$\lim_{t \rightarrow \infty} x(t) > 0$ . Consider (2.5.1). Since  $\lim_{t \rightarrow \infty} x(t)$  exists, we must

have  $\lim_{t \rightarrow \infty} dx/dt = 0$ . Consequently, we must have

$$\lim_{t \rightarrow \infty} [(r_0 - r_1 c_0(t)) B(r_0 + c - a) - acx(t)] = 0,$$

or equivalently

$$\lim_{t \rightarrow \infty} x(t) = \frac{(r_0 - r_1 c_0(\infty)) T_0}{ac} = 0$$

which is not possible. This completes the proof.

REMARKS. Theorem 2.7.1, parts (iii) and (iv) indicate that persistence and extinction in model (R) are governed by the relationship between  $r_0$  and  $r_1 c_0(\infty)$ . Moreover, this result is precise in that  $r_0 > r_1 c_0(\infty)$  is necessary and sufficient for persistence.

We recall in Theorem 2.6.2(ii) that if  $r_0 < r_1 \rho_m$ , then we have not only  $\lim_{t \rightarrow \infty} x(t) = 0$ , but also  $x \in L^1(\mathbb{R}_+)$ . These conclusions

remain valid in Theorem 2.7.1.(iii), provided  $r_0 < r_1 c_0(\infty)$ . But if  $r_0 = r_1 c_0(\infty)$ , then  $x$  need not be in  $L^1(\mathbb{R}_+)$ . Such is the case for example when  $r_0 - r_1 c_0(t) \in L^1(\mathbb{R}_+)$ , or if  $r_0 - r_1 c_0 \notin L^1(\mathbb{R}_+)$  but  $c_0(t) \rightarrow c_0(\infty)$  in a monotonically increasing manner. For if

$x \in L^1(\mathbb{R}_+)$ , then  $\int_0^\infty x(t) dt < \infty$  and so taking the limit of the left hand side of (2.6.3) yields

$$\lim_{t \rightarrow \infty} K_0 x(t) e^{ax(t)/T_0} e^{ac/T_0} \int_0^t x(s) ds = 0,$$

while the right hand side yields the contradiction

$$0 < e^{\int_0^\infty [r_0 - r_1 c_0(t)] dt} \leq \infty$$

## 2.8. Relationship Between Solutions of (C) and (R)

The relationship between parameters in (2.3.1) and (2.3.2) were discussed in Hallam, Clark and Jordan (1983). The parameter  $k_1$  can be interpreted as  $1/M_E$ , where  $M_E$  is the volume of the environment. For most ecosystems,  $M_E$  has large numerical value; hence  $k_1$ , in general, should be a small parameter. This suggests consideration of and comparison between the reduced model (R), where  $k_1 = 0$  and the complete model (C).

To differentiate between the solutions of (C) and (R), the components of (C) will be written  $(x^C, c_0^C, c_E^C)$  and those of (R) as  $(x^R, c_0^R, c_E^R)$ . Let  $\varepsilon = k_1 X_M$ . Then, since for a given initial population level  $x_0$ ,  $|k_1 x_0^C - k_1 a_1 c_E^C x^C| < \varepsilon$ , it follows from (2.3.2) that

$$-\varepsilon + g - k_2 c_E^C \leq \frac{dc_E^C}{dt} \leq \varepsilon + g - k_2 c_E^C. \quad (2.8.1)$$

Since from (2.7.1)

$$c_E^R = e^{-k_2 t} [c_{E0} + \int_0^t g(s) e^{k_2 s} ds],$$

an integration of (2.8.1) leads to

$$|c_E^C(t) - c_E^R| \leq \varepsilon/k_2, \quad t \geq 0.$$

Hence, if  $\varepsilon$  is small, the difference between the environmental toxicant concentration in the complete model and in the reduced model is

small. The propagation of error is of the same order in the  $c_0$  equation. Similar arguments lead to

$$|c_0^C - c_0^R| \leq \frac{a_1 \epsilon}{k_2(\ell_1 + \ell_2)} .$$

Thus, the reduced model (R) may be taken as a reasonable approximation to the complete model since the difference between toxicant concentration in these models is of order  $X_M/M_E$ , which in general is a small parameter. It is difficult to find an estimate for  $|x^C - x^R|$ , but our numerical simulation in the next section shows that not only are the toxicant components of the two models close, but the population components  $x^C$  and  $x^R$  are also close.

We now present a theoretical conjecture about component relationships between models (C) and (R). We want to show that for theoretical biological reasons the solutions of the reduced model are also convenient predictors for environmentally sound decisions about solutions of the complete model because of the sign of the expression  $\phi = k_1 \ell_1 c_0^C x^C - k_1 \ell_1 c_0^C x^C$ . The term  $k_1 a_1 c_E^C x^C$  represents total toxicant uptake from the environment by the consumer, while  $k_1 \ell_1 c_0^C x^C$  represents bioavailable toxicant that is egested by the population into the environment. The relative sizes of these terms determine the sign of  $\phi$ .

If there is little or no bioavailable toxicant in the food sources, then we expect  $a_1 c_E^C$  to dominate  $\ell_1 c_0^C$  because this latter expression is only one of several losses of toxicant from the population. On the other hand, if  $\ell_1 c_0^C \geq a_1 c_E^C$ , then either the environment is not an

important source of toxicant or the toxicant is not stressful as it is being flushed out of the organism's system to the environmental pool in a bioavailable form and at a very high rate. However, egestion is frequently not the dominant organismal loss (Korte, 1968), so it is unlikely that  $\ell_1 c_0^C$  exceeds  $a_1 c_E^C$ . For this reason and from available data (e.g., see Section 2.9), we expect  $\ell_1 c_0^C < a_1 c_E^C$ . In this situation, it follows from (C) and (R) that

$$c_E^C(t) \leq c_E^R(t) ; c_0^C(t) \leq c_0^R(t) ; x^C(t) \geq x^R(t) .$$

These inequalities allow utilization of the reduced model to predict upper bounds on environmental concentrations. Hence, if environmentally safe predictions of toxicant concentrations are desired, the reduced model is sufficient for the task. The reduced model can also be employed as a predictor of threshold levels of persistence of the population, since if the population is persistent according to the reduced model, it is also persistent to at least the same level according to the complete model.

## 2.9. Numerical Comparison of (C) and (R)

We will now compare the behavior of models (C) and (R) by generating solutions using the IMSL differential equation solver DVERK, a fifth and sixth order Runge-Kutta-Verner method of numerical integration. The population used is Daphnia magna, and as the toxicant, the diene-organochlorine, dieldrin. This organism-toxicant interaction has been studied and several of the parameter values needed have been estimated or are computable.

### Parameter Estimates

We now discuss the process and rationale for obtaining the necessary parameter values. In his expanding culture experiments on D. magna, Smith (1963) obtained the values  $r_0 = 0.44/\text{day}$  and  $c = 0.127/\text{day}$ . An estimate of  $15 \times 10^{-3} \text{ g}/100\text{cc}$  ( $0.15\text{g}/\text{li.}$ ) for saturation was likewise given. For large volume this seems high so we shall assume a total volume of  $10^{12} \text{ li.}$  and the value of  $B$ , the carrying capacity, is taken to be  $15 \times 10^4 \text{ g}$ .

We now consider the possible ranges of values of  $c_0(\infty)$  and  $c_E(\infty)$ . Knowledge of the relative sizes of  $c_0(\infty)$  and  $c_E(\infty)$  is helpful in making some estimates of parameters whose experimental values are non-existent.

Always,  $0 \leq c_0(\infty) \leq 1$ , and  $0 \leq c_E(\infty) \leq 1$ . But Table 2.9.1 below shows that for most toxicants found in food and in the environment,  $c_0(\infty)$  and  $c_E(\infty)$  are both closer to zero than to one. Table 2.9.1 is not an exhaustive listing. Moreover, experimental values are included in Table 2.9.1 only if the author(s) indicated that equilibrium or near equilibrium conditions have been reached.

Reinert (1972) performed several experiments with D. magna exposed to dieldrin in water (see Table 2.9.1). He indicated toxicant concentration equilibrated in 3 to 4 days and that terminal toxicant concentration in two experiments were given by

- (i)  $c_E(\infty) = 0.21 \times 10^{-8} (\text{g}/\text{li})$ ;  $c_0(\infty) = 0.35 \times 10^{-4} (\text{g}/\text{g})$
- (ii)  $c_E(\infty) = 0.45 \times 10^{-8} (\text{g}/\text{li})$ ;  $c_0(\infty) = 0.62 \times 10^{-4} (\text{g}/\text{g})$ ,

where (g/g) means (gram-toxicant per gram-biomass).

TABLE 2.9.1.

SOME EXPERIMENTAL VALUES FOR  $c_E(\infty)$  AND  $c_0(\infty)$  FOR VARIOUS ORGANISMS AND TOXICANTS

| TOXICANTS   | ORGANISMS                         | $c_E(\infty)$         | $c_0(\infty)$         | SATURATION TIME | SOURCE              |
|-------------|-----------------------------------|-----------------------|-----------------------|-----------------|---------------------|
| 1. Dieldrin | a) Algae<br>(Scenedemus obliquus) | $0.10 \times 10^{-8}$ | $1.3 \times 10^{-6}$  | 24-36 hours     | Reinert (1972)      |
|             |                                   | $0.50 \times 10^{-8}$ | $7.1 \times 10^{-6}$  |                 |                     |
|             |                                   | $2.0 \times 10^{-8}$  | $25.2 \times 10^{-6}$ |                 |                     |
|             | b) Daphnia magna                  | $0.21 \times 10^{-8}$ | $35 \times 10^{-6}$   | 3 - 4 days      |                     |
|             |                                   | $0.45 \times 10^{-8}$ | $62 \times 10^{-6}$   |                 |                     |
|             |                                   | $1.28 \times 10^{-8}$ | $184 \times 10^{-6}$  |                 |                     |
|             | c) Guppies                        | $0.08 \times 10^{-8}$ | $18 \times 10^{-6}$   | 15 days         |                     |
|             |                                   | $0.23 \times 10^{-8}$ | $21 \times 10^{-6}$   |                 |                     |
|             |                                   | $0.42 \times 10^{-8}$ | $54 \times 10^{-6}$   |                 |                     |
| 2. DEHP     | a) Daphnids                       | $5.4 \times 10^{-6}$  | $2.8 \times 10^{-6}$  | 12-24 hours     | Macek et al. (1979) |
|             | b) Bluegill sunfish               | $5.7 \times 10^{-6}$  | $0.64 \times 10^{-6}$ | 3 days          |                     |
| 3. TCB      |                                   |                       | $0.73 \times 10^{-6}$ |                 |                     |
|             | a) Daphnids                       | $3.1 \times 10^{-6}$  | $0.44 \times 10^{-6}$ | 4-8 hours       |                     |
|             | b) Bluegill sunfish               | $2.9 \times 10^{-6}$  | $0.53 \times 10^{-6}$ | 3 days          |                     |
|             |                                   |                       | $0.57 \times 10^{-6}$ |                 |                     |

TABLE 2.9.1 (continued)

| TOXICANTS    | ORGANISMS                           | $C_E(\infty)$         | $C_0(\infty)$               | SATURATION TIME | SOURCE        |
|--------------|-------------------------------------|-----------------------|-----------------------------|-----------------|---------------|
| 4. Leptophos | a) Daphnids                         | $0.15 \times 10^{-6}$ | $.099 \times 10^{-6}$       | 24 hours        |               |
|              | b) Bluegill Sunfish                 | $0.24 \times 10^{-6}$ | $0.179 \times 10^{-6}$      | 10 days         |               |
| 5. Endrin    |                                     |                       | $0.170 \times 10^{-6}^{**}$ |                 |               |
|              | a) Clams                            | $0.24 \times 10^{-6}$ | $0.63 \times 10^{-6}$       | 21 days         |               |
|              | b) Fathead Minnows<br>(P. promelas) | $0.25 \times 10^{-6}$ | $1.7 \times 10^{-6}$        | 14-28 days      |               |
|              |                                     |                       | $1.98 \times 10^{-6}$       |                 |               |
| 6. Cadmium   | a) Brine Shrimp<br>(A. salina)      | $320 \times 10^{-6}$  | $27 \times 10^{-6}$         | 2 days          |               |
|              | b) Grass Shrimp<br>(P. pugio)       | $227 \times 10^{-6}$  | $40 \times 10^{-6}$         | 7 days          |               |
| 7. DDT       |                                     |                       | $45.6 \times 10^{-6}$       | 56 days         |               |
|              | a) Clams                            | $1.18 \times 10^{-6}$ |                             |                 |               |
|              | b) Fathead Minnows<br>(P. promelas) | $1.5 \times 10^{-6}$  | $190 \times 10^{-6}$        | 56 days         |               |
|              |                                     |                       | $250 \times 10^{-6}^*$      |                 |               |
| 8. Kepone    | a) Oysters<br>(C. virginica)        | $0.03 \times 10^{-6}$ | $0.210 \times 10^{-6}$      | 30 days         | Bahner et al. |
|              | b) Mysids<br>(M. bahia)             | $0.03 \times 10^{-6}$ | $0.120 \times 10^{-6}$      | 21 days         |               |
|              |                                     | $0.41 \times 10^{-6}$ | $4.8 \times 10^{-6}$        |                 |               |

TABLE 2.9.1 (continued)

| TOXICANTS       | ORGANISMS                                         | $c_E(\infty)$         | $c_0(\infty)$         | SATURATION TIME | SOURCE       |
|-----------------|---------------------------------------------------|-----------------------|-----------------------|-----------------|--------------|
|                 | c) Grass shrimp<br>( <i>P. pugio</i> )            | $0.02 \times 10^{-6}$ | $0.09 \times 10^{-6}$ | 28 days         |              |
|                 |                                                   | $0.40 \times 10^{-6}$ | $4.57 \times 10^{-6}$ |                 |              |
|                 | d) Sleep Head Minnows<br>( <i>C. variagatus</i> ) | $0.5 \times 10^{-6}$  | $0.37 \times 10^{-6}$ |                 |              |
|                 | e) Spot<br>( <i>L. xanthurus</i> )                | $0.03 \times 10^{-6}$ | $0.09 \times 10^{-6}$ | 30 days         |              |
|                 |                                                   | $0.04 \times 10^{-6}$ | $0.94 \times 10^{-6}$ |                 |              |
| 9. Aroclor 1248 | Fathead Minnows                                   | $3 \times 10^{-6}$    | $190 \times 10^{-6}$  | 100 days        | Defoe et al. |
| Aroclor 1260    | Fathead Minnows                                   | $2.1 \times 10^{-6}$  | $350 \times 10^{-6}$  | 100 days        |              |

\*Environment + dietary

\*\*Estimated

Reinert (1972) fed D. magna algae containing dieldrin (4.2-7.5 ppm) and concluded that dieldrin from water and from food are not directly "additive". Hence, the equation (2.3.1) with the balanced trade off between toxicant pathways seems to be an appropriate formulation.

While we can find no explicit value of  $\beta$  in the literature for D. magna, the value of 10% of organism body weight has been used for ration size in the case of fish (Aoyama, 1978; Skaar et al., 1981). We will also take the average ration size to be 10%. Hence,  $\beta$ , which is the average rate of per unit mass food intake, is  $\beta = 0.10/\text{day}$ . Using the toxicant levels in the algae fed to D. magna as motivation, we assign  $\theta$  the value  $4.2 \times 10^{-6}(\text{g/g})$ . Hence  $\theta\beta = 4.2 \times 10^{-7}(\text{g/g-day})$ . For this simulation, we will arbitrarily select a value for  $d_1$ , since there is no available information on this parameter. We assume  $d_1$  is prescribed and proceed to find estimates for  $a_1$ ,  $\ell_1$ , and  $\ell_2$ . To this end, we consider (2.3.1) at equilibrium and use the values for  $c_0(\infty)$  and  $c_E(\infty)$  listed above in (i) and (ii). The result is the following pair of non-linear equations in  $a_1$  and  $\ell_1 + \ell_2$ :

$$a_1(0.21 \times 10^{-8}) + \frac{d_1}{a_1} (0.1)(4.2 \times 10^{-6}) - (\ell_1 + \ell_2)(35 \times 10^{-6}) = 0$$

$$a_1(0.45 \times 10^{-8}) + \frac{d_1}{a_1} (0.1)(4.2 \times 10^{-6}) - (\ell_1 + \ell_2)(62 \times 10^{-6}) = 0.$$

Solving for  $a_1$  and  $\ell_1 + \ell_2$  yields

$$\lambda_1 + \lambda_2 = 0.00181 \sqrt{d_1}$$

$$a_1 = 20.381 \sqrt{d_1} .$$

The parameter value  $k_2 = 0.005/\text{day}$  is obtained from Schnoor (1981). The parameter  $k_1$  was interpreted earlier as  $1/M_E$  and as such is small. For D. magna and for our prescribed total volume of  $10^{12} \text{ li}$ ,  $k_1$  may take the value of  $k_1 = 10^{-12} = \frac{1}{M_E}$ .

The remaining parameters  $a$  and  $r_1$ , introduced in connection with modelling toxic stress, are not available in the literature. The parameter  $a$  must satisfy the inequality  $a < r_0 + c$ . Hence, if  $r_0 = 0.44$  and  $c = 0.127$ , then  $a$  cannot exceed 0.567. For our initial run, we assign the value  $a = 0.5/\text{day}$ . To determine  $r_1$ , we observe that Theorem 2.7.1 states that the inequalities  $r_0 > r_1 c_0(\infty)$  and  $r_0 \leq r_1 c_0(\infty)$  determine persistence and extinction of the population respectively. From Table 2.9.1, we see that for most toxicants,  $c_0(\infty)$  is a very small number. In Reinert's experiments, the population survived at terminal concentration  $c_0(\infty) = 35 \times 10^{-6}$ . For purposes of our simulation, we assume that  $c_0(\infty) = 99 \times 10^{-5}$  is the minimum body burden that would drive the population to extinction. Hence, if  $r_0 = 0.44$ , and  $r_0 < r_1 c_0(\infty)$ , we obtain  $r_1 > 444.44(\text{g/g-day})$ . We select  $r_1 = 450.0 (\text{g/g-day})$ .

It remains to discuss the exogenous toxicant input  $g(t)$ , which for purposes of simulation is assumed constant. From the relationship  $g_\infty = k_2 c_E(\infty)$ , we find

$$g_\infty = 0.105 \times 10^{-10} (\text{g/li}) .$$

A summary of parameter values and initial conditions used is contained in Table 2.9.2.

### Computer Simulation Results

Figure 2.9.1 illustrates the behavior of the components of models (C) and (R). The population increases rapidly while the body burden is low; it overshoots its equilibrium value and then declines to the equilibrium when toxicant stress becomes important. Maximum population size for both models is attained on approximately day 64.

Figure 2.9.2 illustrates the behavior of the components of models (C) and (R) when the population is driven to extinction, i.e. when  $r_0 < r_1 c_0(\infty)$ . Note again the rapid increase in population during the first 20 days. Maximum population size for both models was attained on about the 29th day.

Figures 2.9.3 and 2.9.4 show the behavior of the population and the body burden, respectively for different values of  $d_1$ . Note the slow convergence of the population trajectory as well as the body burden when  $d_1 = 1$ . Experimental data for most toxicants indicate that it does not take very long for the body burden to reach equilibrium or near equilibrium level (see Table 2.9.1).

Hence, at least for dieldrin,  $d_1$  is a relatively large number. We note in Figure 2.9.4 that a large value of  $d_1$  hastens the convergence of  $c_0(t)$  to  $c_0(\infty)$ . Moreover, in Figure 2.9.3, the population levels are relatively close when  $d_1$  varies from 100 to 415. Hence, for the rest of our computer simulation, we will use the value  $d_1 = 100$ .

TABLE 2.9.2

SUMMARY OF PARAMETER VALUES AND INITIAL CONDITIONS USED IN COMPUTER SIMULATION.  
NON-BLANK ENTRIES IN SUCCEEDING COLUMNS REPRESENT THE CHANGES IN PARAMETER  
VALUES AND/OR INITIAL CONDITIONS IN COLUMN II.

## PARAMETER VALUES

| PARAMETERS | I                    | II                   | III    | IV    | V       | VI | VII | VIII       | IX         |
|------------|----------------------|----------------------|--------|-------|---------|----|-----|------------|------------|
| $r_0$      | .44                  | .44                  |        |       |         |    |     |            |            |
| B          | $15 \times 10^{-4}$  | $15 \times 10^{-4}$  |        |       |         |    |     |            |            |
| c          | 0.127                | 0.127                |        |       |         |    |     |            |            |
| a          | 0.5                  | 0.5                  |        |       |         |    |     |            |            |
| $r_1$      | 450                  | 450                  |        |       |         |    |     |            |            |
| $a_1$      | 20.381               | 203.81               | 288.23 | 353   | 415     |    |     |            |            |
| $d_1$      | 1.0                  | 100                  | 200    | 300   | 415     |    |     |            |            |
| $\phi$     | $4.2 \times 10^{-6}$ | $4.2 \times 10^{-6}$ |        |       |         |    |     |            |            |
| $\beta$    | 0.1                  | 0.1                  |        |       |         |    |     |            |            |
| $\ell_1$   | $.91 \times 10^{-3}$ | .0091                | .0128  | .0158 | .018453 |    |     |            |            |
| $\ell_2$   | $.91 \times 10^{-3}$ | .0091                | .0128  | .0158 | .018453 |    |     |            |            |
| $k_1$      | $10^{-12}$           | $10^{-12}$           |        |       |         |    |     | $10^{-10}$ | $10^{-14}$ |
| $k_2$      | 0.005                | .005                 |        |       |         |    |     |            |            |

TABLE 2.9.2. (continued)

## INITIAL CONDITIONS

| PARAMETERS | I                      | II                     | III | IV | V | VI                   | VII                   | VIII | IX |
|------------|------------------------|------------------------|-----|----|---|----------------------|-----------------------|------|----|
| $x_0$      | $10^4$                 | $10^4$                 |     |    |   |                      |                       |      |    |
| $x$        | $.2 \times 10^{-8}$    | $.2 \times 10^{-8}$    |     |    |   | $.5 \times 10^{-7}$  |                       |      |    |
| $c_{E0}$   | $.105 \times 10^{-10}$ | $.105 \times 10^{-10}$ |     |    |   | $.45 \times 10^{-9}$ | $.64 \times 10^{-10}$ |      |    |
| $g_\infty$ |                        |                        |     |    |   |                      |                       |      |    |

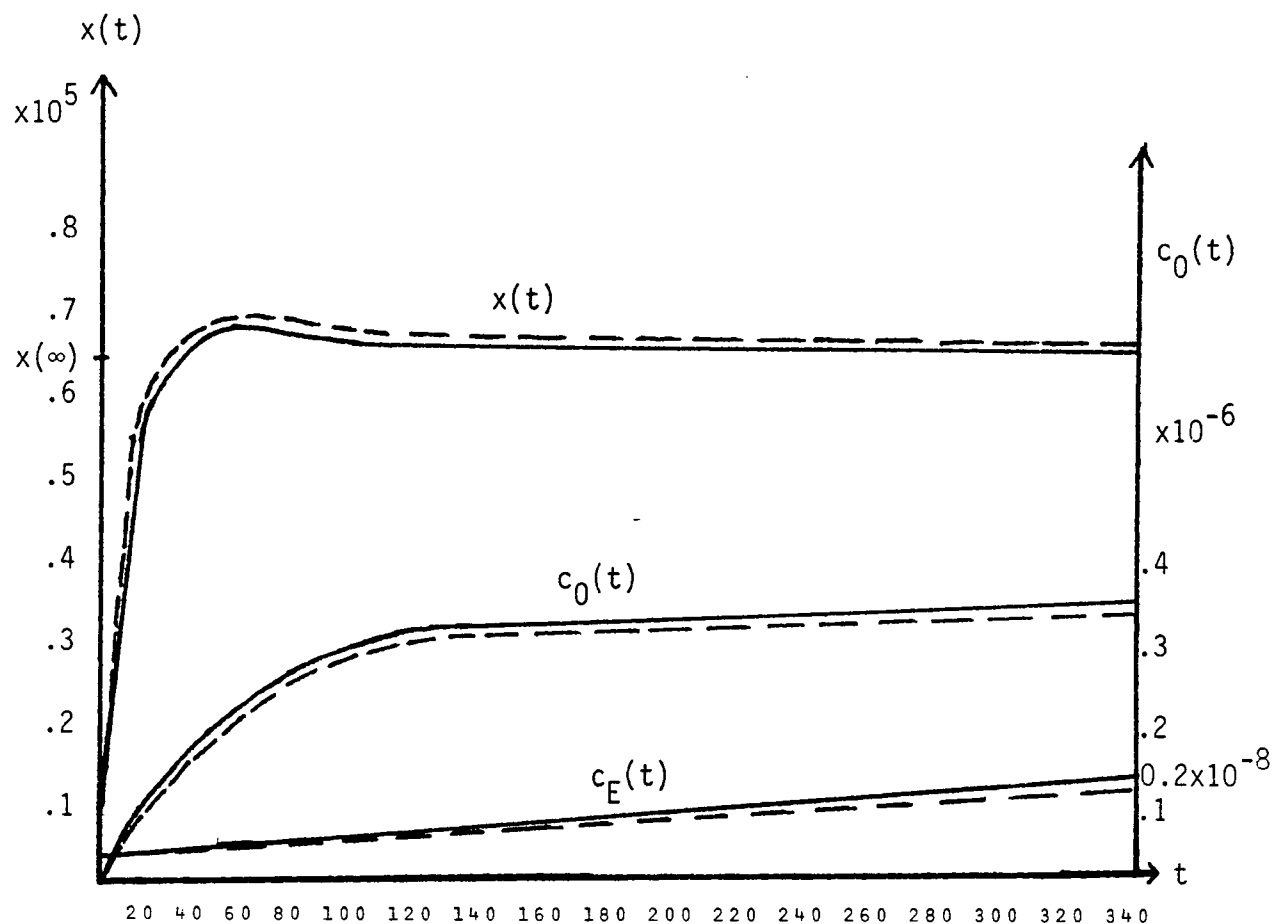


FIGURE 2.9.1. BEHAVIOR OF THE COMPONENTS OF MODELS (C) AND (R). PARAMETER VALUES AND INITIAL CONDITIONS USED ARE THOSE IN TABLE 2.11. DASHED CURVES REPRESENT THE COMPONENTS OF MODEL (C).

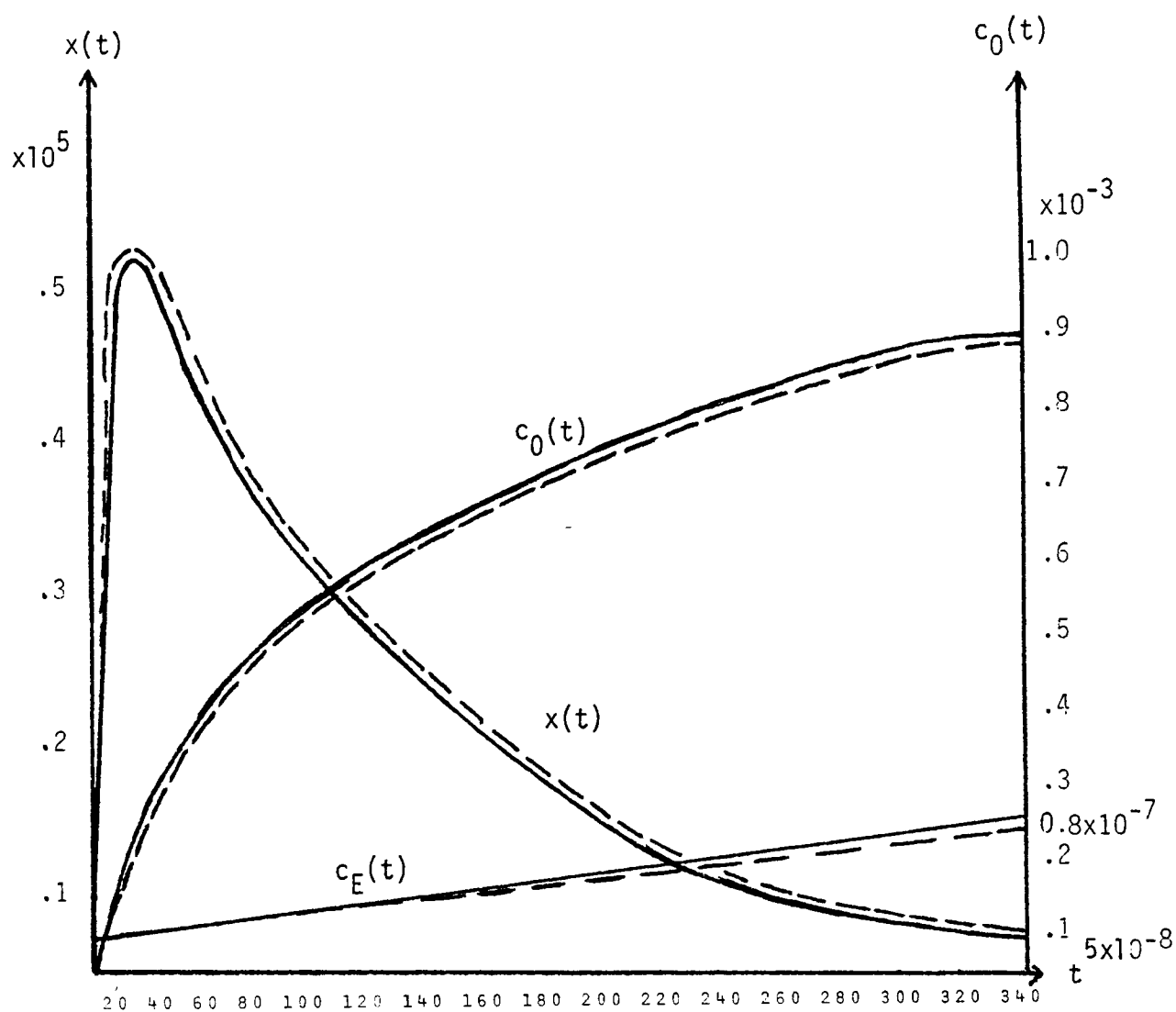


FIGURE 2.9.2. EXTINCTION BEHAVIOR OF MODELS (C) AND (R) .  
 DASHED CURVES REPRESENT THE COMPONENTS OF MODEL (C) .  
 PARAMETER LINES AND INITIAL CONDITIONS USED ARE THOSE  
 IN TABLE 2, III.

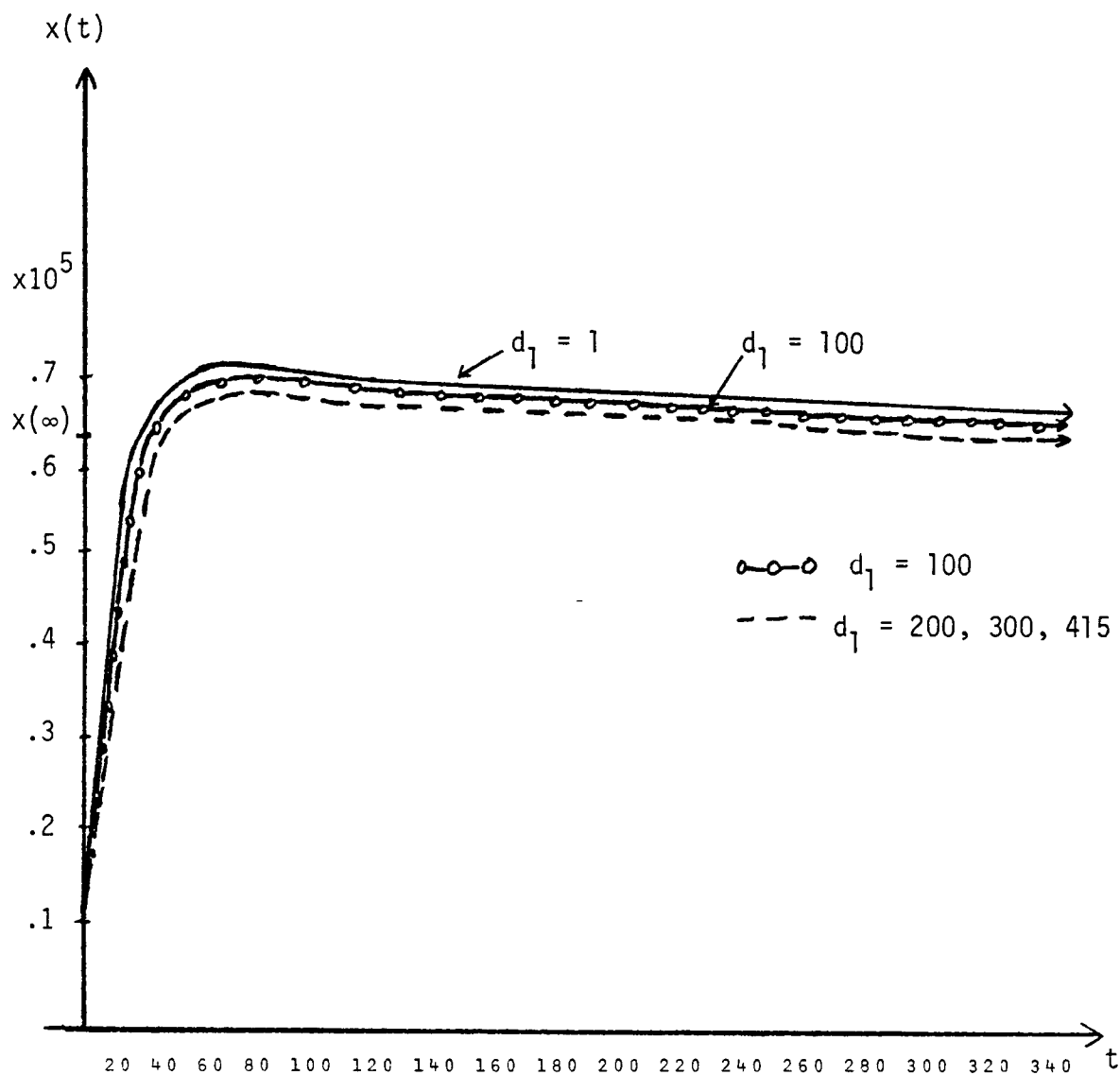


FIGURE 2.9.3. BEHAVIOR OF THE POPULATION FOR DIFFERENT VALUES OF  $d_1$ . HERE THE FOLLOWING VALUES OF  $d_1$  ARE USED:  $d_1 = 1, 100, 200, 300, 415$ .

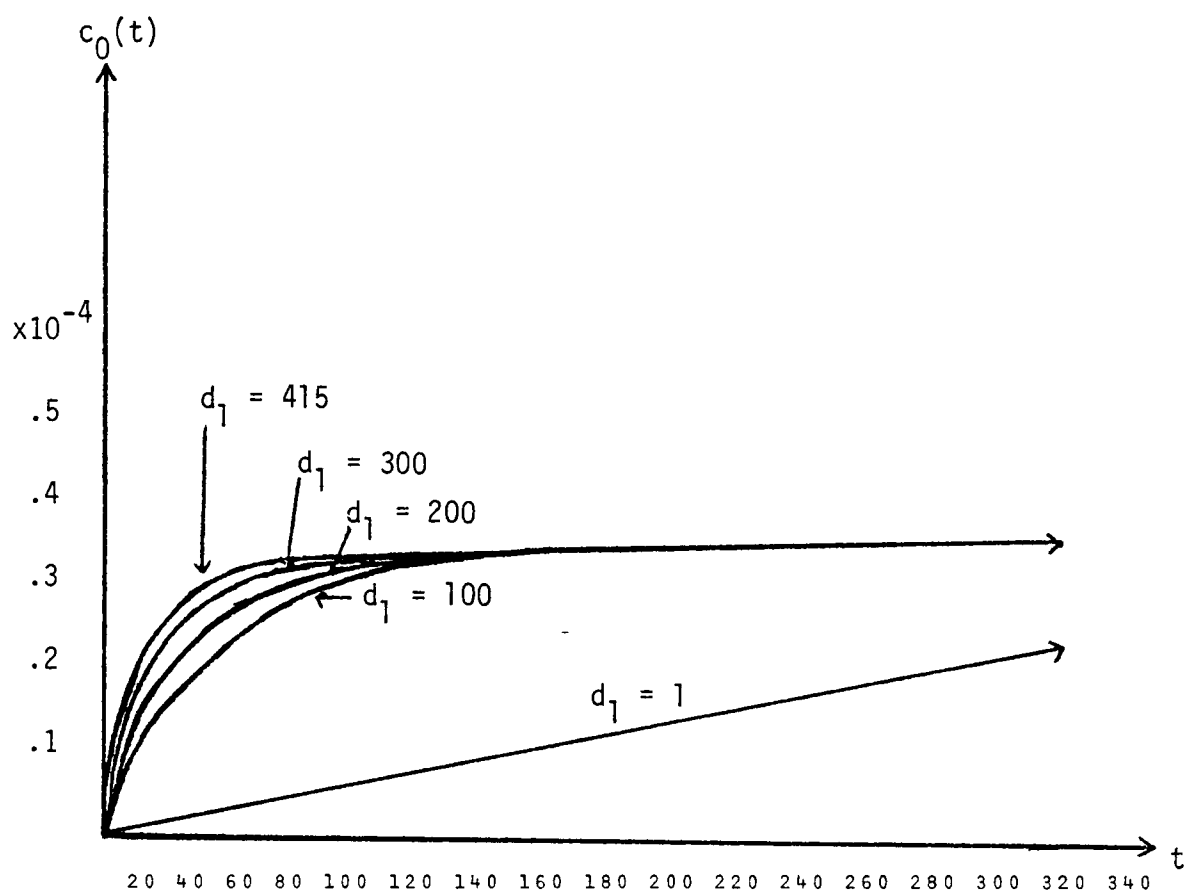


FIGURE 2.9.4. BEHAVIOR OF THE BODY BURDEN FOR DIFFERENT VALUES OF  $d_1$ . HERE  $d_1$  TAKES THE VALUES 1, 100, 200, 300, AND 415.

It is easy to see, using the reduced model and the parameter values in Table 2.9.2, VII, that a constant exogenous input of  $0.64 \times 10^{-10}$  (g/li-day), will yield a steady state environmental toxicant concentration of  $1.28 \times 10^{-3}$  (g/li) . Using Reinert's data for dieldrin, the corresponding body burden is  $184 \times 10^{-6}$  (g/g). Simulation results however, showed a body burden of about  $1.55 \times 10^{-4}$  (g/g) , which is about 16% less than the expected value. But given experimental error possibilities and the fact that we made several hypotheses and several parameter guesses and utilized data from various sources, our simulation value for  $c_0(\infty)$  may still be comparable with Reinert's experimental value.

Simulation results, using parameter values in Table 2.9.2, VIII and IX, are essentially the same as in Figure 2.9.1, except that the corresponding components of models (C) and (R) are not as close when  $k_1 = 10^{-10}$  as when  $k_1 = 10^{-12}$  or when  $k_1 = 10^{-14}$  .

## 2.10. Conclusions and Comments

A model of a toxicant-population interaction has been proposed and its behavior analyzed. The toxicant kinetics are of first order as is the dose-response function that couples the toxicant dynamics to those of the population. The population is assumed governed by a modification of Smith's model. A main feature of our development is that toxicant input to the population is viable from both the environmental and food pathways.

A reduced model, wherein it is assumed the population does little or no regulation of toxicant in the environment is introduced and the

relationship between the complete model and the associated model is analyzed. It is shown analytically that toxicant concentrations of the complete model and the reduced model remain close to each other. It is also argued that under natural conditions, the reduced model is an adequate, environmental sound predictor of threshold values for toxicant concentration as well as the level of persistence of the population. A numerical study of the interaction of D. magna and dieldrin support this argument.

The main mathematical results focus on systems level effects of toxicant by indicating persistence and extinction criteria. For oscillating (nonconvergent) toxicant inputs, the persistence results depend upon the maximal level of toxicant input, and for extinction, the dependence is on the minimum level of toxicant input. For convergent toxicant input, the analytical work is precise in that the conditions for persistence and extinction are complementary.

Comments are now made regarding our choice of a linear dose-response function in section 2.4. We will discuss the implicit non-threshold assumption associated with the form of  $H(c_0)$  and then compare the manner by which a linear dose-response function and a sigmoid dose-response function estimate risks due to exposure outside the range of values consistent with available data in the literature. Specifically, we want to compare the values of both dose-response function types at low exposure levels. The comparison is brought about by the fact that it is generally necessary to base judgments on higher dose data from laboratory studies when making judgments as to the risk from low level exposure to harmful chemicals. In effect,

we extrapolate to low dose range the relatively good information about risk from high dose exposure (Land, C.E., 1980).

We note that in taking  $H(c_0) = r_1 c_0$ , we are implicitly assuming that exposure to toxic chemicals at any level carries some risks, however small that risk might be, i.e., that there is no threshold value for  $c_0$ . We are assuming that we do not need to reach a minimum level of body burden, say  $c_{0min}$ , before injury to the population is manifested.

On the other hand, if we assume that a certain minimum level of body burden (a threshold value) is necessary before a population is affected by a toxicant, then the form of  $H$  as defined in (2.4.2) is no longer valid. A linear dose-response function with threshold may take the form

$$H_1(c_0) = \begin{cases} 0 & \text{if } c_0 \leq c_{0min} \\ r_1(c_0 - c_{0min}) & \text{if } c_0 > c_{0min} \end{cases}$$

Note that in this case, low level exposure to a toxicant does not affect the population. As long as the body burden remains below the threshold value  $c_{0min}$ , the population is relatively unharmed by the toxicant.

The idea of threshold values is important in many applications. Filov, et al. (1979), have indicated that "the toxicological significance of threshold concentrations is determined by the fact that they provide a starting point for establishing permissible concentrations of toxic gases, vapors, and dusts in the air of workplaces." But

there are problems associated with threshold values; for example, for a given toxicant, the threshold value  $c_{0min}$  is not uniquely determined. The problem is that threshold values depend not only on species and individual differences in sensitivity, but first and foremost, on the method used to establish them. Consequently, there is no agreement as to what a threshold dose is and what a threshold effect is.

Our non-acceptance of the idea of threshold values is of course influenced by the fact that these values are difficult to establish. Moreover, we subscribe to a principle, adopted in 1954 by the National Council on Radiation, which states that any level of radiation exposure may carry some risk, i.e. a threshold cannot be assumed for all types of radiation injury (Brown, 1976). We believe that the above-stated principle is valid for all types of exposure to harmful chemicals. It may be argued, however, that we are subscribing to a principle in radiation which may have no bearing at all in pollution studies. That this is not the case is forcefully argued by G.C. Butler (1979). He has indicated that a radiation approach to pollution problems is practical and that it indicates the kind of information, thinking, and procedure that can be useful to pollution problems.

We now consider the problem of estimating risks at low dose range. A sigmoid dose-response function without threshold may take the form

$$H_2(c_0) = -\frac{A}{1+c} + \frac{A}{1+c e^{-\lambda A c_0}}$$

where  $A$ ,  $c$ , and  $\lambda$  are positive constants. Let us assume that parameter values in  $H$  and  $H_2$  can be chosen so that, in the

range of values consistent with data in the literature, the values of  $H$  and  $H_2$  are relatively close. A typical configuration is illustrated in Figure 2.10.1.

It is clear from Figure 2.10.1 that the linear dose-response function overestimates the risks at low levels of exposure. Therefore, critics of linear dose-response functions without threshold, claim that using linearity overestimates the risk at low doses by such an order of magnitude that it makes poor sense to focus time, money and resources to reduce an already minimum risk (Brown, 1976).

We maintain however, that where survival is the issue, it pays to incorporate safety factors in judgment and to aim at conservatism, which was our guiding principle when we adopted the reduced model. We intend to use this principle and linear dose-response function without threshold throughout this study.

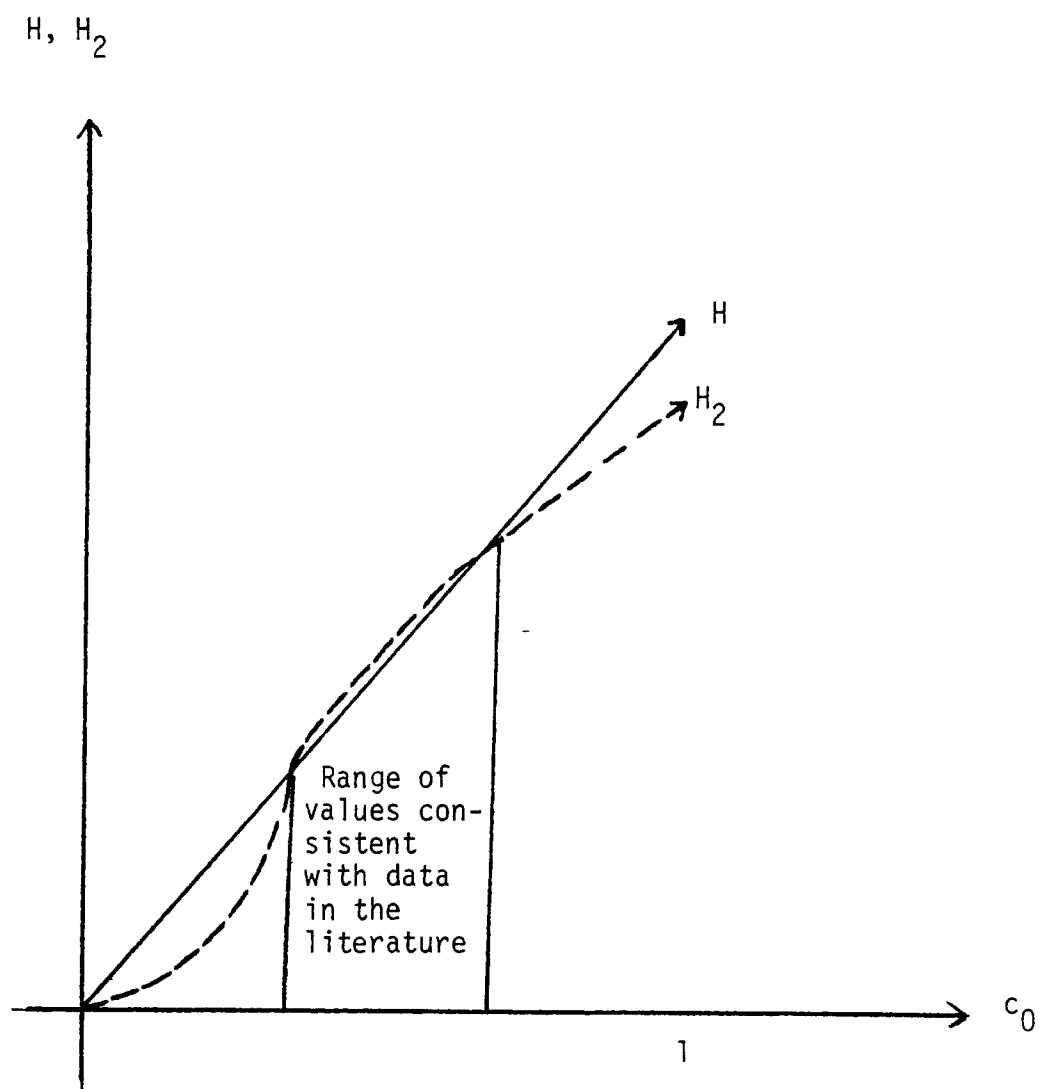


FIGURE 2.10.1. COMPARISON OF THE LINEAR DOSE-RESPONSE AND SIGMOID DOSE-RESPONSE FUNCTIONS WITHOUT THRESHOLD.

## CHAPTER 3

## RESOURCE-CONSUMER-TOXICANT MODELS

## 3.1. Introduction

The abundance and quality of resources govern many aspects of population growth; hence any model of a population must account for the bioavailability of resources in an implicit or an explicit manner. Some recent studies (Hallam, Clark, and Lassiter, 1982; Hallam, Clark, and Jordan, 1983) on the dynamics of population-toxicant interactions have employed population models where the resource is formulated implicitly in the model. This approach necessitates development of toxicant exposure solely via the environmental pathway. As an initial step to include food chain uptake of toxicant in a population model, we have employed in Chapter 2 a modification of a model by Smith (1963). But this technique requires that the resource be renewable and at equilibrium. In this chapter, we develop and analyze several models of population-toxicant interactions that include an explicit representation for a dynamic resource. The asymptotic behaviors of these models are studied, persistence and extinction criteria are developed, and common features of the behaviors are correlated. Each model contains components representing the population-resource dynamics, the body burden dynamics, and the environmental concentration of toxicant. The state variables are  $x = x(t)$ ,  $c_0 = c_0(t)$ ,  $c_E = c_E(t)$  and  $a = a(t)$ , where  $x$ ,  $c_0$ , and  $c_E$  are as defined in Chapter 2 and  $a = a(t)$  is the amount of bioavailable resource.

It was argued in Chapter 2 that the reduced model is a good approximation to the complete model in that the difference between concentrations of toxicant of these models is of order  $X_M/M_E$ , where  $X_M$  is an upper bound on the population biomass. In general,  $X_M/M_E$  is a small parameter. Hence, using the same argument in Chapter 2, we employ the reduced model for the concentration dynamics in the resource-consumer-toxicant system.

In the next three sections, we formulate and analyze the dynamics of three resource-consumer-toxicant systems. Each of the resulting models is analyzed from a persistence-extinction and equilibrium perspective. The first resource-consumer-toxicant model proposed includes a resource-consumer subsystem of Leslie type; the second model uses a resource-consumer subsystem of Gallopin type; and the third deals with a model of a continuous culture type. In the first two models, we require that there be a constant concentration  $\theta$  of toxicant in the resource; in all three models, it is required that there be a feedback between toxicant intake pathways.

### 3.2. Leslie-Type Resource-consumer Model

A resource-consumer system may contain some of the complex processes in a predator-prey system (Ishihara, et al., 1972). The consumer population can grow only at the expense of the resource and its growth rate is a function of available resource. Assumptions that incorporate these features and are employed to formulate the model include the following:

(A1): The dynamics of the consumer population is logistic in character with the carrying capacity being a function of the amount of available resource.

(A2): Resource consumption is proportional to the biomass of the consumer population and to the amount of resource available for ingestion.

(A3): Feeding by the consumer population is solely responsible for a decrease in the food supply.

Let  $f = f(t)$  denote the rate of input of resource into the system. A resource-consumer model that satisfies (A1), (A2), and (A3) is:

$$\frac{dx}{dt} = x[r - \alpha x/a] \quad (3.2.1)$$

$$\frac{da}{dt} = f - \gamma ax . \quad (3.2.2)$$

The coupling of the toxicant and the consumer population is accomplished by assuming a dose-response function of the form given by equations (2.4.1) and (2.4.2), i.e.,  $r = r_0 - r_1 c_0$ . Hence, (3.2.1) becomes

$$\frac{dx}{dt} = x[r_0 - r_1 c_0 - \alpha x/a] . \quad (3.2.3)$$

The Leslie type resource-consumer-toxicant model (L) analyzed here consists of equations (3.2.3), (3.2.2), (2.3.1), and (2.7.1):

$$\frac{dx}{dt} = x[r_0 - r_1 c_0 - \alpha x/a]$$

$$\frac{da}{dt} = f - \gamma ax$$

$$\frac{dc_0}{dt} = a_1 c_E + \frac{d_1 \theta \beta}{a_1} - (\ell_1 + \ell_2) c_0$$

$$\frac{dc_E}{dt} = g - k_2 c_E .$$

The initial conditions are

$$x(0) = x_0 > 0 , a(0) = a_0 > 0 , c_0(0) = 0 , c_E(0) = c_{E0} > 0 .$$

### Persistence-Extinction Analysis of the Leslie Type Model.

Comparison of density dependent effects and biotic potential effects as measured in (3.2.3) by  $x/a$  and  $r_0 - r_1 c_0$ , respectively, shows that if resource is abundant and biotic potential is positive, then growth is feasible. If biotic potential is negative, then even if resource is abundant, the consumer population goes to extinction. Whenever resource is scarce, ( $x/a$  is large), density dependent effects are important. If biotic potential is positive but small, the population can go to extinction. We make these observations precise in:

THEOREM 3.2.1. Let  $0 \leq g_m \leq g(t) \leq g_M$ ,

$$\rho_m = \frac{d_1 \theta \beta}{a_1 (\ell_1 + \ell_2)} + \frac{a_1 g_m}{k_2 (\ell_1 + \ell_2)} ,$$

$$\rho_M = \frac{d_1 \theta \beta}{a_1(\ell_1 + \ell_2)} + \frac{a_1 g_M}{k_2(\ell_1 + \ell_2)}$$

Then model (L) exhibits the following behavior:

- (i) If the consumer population has negative biotic potential, in the sense that  $r_0 < r_1 \rho_M$ , then the consumer population goes to extinction, that is,  $\lim_{t \rightarrow \infty} x(t) = 0$ .
- (ii) If  $r_0 > r_1 \rho_M$  and  $\limsup_{t \rightarrow \infty} f(t) > 0$ , then the resource-consumer community persists:  $\limsup_{t \rightarrow \infty} a(t) > 0$  and  $\limsup_{t \rightarrow \infty} x(t) > 0$ .

PROOF. It is convenient first to find integral representations for the system components of (L). Since (3.2.3) can be viewed as a non-autonomous logistic equation which is an equation of Bernoulli type, the population may be represented as

$$x(t) = \frac{x_0 e^{\int_0^t [r_0 - r_1 c_0(s)] ds}}{1 + \alpha x_0 \int_0^t e^{\int_0^s [r_0 - r_1 c_0(s_1)] ds_1} [a(s)]^{-1} ds} \quad (3.2.4)$$

Equation (3.2.2) is a linear equation in  $a$ ; thus

$$a(t) = e^{-\gamma \int_0^t x(s) ds} \left[ a_0 + \int_0^t f(s) e^{\gamma \int_0^s x(s_1) ds_1} ds \right]. \quad (3.2.5)$$

The integral representations of  $c_0$  and  $c_E$  are given by (2.6.1) and (2.6.2) with  $k_1 = 0$ , respectively. As in the proof of Theorem

2.6.2, employing the hypothesis on  $g$ , we find that

$$c_0(t) \geq \frac{d_1 \theta \beta}{a_1(\ell_1 + \ell_2)} + \frac{a_1 g_m}{k_2(\ell_1 + \ell_2)} + o(1) = \rho_m + o(1)$$

where, as before,  $o(1)$  represents a function that approaches zero as  $t$  approaches infinity. From (3.2.4) and from the above lower estimate on  $c_0$ , we obtain

$$\begin{aligned} x(t) &\leq x_0 e^{\int_0^t [r_0 - r_1 c_0(s)] ds} \\ &\leq x_0 e^{\int_0^t [r_0 - r_1(\rho_m + o(1))] ds} \end{aligned}$$

Since  $r_0 - r_1 \rho_m < 0$  and  $o(1)$  can be made arbitrarily small, we obtain  $\lim_{t \rightarrow \infty} x(t) = 0$ . This proves (i).

To prove (ii), we first show that  $\limsup_{t \rightarrow \infty} a(t) > 0$  and we do this by contradiction. Suppose  $\lim_{t \rightarrow \infty} a(t) = 0$ . From (3.2.5), we can write

$$0 = \lim_{t \rightarrow \infty} a(t) = \lim_{t \rightarrow \infty} \frac{a_0 + \int_0^t f(s) e^{\gamma \int_0^s x(s_1) ds_1} ds}{\gamma \int_0^t x(s) ds} \quad (3.2.6)$$

Since the numerator is bounded away from zero, it is necessary for the denominator in (3.2.6) to tend to infinity for large  $t$ . Hence, L'Hopital's Rule implies

$$\lim_{t \rightarrow \infty} a(t) = \lim_{t \rightarrow \infty} \frac{f(t)}{\gamma x(t)} = 0. \quad (3.2.7)$$

Since  $\limsup_{t \rightarrow \infty} f(t) > 0$ , then the only way (3.2.7) can hold is for

$\limsup_{t \rightarrow \infty} x(t) = \infty$ . Consider now  $c_0(t)$ . In (2.6.6) we obtain the upper estimate (with  $k_1 = 0$ )

$$c_0(t) \leq \rho_M + \underline{o}(1).$$

Hence

$$r_0 - r_1 c_0(t) \geq r_0 - r_1(\rho_M + \underline{o}(1))$$

for all  $t$  in  $\mathbb{R}_+$ . Since  $\underline{o}(1)$  can be made arbitrarily small and since  $r_0 > r_1 \rho_M$ , we can choose  $t_1$  sufficiently large such that

$$r_0 - r_1 c_0(t) \geq r_0 - r_1(\rho_M + \underline{o}(1)) > 0 \quad (3.2.8)$$

for all  $t \geq t_1$ . Consequently,

$$\begin{aligned} \exp\left(\int_{t_1}^t [r_0 - r_1 c_0(s)] ds\right) &\geq \\ &\geq \exp\left(\int_{t_1}^t [r_0 - r_1(\rho_M + \underline{o}(1))] ds\right) > 1. \end{aligned} \quad (3.2.8a)$$

Also, since  $\lim_{t \rightarrow \infty} a(t) = 0$ , we can choose  $t_2 > 0$  such that  $a(t) < 1$  for all  $t \geq t_2$ . Let  $t_0 = \max(t_1, t_2)$ . Then, if  $t \geq t_0$ , we can

write

$$\frac{\exp\left(\int_{t_0}^t [r_0 - r_1 c_0(s)] ds\right)}{a(t)} \geq \frac{\exp\left(\int_{t_0}^t [r_0 - r_1(\rho_M + o(1))] ds\right)}{a(t)} \\ > \frac{1}{a(t)} > 1 .$$

Consequently, the denominator in (3.2.4) must approach infinity.

An application of L'Hopital's Rule yields

$$\lim_{t \rightarrow \infty} x(t) = \lim_{t \rightarrow \infty} \frac{a(t)(r_0 - r_1 c_0(t))}{\alpha} = 0 .$$

This contradiction shows that  $\limsup_{t \rightarrow \infty} a(t) > 0$  .

To demonstrate the persistence of  $x$  , we assume for the purpose of contradiction that  $\lim_{t \rightarrow \infty} x(t) = 0$  . Then, because of (3.2.8a),

$\lim_{t \rightarrow \infty} x(t) = 0$  is possible only if the dominator of (3.2.4) becomes

infinite. Applying L'Hopital's Rule in (3.2.4) yields

$$\lim_{t \rightarrow \infty} x(t) = \lim_{t \rightarrow \infty} \frac{a(t)(r_0 - r_1 c_0(t))}{\alpha} . \quad (3.2.8b)$$

But since (3.2.8) holds and  $\limsup_{t \rightarrow \infty} a(t) > 0$  , then

$$\lim_{t \rightarrow \infty} \frac{a(t)(r_0 - r_1 c_0(t))}{\alpha} > 0 ,$$

which is a contradiction. Consequently,  $\limsup_{t \rightarrow \infty} x(t) > 0$  . This completes the proof.

REMARKS. It is clear from (3.2.8b) that when  $r_0 > r_1 \rho_M$ , extinction of the consumer population is equivalent to extinction of the resource, that is,

$$\lim_{t \rightarrow \infty} x(t) = 0 \text{ if and only if } \lim_{t \rightarrow \infty} a(t) = 0 .$$

From (3.2.7), it follows immediately that if  $\lim_{t \rightarrow \infty} a(t) = 0$  then

$$\lim_{t \rightarrow \infty} f(t) = 0 . \text{ Conversely, if } \lim_{t \rightarrow \infty} f(t) = 0 \text{ and if } \lim_{t \rightarrow \infty} a(t)$$

exists, then  $\lim_{t \rightarrow \infty} a(t) = 0$ . Hence, as should be the case, a renewable resource is required for persistence of the population. Moreover, from (3.2.8b), it is clear that population levels are ultimately governed by resource as well as by the body burden levels.

As in Chapter 2, it is in general difficult to discuss the system behavior when  $r_1 \rho_m \leq r_0 \leq r_1 \rho_M$ , unless additional hypotheses are imposed. If toxicant input approaches a limit, that is,  $\lim_{t \rightarrow \infty} g(t) = g_\infty$  exists, it follows that  $\lim_{t \rightarrow \infty} c_E(t) = c_E(\infty)$  and  $\lim_{t \rightarrow \infty} c_0(t) = c_0(\infty)$  exist. This leads to the following persistence-extinction result.

THEOREM 3.2.2. Assume  $\lim_{t \rightarrow \infty} g(t) = g_\infty \geq 0$  and  $\lim_{t \rightarrow \infty} f(t) = f_\infty > 0$ .

- (a) If ultimate biotic potential is negative,  $r_0 < r_1 c_0(\infty)$ , then the consumer population goes to extinction,

$$\lim_{t \rightarrow \infty} x(t) = 0 .$$

- (b) If ultimate biotic potential is positive,  $r_0 > r_1 c_0(\infty)$ , then the consumer population is persistent. Each solution

tends to a limit with components given by

$$(i) \quad x(\infty) = \left[ \frac{f_{\infty}(r_0 - r_1 c_0(\infty))}{\alpha \gamma} \right]^{\frac{1}{2}}$$

$$(ii) \quad a(\infty) = \left[ \frac{\alpha f_{\infty}}{\gamma(r_0 - r_1 c_0(\infty))} \right]^{\frac{1}{2}}$$

$$(iii) \quad c_0(\infty) = \frac{d_1 \theta \beta}{a_1(\ell_1 + \ell_2)} + \frac{a_1 g_{\infty}}{k_2(\ell_1 + \ell_2)}$$

$$(iv) \quad c_E(\infty) = \frac{g_{\infty}}{k_2}$$

PROOF. The extinction result of part (a) follows immediately from (3.2.4). To verify the persistence result in part (b), an argument like those employed in the proof of Theorem 3.2.1(ii) shows that if  $\limsup_{t \rightarrow \infty} f(t) > 0$ , then  $\limsup_{t \rightarrow \infty} a(t) > 0$ . The hypothesis  $r_0 > r_1 c_0(\infty)$  allows application of L'Hopital's Rule in (3.2.4) to obtain

$$\limsup_{t \rightarrow \infty} x(t) = \limsup_{t \rightarrow \infty} a(t)[r_0 - r_1 c_0(\infty)]/\alpha \quad (3.2.9)$$

from which persistence follows.

To show that all solutions have limits, we first show that  $x(t) \notin L^1(\mathbb{R}_+)$ . To this end, suppose for the purpose of contradiction, that  $x(t) \in L^1(\mathbb{R}_+)$ . Since  $\lim_{t \rightarrow \infty} f(t) = f_{\infty} > 0$  and

$$0 < e^{-\gamma \int_0^{\infty} x(s) ds} < \infty,$$

then from (3.2.5), we see that  $a(t)$  is unbounded and  $\lim_{t \rightarrow \infty} a(t) = \infty$ .

Consider now the integrand in the denominator of (3.2.4). Using L'Hopital's Rule, we can write

$$\begin{aligned}
 & \lim_{t \rightarrow \infty} \frac{e^{\int_0^t [r_0 - r_1 c_0(s)] ds}}{a(t)} \\
 &= \lim_{t \rightarrow \infty} \frac{e^{\int_0^t [r_0 - r_1 c_0(s)] ds}}{e^{-\gamma \int_0^t x(s) ds} \left[ a_0 + \int_0^t f(s) e^{\int_0^s x(s_1) ds_1} ds \right]} \\
 &= e^{\gamma \int_0^\infty x(s) ds} \lim_{t \rightarrow \infty} \frac{[r_0 - r_1 c_0(t)] e^{\int_0^t [r_0 - r_1 c_0(s)] ds}}{f(t) e^{\gamma \int_0^t x(s) ds}} \\
 &= \frac{r_0 - r_1 c_0(\infty)}{f_\infty} \lim_{t \rightarrow \infty} e^{\int_0^t [r_0 - r_1 c_0(s)] ds} \\
 &= \infty .
 \end{aligned}$$

Thus, the denominator of (3.2.4) becomes infinite and so an application of L'Hopital's Rule yields

$$\liminf_{t \rightarrow \infty} x(t) = \liminf_{t \rightarrow \infty} \frac{r_0 - r_1 c_0(t)}{\alpha} \cdot a(t) = \infty$$

which is a contradiction. Hence,  $x(t) \notin L^1(\mathbb{R}_+)$ .

We now show both  $a$  and  $x$  are bounded. An application of L'Hopital's Rule to (3.2.5) yields

$$\limsup_{t \rightarrow \infty} a(t) = \limsup_{t \rightarrow \infty} \frac{f(t)}{\gamma x(t)} . \quad (3.2.10)$$

Suppose  $\limsup_{t \rightarrow \infty} x(t) = \infty$ . Then from (3.2.9), we see that

$\limsup_{t \rightarrow \infty} a(t) = \infty$ . From (3.2.10), since  $\lim_{t \rightarrow \infty} f(t) = f_{\infty} > 0$ , we

must have  $\lim_{t \rightarrow \infty} x(t) = 0$ , which is a contradiction. Thus,

$\limsup_{t \rightarrow \infty} x(t) < \infty$  and consequently,  $\limsup_{t \rightarrow \infty} a(t) < \infty$ .

Our next step is to show that  $\lim_{t \rightarrow \infty} dx/dt = 0$ . Let

$K > \limsup_{t \rightarrow \infty} a(t)$ . Then  $a(t) \leq K$  for all values of  $t$  sufficiently

large. Since for these values of  $t$ ,  $-1/a(t) \leq -1/K$ , we can write

$$\frac{dx}{dt} \leq x[r_0 - r_1 c_0 - \alpha x/K] .$$

Consider the logistic problem

$$\frac{dz}{dt} = z[r_0 - r_1 c_0 - \frac{\alpha z}{K}] , \quad z(0) = x_0 .$$

It is easily verified that if  $r_0 > r_1 c_0(\infty)$ , then

$$\lim_{t \rightarrow \infty} z(t) = \frac{K[r_0 - r_1 c_0(\infty)]}{\alpha} .$$

Consequently,  $\lim_{t \rightarrow \infty} dz/dt = 0$ .

We claim that  $\liminf_{t \rightarrow \infty} a(t) > 0$ . To show this, note in (3.2.10), that we can write

$$\limsup_{t \rightarrow \infty} a(t) = \lim_{t \rightarrow \infty} \frac{f(t)}{x(t)} \cdot \limsup_{t \rightarrow \infty} 1/x(t) .$$

Since  $\limsup_{t \rightarrow \infty} a(t) < \infty$ , we see that  $1/x(t)$  is bounded. Since  $x(t) \neq 0$  for all  $t$  (no finite time extinction) and does not change sign, then  $\limsup_{t \rightarrow \infty} x(t) = 1/\liminf_{t \rightarrow \infty} x(t)$ . Thus  $\liminf_{t \rightarrow \infty} x(t) > 0$ . Hence, an application of L'Hopital's Rule on (3.2.4) yields

$$0 < \liminf_{t \rightarrow \infty} x(t) = \frac{r_0 - r_1 c_0^{(\infty)}}{\alpha} \liminf_{t \rightarrow \infty} a(t) .$$

Hence, we may choose  $k$  such that  $\liminf_{t \rightarrow \infty} a(t) > k > 0$ .

Now, for large  $t$ ,  $a(t) \geq k$  and so  $-1/a(t) \geq -1/k$ . We can write

$$\frac{dx}{dt} \geq x[r_0 - r_1 c_0 - \frac{\alpha x}{k}] .$$

If we consider the logistic problem

$$\frac{dw}{dt} = w[r_0 - r_1 c_0 - \frac{\alpha w}{k}] , \quad w(0) = x_0 ,$$

then  $\lim_{t \rightarrow \infty} dw/dt = 0$ . We can write

$$\frac{dw}{dt} \leq \frac{dx}{dt} \leq \frac{dz}{dt}$$

Thus,  $\lim_{t \rightarrow \infty} \frac{dx}{dt} = 0$  . Consequently

$$\lim_{t \rightarrow \infty} x(t) [r_0 - r_1 c_0(t) - \frac{x(t)}{a(t)}] = 0 .$$

Since  $\liminf_{t \rightarrow \infty} x(t) > 0$  , we must have

$$\lim_{t \rightarrow \infty} [r_0 - r_1 c_0(t) - \frac{x(t)}{a(t)}] = 0 .$$

Hence

$$\lim_{t \rightarrow \infty} \frac{x(t)}{a(t)} = \frac{r_0 - r_1 c_0(\infty)}{\alpha} .$$

From (3.2.9) it is clear that if either  $x$  or  $a$  has a limit, so does the other. So assume for the purpose of contradiction that  $a(t)$  has no limit at infinity. Then, using

$$\limsup_{t \rightarrow \infty} a(t) \neq \liminf_{t \rightarrow \infty} a(t) ,$$

choose sequences  $\{t_n^M\}_{n=1}^{\infty}$  and  $\{t_n^m\}_{n=1}^{\infty}$  , such that  $t_n^M \rightarrow \infty$  ,  $t_n^m \rightarrow \infty$  as  $n \rightarrow \infty$  and  $a(t_n^M)$  is a local maximum and  $a(t_n^m)$  is a local minimum for each  $n$  . Consequently

$$\frac{da(t_n^M)}{dt} = \frac{da(t_n^m)}{dt} = 0 \quad \text{for each } n .$$

Using (3.2.2), we can write:

$$(i) \quad f(t_n^M) - \gamma a(t_n^M) x(t_n^M) = 0$$

$$(ii) \quad f(t_n^m) - \gamma a(t_n^m) x(t_n^m) = 0 .$$

Dividing (i) by  $[a(t_n^M)]^2$  and rearranging, we obtain

$$\frac{f(t_n^M)}{[a(t_n^M)]^2} = \frac{x(t_n^M)}{a(t_n^M)} , \quad n = 1, 2, 3, \dots .$$

Similarly from (ii) we get

$$\frac{f(t_n^m)}{[a(t_n^m)]^2} = \frac{x(t_n^m)}{a(t_n^m)} , \quad n = 1, 2, 3 \dots .$$

Since  $\lim_{t \rightarrow \infty} x(t)/a(t)$  exists, we see that

$$\begin{aligned} \lim_{n \rightarrow \infty} \frac{f(t_n^M)}{[a(t_n^M)]^2} &= \lim_{n \rightarrow \infty} \frac{f(t_n^m)}{[a(t_n^m)]^2} \\ &= \frac{\gamma[r_0 - r_1 c_0(\infty)]}{\alpha} . \end{aligned}$$

Since  $f$  has a limit, we must have

$$\lim_{n \rightarrow \infty} 1/[a(t_n^M)]^2 = \lim_{n \rightarrow \infty} 1/[a(t_n^m)]^2 .$$

From our definition of  $\{t_n^M\}$  and  $\{t_n^m\}$  it follows that

$$\liminf_{t \rightarrow \infty} 1/[a(t)]^2 = \limsup_{t \rightarrow \infty} 1/[a(t)]^2 ,$$

and so  $\lim_{t \rightarrow \infty} 1/[a(t)]^2$  exists. Since  $a(t)$  is positive, it is easily shown that  $1/a(t)$  has a limit, from which it follows that  $a(t)$  has a limit. Consequently,  $\limsup_{t \rightarrow \infty} a(t) = \liminf_{t \rightarrow \infty} a(t)$ , which contradicts our assumption. Hence,  $a(t)$  has a limit at infinity and so does  $x(t)$ .

From the relations

$$\lim_{t \rightarrow \infty} x(t) = \frac{r_0 - r_1 c_0}{\alpha} \cdot \lim_{t \rightarrow \infty} a(t)$$

and

$$\lim_{t \rightarrow \infty} a(t) = \frac{f_{\infty}}{\gamma \lim_{t \rightarrow \infty} x(t)},$$

the limits claimed in (i) and (ii) follow immediately.

### 3.3. Gallopin's Resource-Consumer Model

The second resource-consumer model considered here describes the dynamics of a population in a resource-limited environment. The resource-consumer model as originally proposed by Gallopin (1971), consists of a pair of non-linear differential equations; one describing the population size and the other describing the utilization of resource:

$$\frac{dx}{dt} = \alpha_2 \beta_1 x \left[ 1 - \frac{k}{\alpha_2 \beta_1} - e^{-\alpha_1 a/x} \right], \quad (3.3.1)$$

$$\frac{da}{dt} = f - \alpha_2 x \left[ 1 - e^{-\alpha_1 a/x} \right] \quad (3.3.2)$$

where as before,  $x = x(t)$  represents the biomass (in resource units) of the population and  $a = a(t)$  represents the bioavailable resources. The parameter  $\alpha_1$  is a dimensionless positive constant;  $\beta_1$  is also a dimensionless positive constant representing an assimilation efficiency;  $\alpha_2$  is the ingestion rate per unit mass that corresponds to maintenance of complete satiation and  $k$  is the maintenance cost per unit mass. The term  $\alpha_2\beta_1 - k$  in (3.3.1) is the difference between the maintenance cost per unit mass and the assimilation rate per unit mass. That  $\alpha_2\beta_1 - k$  is the rate at which food is transformed into biomass and hence, is the intrinsic growth rate of the population. In this chapter, we will assume that  $\alpha_2\beta_1 > k$ . From biological considerations, this conditions means that the population has potential for growth if given enough food.

As with the Leslie type model, the coupling of the toxicant and the consumer population is again accomplished by assuming a linear-dose response function so that  $r = r_0 - r_1c_0$  where  $r_0 = \alpha_2\beta_1 - k$ . The Gallopin-type resource-consumer-toxicant model (G) consists of:

$$\frac{dx}{dt} = x[r_0 - r_1c_0 - \alpha_2\beta_1 e^{-\alpha_1 a/x}] \quad (3.3.3)$$

$$\frac{da}{dt} = f - \alpha_2 x[1 - e^{-\alpha_1 a/x}] \quad (3.3.2)$$

$$\frac{dc_0}{dt} = a_1c_E - l_1c_0 - l_2c_0 + \frac{d_1 \alpha_2\beta_1\theta}{a_1} \quad (3.3.4)$$

$$\frac{dc_E}{dt} = g - k_2c_E \quad (3.3.5)$$

with initial conditions:

$$x(0) = x_0 > 0, a(0) = a_0 > 0, c_0(0) = 0, c_E(0) = c_{E0} > 0.$$

The other parameters and functions are as interpreted in the Leslie-type model.

A distinction between the Leslie-type and Gallopin-type formulation is that the resource component in Gallopin's model contains a consumer density dependent representation. In both models, however, the toxicant dynamics and the effects on the population are reflected in a linear dose-response function.

### Persistence-Extinction Results for Gallopin's Model

The extinction of the population depends intricately upon the input of toxicant. Moreover, if the consumer population exhibits a potential for growth and there is input of resource, then the population is persistent. These remarks are made precise in the next theorem.

THEOREM 3.3.1. Let  $0 \leq g_m \leq g(t) \leq g_M$  and define

$$\rho_m = \frac{d_1 \alpha_2 \beta_1 \theta}{a_1(\ell_1 + \ell_2)} + \frac{a_1 g_m}{k_2(\ell_1 + \ell_2)},$$

$$\rho_M = \frac{d_1 \alpha_2 \beta_1 \theta}{a_1(\ell_1 + \ell_2)} + \frac{a_1 g_M}{k_2(\ell_1 + \ell_2)}.$$

- (i) Extinction of  $x$ : If the population has negative growth potential in the sense that  $r_0 < r_1 \rho_m$ , then extinction results, that is,  $\lim_{t \rightarrow \infty} x(t) = 0$ .

- (ii) Persistence of  $x$ : If the population has a positive growth potential in the sense that  $r_0 > r_1 c_M$  and if  $\limsup_{t \rightarrow \infty} f(t) > 0$ , then the population is persistent,  $\limsup_{t \rightarrow \infty} x(t) > 0$ .

PROOF. To prove (i), note that from (3.3.3), we can write

$\frac{dx}{dt} \leq x[r_0 - r_1 c_0]$ . Consider the linear problem

$$\frac{dy}{dt} = y[r_0 - r_1 c_0], \quad y(0) = x_0.$$

Solving for  $y$ , we obtain

$$y(t) = y_0 e^{\int_0^t [r_0 - r_1 c_0(s)] ds}.$$

As in the proofs of our previous models, it can be shown that

$$c_0(t) \geq \frac{d_1 \alpha_2 \beta_1 \theta}{a_1 (\ell_1 + \ell_2)} + \frac{a_1 g_m}{k_2 (\ell_2 + \ell_2)} + o(1) \geq \rho_m + o(1).$$

Hence,

$$y(t) \leq y_0 e^{\int_0^t (r_0 - r_1 [\rho_m + o(1)]) ds}.$$

Since  $r_0 < r_1 \rho_m$ , then for  $t$  sufficiently large,

$r_0 - r_1 [\rho_m + o(1)] < 0$ , so that  $\lim_{t \rightarrow \infty} y(t) = 0$ . From differential

inequality theory we conclude  $\lim_{t \rightarrow \infty} x(t) = 0$ . This establishes (i).

An upper estimate for  $c_0$  is easily seen to be  $c_0 \leq \rho_M + o(1)$ .

Consider (3.3.3). We can write

$$\frac{dx}{dt} \geq x[r_0 - r_1(\rho_M + \underline{o}(1)) - \alpha_2 \beta_1 e^{-\alpha_1 a/x}] .$$

Consider the system

$$\frac{dy}{dt} = y[r_0 - r_1(\rho_M + \underline{o}(1)) - \alpha_2 \beta_1 e^{-\alpha_1 a/y}], \quad y(0) = x_0. \quad (3.3.5)$$

This is a Gallop-in-type problem and so the population will grow if given enough food, i.e.,  $\limsup_{t \rightarrow \infty} y(t) > 0$  if

$$\limsup_{t \rightarrow \infty} a(t) > 0 \quad \text{and} \quad r_0 > r_1(\rho_M + \underline{o}(1)) .$$

But from (3.3.2), we can write

$$\frac{da}{dt} \geq f - \alpha_2 x + \alpha_2 x \left[1 - \frac{\alpha_1 a}{x}\right] = f - \alpha_2 \alpha_1 a .$$

Hence,

$$a(t) \geq e^{-\alpha_1 \alpha_2 t} \left[ a_0 + \int_0^t f(s) e^{(\alpha_1 \alpha_2)s} ds \right] .$$

An application of L'Hopital's Rule yields

$$\limsup_{t \rightarrow \infty} a(t) \geq \limsup_{t \rightarrow \infty} \frac{f(t)}{\alpha_1 \alpha_2} > 0 ;$$

thus  $\limsup_{t \rightarrow \infty} y(t) > 0$  and from differential inequality theory

$$\limsup_{t \rightarrow \infty} x(t) > 0 .$$

### Asymptotic Analysis of the Model

THEOREM 3.3.2. If  $\lim_{t \rightarrow \infty} f(t) = f_{\infty}$  and  $\lim_{t \rightarrow \infty} g(t) = g_{\infty}$  exist and are positive and  $r_0 > r_1 c_0(\infty)$ , then the system (G) has an asymptotically stable positive equilibrium whose components are given by

$$(i) \quad \lim_{t \rightarrow \infty} x(t) = x(\infty) = \frac{\beta_1 f_{\infty}}{\alpha_2 \beta_1 - (r_0 - r_1 c_0(\infty))}$$

$$(ii) \quad \lim_{t \rightarrow \infty} a(t) = a(\infty) = -\frac{x(\infty)}{\alpha_1} \ln \left[ \frac{r_0 - r_1 c_0(\infty)}{\beta_1 \alpha_2} \right]$$

$$(iii) \quad \lim_{t \rightarrow \infty} c_0(t) = \frac{\alpha_1 \beta_1 \alpha_2 \theta}{a_1(\ell_1 + \ell_2)} + \frac{a_1 g_{\infty}}{k_2(\ell_1 + \ell_2)}$$

$$(iv) \quad \lim_{t \rightarrow \infty} c_E(t) = \frac{g_{\infty}}{k_2}$$

PROOF. The limits in (iii) and (iv) follow immediately from an application of L'Hopital's Rule to the integral representations of (3.3.4) and (3.3.5) respectively. The limits in (i) and (ii) are obtained by setting the left hand side of (3.3.3) and (3.3.2) to zero and solving the resulting system of equations for  $a$  and  $x$ .

To establish the asymptotic stability of this equilibrium solution, we use a theorem in Coddington and Levinson (1955, p. 327). Thus, let

$$f_1(x, a, c_0, c_E) = x[r_0 - r_1 c_0 - \alpha_2 \beta_1 e^{-\alpha_1 a/x}]$$

$$f_2(x, a, c_0, c_E) = f - \alpha_2 x[1 - e^{-\alpha_1 a/x}] .$$

A linearization of  $f_1$  and  $f_2$  about the solution  $(x(\infty), a(\infty), c_0(\infty), c_E(\infty))$  yields:

$$\begin{aligned} \frac{dx}{dt} = & A_1(x - x(\infty)) + A_2(a - a(\infty)) + A_3(c_0 - c_0(\infty)) \\ & + g_1(x, a, c_0, c_E) , \end{aligned}$$

$$\frac{da}{dt} = B_1(x - x(\infty)) + B_2(a - a(\infty)) + f - f_\infty + g_2(x, a, c_0, c_E) ,$$

where

$$A_1 = - \frac{\alpha_1 \alpha_2 \beta_1 a(\infty)}{x(\infty)} e^{-\alpha_1 a(\infty)/x(\infty)} ,$$

$$A_2 = \alpha_1 \alpha_2 \beta_1 e^{-\alpha_1 a(\infty)/x(\infty)} ,$$

$$A_3 = -r_1 x(\infty) ,$$

$$B_1 = -\alpha_2 + \alpha_2 \left(1 + \frac{\alpha_1 a(\infty)}{x(\infty)}\right) e^{-\alpha_1 a(\infty)/x(\infty)} ,$$

$$B_2 = -\alpha_1 \alpha_2 e^{-\alpha_1 a(\infty)/x(\infty)} ;$$

$g_1$  and  $g_2$  contain all terms with higher order derivatives.

Note that we can also write

$$\frac{dc_0}{dt} = a_1(c_E - c_E(\infty)) - (\ell_1 + \ell_2)(c_0 - c_0(\infty))$$

$$\frac{dc_E}{dt} = (g - g_\infty) - k_2(c_E - c_E(\infty)) .$$

Let

$$X = \begin{bmatrix} x - x(\infty) \\ a - a(\infty) \\ c_0 - c_0(\infty) \\ c_E - c_E(\infty) \end{bmatrix}$$

Then model (G) can be written in the equivalent form

$$\frac{dX}{dt} = AX + F(t, X) + G(t, X) , \quad (3.3.6)$$

where

$$F(t, X) = \begin{bmatrix} g_1(x, a, c_0, c_E) \\ g_2(x, a, c_0, c_E) \\ 0 \\ 0 \end{bmatrix}$$

and

$$G(t, X) = \begin{bmatrix} 0 \\ f - f_\infty \\ 0 \\ g - g_\infty \end{bmatrix}$$

$$A = \begin{bmatrix} A_1 & A_2 & A_3 & 0 \\ B_1 & B_2 & 0 & 0 \\ a_1 & -(\ell_1 + \ell_2) & 0 & 0 \\ 0 & 0 & 0 & -k_2 \end{bmatrix}$$

We now show that the characteristic roots of  $A$  are negative or have negative real parts. The characteristic equation of  $A$  is given by

$$P(\lambda) = (-k_2 - \lambda)(-(\ell_1 + \ell_2) - \lambda)[(A_1 - \lambda)(B_2 - \lambda) - B_1 A_2] .$$

Two roots are  $\lambda_3 = -(\ell_1 + \ell_2)$  and  $\lambda_4 = -k_2$ . From

$$(A_1 - \lambda)(B_2 - \lambda) - B_1 A_2 = 0$$

we obtain

$$\lambda = \frac{(A_1 + B_2) \pm [(A_1 + B_2)^2 - 4(A_1 B_2 - B_1 A_2)]^{\frac{1}{2}}}{2} \quad (3.3.7)$$

Observe that  $A_1 + B_2 < 0$  and  $A_1 B_2 > 0$ . Consider

$$B_1 = -\alpha_2 + \alpha_2 \left(1 + \frac{\alpha_1 a(\infty)}{x(\infty)}\right) e^{-\alpha_1 a(\infty)/x(\infty)} .$$

Since

$$1 + \frac{\alpha_1 a(\infty)}{x(\infty)} < e^{\alpha_1 a(\infty)/x(\infty)} ,$$

then

$$\alpha_2 \left(1 + \frac{\alpha_1 a(\infty)}{x(\infty)}\right) < \alpha_2 e^{\alpha_1 a(\infty)/x(\infty)} .$$

Rewriting, we obtain

$$-\alpha_2 + \alpha_2 \left(1 + \frac{\alpha_1 a(\infty)}{x(\infty)}\right) e^{-\alpha_1 a(\infty)/x(\infty)} < 0 .$$

Consequently,  $B_1 < 0$  . Since  $A_2 > 0$  , we see that

$$A_1 B_2 - B_1 A_2 > 0 .$$

We conclude that the two roots  $\lambda_1, -\lambda_2$  in (3.3.7) are either negative or have negative real parts.

Consider now  $f(t, X)$  . From Taylor's Theorem with remainder, we can write

$$\begin{aligned} g_1(x, a, c_0, c_E) &= A_5(z_1 - x(\infty))^2 + A_6(z_2 - a(\infty))^2 \\ &+ A_9(z_1 - x(\infty))(z_2 - a(\infty)) + A_{10}(z_1 - x(\infty))(z_3 - c_0(\infty)) \end{aligned}$$

and

$$\begin{aligned} g_2(x, a, c_0, c_E) &= B_5(z_1 - x(\infty))^2 + B_6(z_2 - a(\infty))^2 \\ &+ B_9(z_1 - x(\infty))(z_2 - a(\infty)) , \end{aligned}$$

where  $z_1, z_2$ , and  $z_3$  lie between  $x(t)$  and  $x(\infty)$ ,  $a(t)$  and  $a(\infty)$ ,  $c_0(t)$  and  $c_0(\infty)$ , respectively;

$$A_5 = -\alpha_1^2 \alpha_2 \beta_1 \frac{[a(\infty)]^2}{[x(\infty)]^3} e^{-\alpha_1 a(\infty)/x(\infty)},$$

$$A_6 = -\frac{\alpha_1^2 \alpha_2 \beta_1}{x(\infty)} e^{-\alpha_1 a(\infty)/x(\infty)},$$

$$A_9 = \alpha_1^2 \alpha_2 \beta_1 \frac{a(\infty)}{[x(\infty)]^2} e^{-\alpha_1 a(\infty)/x(\infty)},$$

$$A_{10} = -r_1,$$

$$B_5 = \alpha_1^2 \alpha_2 \frac{[a(\infty)]^2}{[x(\infty)]^3} e^{-\alpha_1 a(\infty)/x(\infty)},$$

$$B_6 = \frac{\alpha_1^2 \alpha_2}{x(\infty)} e^{-\alpha_1 a(\infty)/x(\infty)},$$

$$B_9 = -\frac{\alpha_1^2 a(\infty)}{[x(\infty)]^2} e^{-\alpha_1 a(\infty)/x(\infty)}.$$

If  $B = (b_{ij})$  is a matrix, define  $||B|| = [\sum_{i,j} b_{ij}^2]^{\frac{1}{2}}$ . Hence

$$||F(t, x)||^2 = [g_1(x, a, c_0, c_E)]^2 + [g_2(x, a, c_0, c_E)]^2,$$

$$||X||^2 = (x - x(\infty))^2 + (a - a(\infty))^2 \\ + (c_0 - c_0(\infty))^2 + c_E - c_E(\infty))^2.$$

Since  $|z_1 - x(\infty)| \leq |x - x(\infty)|$ ,  $|z_2 - a(\infty)| \leq |a - a(\infty)|$ , and  $|z_2 - c_0(\infty)| \leq |c_0 - c_0(\infty)|$ , then

$$\begin{aligned} ||X|| &\geq |x - x(\infty)| \\ &\geq |a - a(\infty)| \\ &\geq |c_0 - c_0(\infty)|. \end{aligned}$$

We can write

$$\begin{aligned} \frac{||F(t, X)||^2}{||X||^2} &\leq (A_5^2 + A_{10}^2 + 2|A_5 A_6| + B_5^2 + 2|B_5 B_6|) \cdot \quad (3.3.8) \\ &\cdot |x - x(\infty)|^2 + (A_6^2 + A_9^2 + B_6^2 + B_9^2) |a - a(\infty)|^2 \\ &+ (2|A_5 A_9| + 2|A_6 A_9| + 2|B_5 B_9| + 2|B_6 B_9|) \cdot \\ &\cdot |x - x(\infty)| |a - a(\infty)| + 2|A_9 A_{10}| |a - a(\infty)| |c_0 - c_0(\infty)| \\ &+ (2|A_5 A_{10}| + 2|A_6 A_{10}|) |x - x(\infty)| |c_0 - c_0(\infty)| \end{aligned}$$

Let  $K$  be the maximum of all constant coefficients in (3.3.8). Then we can write

$$\begin{aligned} \frac{||F(t, X)||^2}{||X||^2} &\leq K\{|x - x(\infty)|^2 + |a - a(\infty)|^2 + |x - x(\infty)| |a - a(\infty)| \\ &+ |x - x(\infty)| |c_0 - c_0(\infty)| + |a - a(\infty)| |c_0 - c_0(\infty)| \}. \end{aligned}$$

Let  $\varepsilon > 0$  be given and assume  $||X|| \leq \delta$ , where  $\delta$  is to be chosen

later. We can write

$$\frac{||F(t, X)||^2}{||X||^2} \leq 5K \delta^2 .$$

Choose  $\delta = \frac{\epsilon}{(5K)^{\frac{1}{2}}}$  and choose  $t$  such that  $||X|| \leq \delta$  for  $t \geq t_\epsilon$ .

Then  $||F(t, X)|| \leq \epsilon ||X||$  for all  $t \geq t_\epsilon$ . Also note that  $G(t, X) \rightarrow 0$  as  $t \rightarrow \infty$ , since  $f \rightarrow f_\infty$  and  $g \rightarrow g_\infty$  as  $t \rightarrow \infty$ . Asymptotic stability is now established.

### 3.4. Continuous Culture Model

The dynamics of a continuous culture model have been described in the literature (Herbert, et al., 1956; Hsu, et al., 1977). The basic equations of this model are the pair:

$$\frac{dx}{dt} = x[\mu(s) - D] \tag{3.4.1}$$

$$\frac{ds}{dt} = D[s_R - s] - x \mu(s)/y \tag{3.4.2}$$

where  $x = x(t)$  denotes the concentration of the organism at time  $t$ ,  $s$  denotes the concentration of limiting substrate,  $\mu(s) = \mu_m s/(K + s)$  denotes the specific growth rate of the organisms with  $\mu_m$  the maximum growth rate,  $K$  is the half saturation constant,  $y$  is the yield coefficient,  $D$  is the dilution rate and  $s_R$  is the concentration of the substrate entering the culture vessel.

The parameter  $\mu_m$  and  $K$  reflect characteristics of the particular organism-substrate complex under study (Holmberg, 1982). The

intrinsic growth rate,  $\mu(s)$  has two features that are important biologically (Hsu, et al., 1977; Filov, et al., 1973):

- (i) When the substrate concentration is low, the growth rate is proportional to substrate concentration:  $\mu(s) \sim \mu_m s/K$ .
- (ii) At high substrate concentration, the growth rate saturates at  $\mu_m$  and is thus independent of substrate concentration.

The continuous culture model has an equilibrium given by

$$s(\infty) = KD/(\mu_m - D) ,$$

$$x(\infty) = y[s_R - s(\infty)] .$$

In order that this equilibrium be positive, the inequalities

$$\mu_m > D \quad \text{and} \quad s_R > s(\infty)$$

must be satisfied. We shall tacitly assume in the sequel that  $\mu_m > D$  so that the population has the potential to grow given sufficient substrate.

Continuous culture techniques and models have existed for many years (Novick and Szilard, 1950; Herbert, et al., 1956; Hsu, et al., 1977). Moreover, the chemostat, being "the best laboratory idealization of nature for population studies" (Hsu, et al., 1977) is a good vehicle to study effects of toxicants on population. We will modify the fundamental equations of the continuous culture model to incorporate toxicant effects, couple this system with the dynamics of  $c_0$  and

$c_E$  , and then determine the qualitative behavior of the resulting resource-consumer-toxicant interaction.

We assume that the population is subjected to a chronic dose of toxicant so the inflowing medium contains a constant concentration of toxicant, say  $c_R$  , and the culture vessel medium attains a dynamic toxicant concentration of  $c_E$  . The resource introduced into the system is assumed to be contaminated by toxicant; in particular, it is required that the resource contains a constant toxicant concentration,  $c_1$  . This setting allows the resource and the environment to be sources of the toxicant for the consumers.

To be consistent with our previous studies, we assume that the growth rate of the population is decreased by an amount proportional to  $c_0(t)$  ; hence, the growth rate of the consumer population with toxicant present is

$$\bar{\mu} = \mu(s) - r_1 c_0 . \quad (3.4.3)$$

The continuous culture model with toxicant stress becomes

$$\frac{dx}{dt} = x[\mu(s) - r_1 c_0 - D] \quad (3.4.4)$$

$$\frac{ds}{dt} = D[s_R - s] - x[\mu(s) - r_1 c_0]/y . \quad (3.4.5)$$

We now turn to the dynamics of  $c_0$  and  $c_E$  . Let  $G$  be the rate of toxicant uptake from the resource per unit mass. We utilize the definition of the yield coefficient to find that the resource

consumed per unit mass is  $\bar{\mu}/y$ . Thus we take

$$G = d_1 c_1 (\mu(s) - r_1 c_0) / a_1 y$$

where  $d_1/a_1$  represents a coupling of the toxicant pathways (as in the previous two models). This leads to the equation

$$\frac{dc_0}{dt} = a_1 c_E + d_1 c_1 [\mu(s) - r_1 c_0] / a_1 y - (\lambda_1 + \lambda_2) c_0. \quad (3.4.6)$$

When this equation is contrasted with the dynamics for  $c_0$  used in connection with the Leslie- and Gallopian-type models, an additional complexity arises in that the dynamics of the resource are now coupled in the  $c_0$  equation.

If the toxicant uptake from the medium is small, the toxicant input and toxicant washout are the dominant factors modifying the concentration of toxicant in the culture vessel. Employing this as an hypothesis, we may express the dynamics of  $c_E$

$$\frac{dc_E}{dt} = D[c_R - c_E]. \quad (3.4.7)$$

The continuous culture resource-consumer-toxicant model (CT) consists of equation (3.4.4), (3.4.5), (3.4.6), and (3.4.7):

$$\frac{dx}{dt} = x[\mu(s) - r_1 c_0 - D]$$

$$\frac{ds}{dt} = D[s_R - s] - x[\mu(s) - r_1 c_0]/y$$

$$\frac{dc_0}{dt} = a_1 c_E + d_1 c_1 [\mu(s) - r_1 c_0]/a_1 y - (\ell_1 + \ell_2) c_0$$

$$\frac{dc_E}{dt} = D[c_R - c_E]$$

together with the initial conditions

$$x(0) = x_0 > 0, \quad s(0) = s_R, \quad c_0(0) = 0, \quad c_E(0) = c_{E0} > 0$$

and

$$\mu(s) = \mu_m s/(K + s).$$

THEOREM 3.4.1. The components of the solution of systems (CT) are positive and bounded on  $\mathbb{R}_+$ .

PROOF. The fact that  $x$ ,  $c_0$  and  $c_E$  are positive follows immediately from their respective integral representations:

$$x(t) = x_0 e^{\int_0^t \left[ \frac{\mu_m s(z)}{K + s(z)} - r_1 c_0(z) - D \right] dz}, \quad (3.4.8)$$

$$c_0(t) = e^{-\left[ \frac{d_1 c_1 r_1}{a_1 y} + \ell_1 + \ell_2 \right] t} \cdot$$

$$\cdot \int_0^t \left[ a_1 c_E(z) + \frac{d_1 c_1}{a_1 y} \frac{\mu_m s(z)}{K + s(z)} \right] dz.$$

$$\cdot e^{\left[\frac{d_1 c_1 r_1}{a_1 y} + \ell_1 + \ell_2\right]z} dz, \quad (3.4.9) \text{ continued}$$

$$c_E(t) = e^{-Dt} \left[ c_{E0} + \int_0^t D c_R e^{Dz} dz \right]. \quad (3.4.10)$$

The positivity of  $s$  is shown by contradiction. For suppose  $s(t_1) < 0$ . Then, since  $s_0 > 0$ , there exists a  $t_0 > 0$  such that  $s(t_0) = 0$  and  $s(t) > 0$  for  $0 \leq t < t_0$ . Now for  $0 \leq t \leq t_0$ , it follows that

$$\frac{ds}{dt} \geq -Ds - \frac{\mu_m x s}{Ky} + \frac{r_1 x c_0}{y}$$

An integration leads to

$$s(t_0) \geq e^{-\int_0^{t_0} \left[ D + \frac{\mu_m x(t)}{Ky} \right] dt} \cdot \left[ s_0 + \int_0^{t_0} \frac{r_1 x(t) c_0(t)}{y} e^{\int_0^t \left( D + \frac{\mu_m x(z)}{Ky} \right) dz} dt \right] > 0.$$

This contradiction establishes that  $s(t) > 0$  for all  $t$  in  $\mathbb{R}_+$ .

To show that the components  $x$  and  $s$  of model (CT) are bounded, it is convenient to consider the function

$$w(t) = (x(t)/y) + s(t)$$

Using (3.4.4) and (3.4.5) leads to

$$\frac{dw}{dt} = -Dw + Ds_R . \quad (3.4.11)$$

This is a first order linear differential equation and has all solutions bounded; hence  $x$  and  $s$  are bounded.

The boundedness of  $c_0$  and  $c_E$  follows from (3.4.9) and (3.4.10), respectively; in fact,

$$\limsup_{t \rightarrow \infty} c_0(t) \leq \frac{a_1 c_R + \frac{d_1 c_1 \mu_m}{a_1 y}}{\frac{r_1 d_1 c_1}{y} + \ell_1 + \ell_2}$$

This completes the proof.

THEOREM 3.4.2. If  $\lim_{t \rightarrow \infty} s(t) = s(\infty)$  exists, then the system (CT) has a feasible equilibrium  $(x(\infty), s(\infty), c_0(\infty), c_E(\infty))$  given by

$$\begin{aligned} \text{(i)} \quad & \lim_{t \rightarrow \infty} c_E(t) = c_E(\infty) = c_R . \\ \text{(ii)} \quad & \lim_{t \rightarrow \infty} x(t) = x(\infty) = y(s_R - s(\infty)) , \\ \text{(iii)} \quad & \lim_{t \rightarrow \infty} c_0(t) = c_0(\infty) = \frac{a_1 c_R + \frac{d_1 c_1 D}{a_1 y}}{\ell_1 + \ell_2} \\ \text{(iv)} \quad & \lim_{t \rightarrow \infty} s(t) = s(\infty) = \frac{K(r_1 c_0(\infty) + D)}{\mu_m - r_1 c_0(\infty) - D} , \end{aligned}$$

provided  $\mu_m - D > r_1 c_0(\infty)$  and  $s_R > s(\infty)$ . Moreover, this equilibrium solution is locally asymptotically stable.

PROOF. The limit in (i) follows from an application of L'Hopital's Rule to (3.4.10).

To show (ii), note that from (3.4.11) we can write

$$w(t) = e^{-Dt} [w(0) + s_R(e^{Dt} - 1)]$$

or, equivalently,

$$\frac{x(t)}{y} + s(t) = e^{-Dt} (x_0 + s_R) + s_R(1 - e^{-Dt}) . \quad (3.4.12)$$

Taking limits yields (ii) immediately.

To show (iii), we apply L'Hopital's Rule in (3.4.9) to obtain

$$\lim_{t \rightarrow \infty} c_0(t) = \frac{a_1 c_R + \frac{c_1}{y} \lim_{t \rightarrow \infty} \frac{\mu_m s(t)}{K + s(t)}}{r_1 \frac{c_1}{y} + \ell_1 + \ell_2} .$$

Since by hypothesis  $s(t)$  has a limit, we see that  $\lim_{t \rightarrow \infty} c_0(t)$

exists. To obtain the limit in (iii), note that from (3.4.8) we can write

$$0 < \lim_{t \rightarrow \infty} x(t) = \lim_{t \rightarrow \infty} x_0 \exp \left( \int_0^t \left[ \frac{\mu_m s(z)}{K + s(z)} - r_1 c_0(z) - D \right] dz \right) < \infty . \quad (3.4.13a)$$

Let

$$F(t) = \frac{\mu_m s(t)}{K + s(t)} - r_1 c_0(t) - D .$$

Then  $F$  has a limit and we claim that  $\lim_{t \rightarrow \infty} F(t) = 0$ . For if

$\lim_{t \rightarrow \infty} F(t) = L \neq 0$ , then for each  $\varepsilon > 0$ , there exists  $t_0 > 0$

sufficiently large such that for all  $t \geq t_0$ ,  $|F(t) - L| < \varepsilon$ ,  
or equivalently

$$L - \varepsilon < F(t) < L + \varepsilon .$$

If  $L > 0$ , choose  $\varepsilon > 0$  so that  $L - \varepsilon > 0$ . Using the left hand inequality, we obtain

$$\int_{t_0}^t F(z) dz > (L - \varepsilon)(t - t_0) .$$

Consequently,  $\lim_{t \rightarrow \infty} \int_0^t F(z) dz = \infty$  and so  $\lim_{t \rightarrow \infty} x(t) = \infty$  which contradicts (3.4.13a). If  $L < 0$ , choose  $\varepsilon > 0$  so that  $L + \varepsilon < 0$ .

Then, using the right hand inequality yields

$$\int_{t_0}^t F(z) dz < (L + \varepsilon)(t - t_0) .$$

Hence  $\lim_{t \rightarrow \infty} \int_0^t F(z) dz = -\infty$ . As a result,  $\lim_{t \rightarrow \infty} x(t) = 0$ , which again contradicts (3.4.13a). Thus

$$\lim_{t \rightarrow \infty} \left[ \frac{\mu_m s(t)}{K + s(t)} - r_1 c_0(t) - D \right] = 0 . \quad (3.4.14)$$

Consequently,

$$\lim_{t \rightarrow \infty} \frac{\mu_m s(t)}{K + s(t)} = r_1 c_0^{(\infty)} + D .$$

Substituting this limit in (3.4.13) and solving for  $c_0(\infty)$ , we obtain the limit in (iii).

The limit in (iv) is obtained by writing (3.4.14) in the form

$$\frac{\mu_m s(\infty)}{K + s(\infty)} - r_1 c_0(\infty) - D = 0$$

and solving for  $s(\infty)$ .

It remains to show the asymptotic stability of this feasible equilibrium. To this end, let

$$f_1(x, s, c_0, c_E) = x \left[ \frac{\mu_m s}{K + s} - r_1 c_0 - D \right]$$

$$f_2(x, s, c_0, c_E) = D[s_R - s] - \frac{x}{y} \left[ \frac{\mu_m s}{K + s} - r_1 c_0 \right]$$

$$f_3(x, s, c_0, c_E) = a_1 c_E + \frac{c_1}{y} \left[ \frac{\mu_m s}{K + s} - r_1 c_0 \right] - (\ell_1 + \ell_2) c_0$$

$$f_4(x, s, c_0, c_E) = D[c_R - c_E] .$$

Expanding  $f_1, f_2, f_3$ , and  $f_4$  by Taylor series about the feasible equilibrium  $(x(\infty), s(\infty), c_0(\infty), c_E(\infty))$ , we obtain

$$\begin{aligned} f_1(x, s, c_0, c_E) &= A_2(s - s(\infty)) + A_3(c_0 - c_0(\infty)) \\ &\quad + g_1(x, s, c_0, c_E) , \end{aligned}$$

$$\begin{aligned} f_2(x, s, c_0, c_E) &= B_1(x - x(\infty)) + B_2(s - s(\infty)) \\ &\quad + B_3(c_0 - c_0(\infty)) + g_2(x, s, c_0, c_E) , \end{aligned}$$

$$f_3(x, s, c_0, c_E) = C_2(s - s(\infty)) + C_3(c_0 - c_0(\infty)) \\ + C_4(c_E - c_E(\infty)) + g_3(x, s, c_0, c_E) ,$$

$$f_4(x, s, c_0, c_E) = -D(c_E - c_E(\infty)) ,$$

where

$$A_2 = \frac{\mu_m K x(\infty)}{(K + s(\infty))^2} ,$$

$$A_2 = -r_1 x(\infty) ,$$

$$B_1 = -D/y ,$$

$$B_2 = -D - \frac{x(\infty)}{y} \left[ \frac{\mu_m K}{(K + s(\infty))^2} \right] ,$$

$$B_3 = r_1 x(\infty)/y ,$$

$$C_2 = \frac{c_1 \mu_m K}{y(K + s(\infty))^2} ,$$

$$C_3 = -C_1 r_1 / y - (\lambda_1 + \lambda_2) ,$$

$$C_4 = a_1 \quad \text{and} \quad c_E(\infty) = c_R ;$$

$g_1, g_2, g_3$  contain all terms with higher order derivatives. Let

$$Y(t) = \begin{bmatrix} x - x(\infty) \\ s - s(\infty) \\ c_0 - c_0(\infty) \\ c_E - c_E(\infty) \end{bmatrix}$$

$$A = \begin{bmatrix} 0 & A_2 & A_3 & 0 \\ B_1 & B_2 & B_3 & 0 \\ 0 & C_2 & C_3 & C_4 \\ 0 & 0 & 0 & -D \end{bmatrix}$$

$$G(x, s, c_0, c_E) = \begin{bmatrix} g_1(x, s, c_0, c_E) \\ g_2(x, s, c_0, c_E) \\ g_3(x, s, c_0, c_E) \end{bmatrix}$$

Model (CT) can now be written in the form

$$\frac{dY}{dt} = AY + G(x, s, c_0, c_E)$$

We will use Hurwitz's criteria to show that the eigenvalue of  $A$  have negative real parts. The characteristic equation for  $A$  is given by

$$P(\lambda) = (\lambda + D)(\lambda^3 + P_2 \lambda^2 + P_1 \lambda + P_0) \quad (3.4.15)$$

where

$$P_2 = -(B_2 + C_3)$$

$$= D + \frac{\mu_m K x(\infty)}{y(K + s(\infty))^2} + r_1 C_1 / y + \ell_1 + \ell_2 ,$$

$$P_1 = B_2 C_3 - B_1 A_2 - C_2 B_3$$

$$= D(r_1 C_1 / y + \ell_1 + \ell_2) + \frac{\mu_m K}{y(K + s(\infty))^2} (\ell_1 + \ell_2 + D) ,$$

$$P_0 = B_1(A_2 C_3 - C_2 A_3)$$

$$= \frac{D(\ell_1 + \ell_2) \mu_m K x(\infty)}{y(K + s(\infty))^2} .$$

It is clear from (3.4.15) that one of the eigenvalues of  $P$  is  $\lambda_4 = -D < 0$  . So consider

$$\lambda^3 + P_2 \lambda^2 + P_1 \lambda + P_0 = 0 . \quad (3.4.16)$$

Hurwitz's criteria require that

$$P_2 > 0 , P_1 P_2 - P_0 > 0 , P_0 > 0 .$$

It is easily seen that  $P_2 > 0$  and  $P_0 > 0$  . To show that  $P_1 P_2 - P_0 > 0$  , note that  $P_1$  has a term  $D(\ell_1 + \ell_2)$  while  $P_2$  has a term  $\mu_m K x(\infty) / (Y(K + s(\infty))^2)$  . Therefore the product  $P_1 P_2$  has a term

$$\frac{D(\ell_1 + \ell_2) \mu_m K x(\infty)}{y(K + s(\infty))^2} = P_0 .$$

Since the remaining terms in  $P_1 P_2$  are all positive, we conclude that  $P_1 P_2 - P_0 > 0$  . Hence, the roots of (3.4.16) have negative real parts. Therefore, the eigenvalues of  $A$  are either negative or have negative real parts.

It remains to show that

$$\lim_{||Y(t)|| \rightarrow 0} \frac{G(x, s, c_0, c_E)}{||Y(t)||} = 0 .$$

It suffices to take  $||Z|| = (z_1^2 + z_2^2 + \dots + z_n^2)^{\frac{1}{2}}$  where  $Z = (z_1, z_2, \dots, z_n)^T$  . With this norm, we can write

$$\begin{aligned} ||G(x, s, c_0, c_E)||^2 &= (g_1(x, s, c_0, c_E))^2 \\ &\quad + (g_2(x, s, c_0, c_E))^2 + (g_3(x, s, c_0, c_E))^2 ; \\ ||Y(t)||^2 &= (x - x(\infty))^2 + (s - s(\infty))^2 + (c_0 - c_0(\infty))^2 \\ &\quad + (c_E - c_E(\infty))^2 . \end{aligned}$$

It suffices to show that

$$\frac{g_i^2(x, s, c_0, c_E)}{||Y(t)||} \rightarrow 0 , \text{ as } ||Y(t)|| \rightarrow 0 , \quad i = 1, 2, 3 .$$

From Taylor's Theorem with remainder, we can write

$$g_1(x, s, c_0, c_E) = A_6(z_2 - s(\infty))^2 + A_9(z_1 - x(\infty))(z_2 - s(\infty)) \\ + A_{10}(z_1 - x(\infty))(z_3 - c_0(\infty)) ;$$

$$g_2(x, s, c_0, c_E) = B_6(z_2 - s(\infty))^2 + B_9(z_1 - x(\infty))(z_2 - s(\infty)) \\ + B_{10}(z_1 - x(\infty))(z_3 - c_0(\infty)) ;$$

$$g_3(x, s, c_0, c_E) = C_6(z_2 - s(\infty))^2$$

where  $z_1, z_2$ , and  $z_3$  lie respectively between  $x$  and  $x(\infty)$ ,  $s$  and  $x(\infty)$ ,  $c_0$  and  $c_0(\infty)$ ; and where

$$A_6 = 2\mu_m K x(\infty)/(K + s(\infty))^3 ,$$

$$A_9 = \mu_m K/(K + s(\infty))^2 ,$$

$$A_{10} = -r_1 ,$$

$$B_6 = 2x(\infty)/y \cdot \mu_m K/(K + s(\infty))^2 ,$$

$$B_9 = -\mu_m K/y(K + s(\infty))^2 ,$$

$$B_{10} = r_1/y ,$$

$$C_6 = -2C_1 \mu_m K/y(K + s(\infty))^3 .$$

We have

$$\begin{aligned}
[g_1(x, s, c_0, c_E)]^2 &= A_6^2(z_2 - s(\infty))^4 + A_9(z_1 - x(\infty))^2 \cdot \\
&\cdot (z_2 - s(\infty))^2 + A_{10}^2(z_1 - x(\infty))^2 (z_3 - c_0(\infty))^2 + \\
&+ 2A_6A_9(z_1 - x(\infty))^2 \cdot (z_2 - s(\infty))^2 + 2A_6A_{10}(z_2 - s(\infty))^2 \cdot \\
&\cdot (z_1 - x(\infty))(z_3 - c_0(\infty)) + 2A_9A_{10}(z_1 - x(\infty))^2 \cdot \\
&\cdot (z_2 - s(\infty))(z_3 - c_0(\infty)) \\
&\leq A_6^2(s - s(\infty))^4 + A_9(x - x(\infty))^2 (s - s(\infty))^2 \\
&+ A_{10}^2(x - x(\infty))^2(c_0 - c_0(\infty))^2 + 2|A_6||A_9|(x - x(\infty))^2 \cdot \\
&\cdot (s - s(\infty))^2 + 2|A_6||A_{10}|(s - s(\infty))^2 |x - x(\infty)| \cdot \\
&\cdot |c_0 - c_0(\infty)| + 2|A_9||A_{10}|(x - x(\infty))^2 |s - s(\infty)||c_0 - c_0(\infty)|.
\end{aligned}$$

We also have:

$$\begin{aligned}
||Y(t)||^2 &= (x - x(\infty))^2 + (s - s(\infty))^2 + (c_0 - c_0(\infty))^2 \\
&\quad + (c_E - c_R)^2 \\
&\geq (x - x(\infty))^2
\end{aligned}$$

$$\geq (s - s(\infty))^2$$

$$\geq (c_0 - c_0(\infty))^2 .$$

Hence

$$\begin{aligned} \frac{[g_1(x, s, c_0, c_E)]^2}{||Y(t)||^2} &\leq \frac{A_6^2 |s - s(\infty)|^4}{|s - s(\infty)|^2} \\ &+ \frac{A_9^2 (x - x(\infty))^2 (s - s(\infty))^2}{|x - x(\infty)|^2} \\ &+ \frac{A_{10}^2 |x - x(\infty)|^2 |c_0 - c_0(\infty)|^2}{|x - x(\infty)|^2} \\ &+ \frac{2|A_6||A_9| |x - x(\infty)|^2 |s - s(\infty)|^2}{|s - s(\infty)|^2} \\ &+ \frac{2|A_6||A_{10}| (s - s(\infty))^2 |x - x(\infty)| |c_0 - c_0(\infty)|}{(s - s(\infty))^2} \\ &+ \frac{2|A_9||A_{10}| |x - x(\infty)|^2 |s - s(\infty)| |c_0 - c_0(\infty)|}{|x - x(\infty)|^2} \\ &\leq A_6^2 |s - s(\infty)|^2 + A_9^2 |s - s(\infty)|^2 + A_{10}^2 |c_0 - c_0(\infty)|^2 \\ &+ 2|A_6||A_9| |x - x(\infty)| |s - s(\infty)| + 2|A_6||A_{10}| |x - x(\infty)| , \end{aligned}$$

$$\begin{aligned}
& \cdot |c_0 - c_0(\infty)| \\
& + 2|A_9||A_{10}||s - s(\infty)||c_0 - c_0(\infty)|.
\end{aligned}$$

Since  $||Y(t)|| \rightarrow 0$  if and only if  $x - x(\infty) \rightarrow 0$ ,  $s - s(\infty) \rightarrow 0$

$$c_0 - c_0(\infty) \rightarrow 0, \text{ and } c_E - c_R \rightarrow 0,$$

we see that

$$\frac{[g_1(x, s, c_0, c_E)]^2}{||Y(t)||^2} \rightarrow 0 \text{ as } ||Y(t)|| \rightarrow 0.$$

Similarly,

$$\frac{[g_2(x, s, c_0, c_E)]^2}{||Y(t)||^2} \rightarrow 0 \text{ and } \frac{[g_3(x, s, c_0, c_E)]^2}{||Y(t)||^2} \rightarrow 0$$

as  $||Y(t)|| \rightarrow 0$ . By Liapunov's theorem (Rouche, et al., 1977, p. 31) we conclude that the feasible equilibrium is asymptotically stable.

THEOREM 3.4.3. (Extinction Theorem) If  $\lim_{t \rightarrow \infty} x(t) = 0$  then

either:

$$(i) \mu_m - D \leq r_1 c_0(\infty),$$

or

$$(ii) \mu_m - D > r_1 c_0(\infty) \text{ and } s_R \leq \frac{K[r_1 c_0(\infty) + D]}{\mu_m - r_1 c_0(\infty) - D}.$$

Conversely, if strict inequality holds in (i) and (ii), then

$$\lim_{t \rightarrow \infty} x(t) = 0 .$$

PROOF. From (3.4.12), we obtain

$$\lim_{t \rightarrow \infty} x(t) = 0 \quad \text{if and only if} \quad \lim_{t \rightarrow \infty} s(t) = s_R .$$

Consider the integral representation of  $x(t)$  given by (3.4.8).

Since  $x_0 > 0$  and  $\lim_{t \rightarrow \infty} x(t) = 0$ , we must have

$$\int_0^{\infty} \left[ \frac{\mu_m s(t)}{K + s(t)} - r_1 c_0(t) - D \right] dt = -\infty .$$

Consequently,  $\frac{\mu_m s(t)}{K + s(t)} - r_1 c_0(t) - D \notin L^1(\mathbb{R}_+)$ , and since this integrand has a limit, we must have

$$\frac{\mu_m s(\infty)}{K + s(\infty)} - r_1 c_0(\infty) - D \leq 0 . \quad (3.4.17)$$

Rearranging, and noting that  $s(\infty) = s_R$ , we obtain

$$[\mu_m - r_1 c_0(\infty) - D] s_R \leq K[r_1 c_0(\infty) + D] . \quad (3.4.18)$$

Hence either

(i)  $\mu_m - r_1 c_0(\infty) - D \leq 0$ , in which case (3.4.18) is trivially satisfied, or

(ii)  $\mu_m - r_1 c_0(\infty) - D > 0$  and from (3.4.18), we obtain

### 3.5. Computer Simulation

In order to show explicitly the persistence-extinction behavior of our resource-consumer-toxicant models, numerical solutions for particular values of the parameters and initial conditions are generated, using the IMSL subroutine DVERK. The population used is again Daphnia magna and the toxicant is diene-organochlorine dieldrin.

#### (i) Gallopin Type Model.

We first consider the Gallopin type resource-consumer-toxicant model. The unstressed system has been studied by Gallopin and particular values of the parameters and initial conditions have been determined. For the purpose of our simulation, the values  $\alpha_1 = 0.8$ ,  $\beta_1 = 0.9$  and  $\beta_2 = 0.6$  are assigned. We recall that  $r_0 = \alpha_2 \beta_1 - k$ . If we let  $k = 0.1$ , then  $r_0 = 0.44$ , which is the experimental value of the growth rate of D. magna as determined by Smith (1963). From section 2.9, we use the parameter values  $r_1 = 450.0$ ,  $\theta = 4.2 \times 10^{-6}$ , and  $k_2 = 0.005$  (see Table 2.9.2,II). In Theorem 3.3.2, we recall that the conditions  $\lim_{t \rightarrow \infty} f(t) = f_\infty$  and  $\lim_{t \rightarrow \infty} g(t) = g_\infty$  are positive constants and  $r_0 > r_1 c_0(\infty)$  are sufficient for the system to reach a steady state. Hence, for convenience, we may assume that the exogenous resource and toxicant inputs are constants. In particular,  $f(t) = 3.0$  and  $g(t) = 0.7 \times 10^{-10}$ .

To determine  $a_1$ ,  $d_1$  and  $\ell_1 + \ell_2$ , we consider (3.3.4) at equilibrium. Using the terminal toxicant concentrations in Reinert's experiments, we obtain the pair of non-linear equations in  $a_1$ ,  $d_1$ , and  $\ell_1 + \ell_2$ .

$$s_R \leq \frac{K[r_1 c_0(\infty) + D]}{\mu_m - r_1 c_0(\infty) - D}.$$

Conversely, suppose strict inequality holds in (i) and (ii); then strict inequality holds in (3.4.17). Consequently, there exists  $t_0 > 0$  sufficient large such that

$$\frac{\mu_m s(t)}{K + s(t)} - r_1 c_0(t) - D < 0$$

for all  $t \geq t_0$ . Thus

$$\int_0^\infty \left[ \frac{\mu_m s(t)}{K + s(t)} - r_1 c_0(t) - D \right] dt = -\infty,$$

and so  $\lim_{t \rightarrow \infty} x(t) = 0$ .

REMARKS. We see from the proof of Theorem 3.4.2 that extinction of the consumer occurs if and only if  $\lim_{t \rightarrow \infty} s(t) = s_R$ . Extinction is also determined by the growth potential of the population. Note that the inequality in (i) is a necessary condition for extinction, while if the inequality is strict, the condition is sufficient for extinction. Condition (ii), on the other hand, implies that the consumer population with growth potential may still be driven to extinction if it does not receive enough of the limiting substrate.

$$a_1(0.21 \times 10^{-8}) + \frac{d_1}{a_1} (0.6)(0.9)(4.2 \times 10^{-6}) \\ - (\ell_1 + \ell_2)(35 \times 10^{-6}) = 0$$

$$a_1(0.45 \times 10^{-8}) + \frac{d_1}{a_1} (0.6)(0.9)(4.2 \times 10^{-6}) \\ - (\ell_1 + \ell_2)(62 \times 10^{-6}) = 0 .$$

Assuming  $d_1$  is prescribed, then solving for  $a_1$  and  $\ell_1 + \ell_2$  yields:

$$a_1 = 47.361133 \sqrt{d_1}$$

$$\ell_1 + \ell_2 = 0.00420988 \sqrt{d_1} .$$

If  $d_1 = 1000.0$ , then  $a_1 = 1497.691$  and  $\ell_1 + \ell_2 = 0.13308$ . The parameter values and initial conditions used are summarized in Table 3.5.1.

Figure 3.5.1 shows the asymptotic behavior of the resource-consumer component as well as the body burden component. Since the dynamics of the environmental toxicant concentration  $c_E(t)$  is the same as in Chapter 2, its asymptotic behavior is no longer shown (see Chapter 2, Section 2.9). The resource-consumer component behavior is similar to the behavior of the original resource-population model by Gallopin. At the start, the resource accumulates since the population is still so small that resource ingestion does not significantly affect the available resource. Moreover, during the early period,

the body burden is relatively low. As a result, the population overshoots its steady state value. But when the body burden becomes important, the population declines until it reaches the equilibrium population size.

TABLE 3.5.1

A SUMMARY OF PARAMETER VALUES AND INITIAL CONDITIONS USED IN THE COMPUTER SIMULATION OF THE GALLOPIN TYPE MODEL. NON-BLANK ENTRIES IN COLUMN II REPRESENT CHANGES IN PARAMETER VALUES AND/OR INITIAL CONDITIONS IN COLUMN I.

| PARAMETERS        | I        | II | PARAMETERS         | I                      | II                   |
|-------------------|----------|----|--------------------|------------------------|----------------------|
| $\alpha_1$        | 0.8      |    | $\theta$           | $4.2 \times 10^{-6}$   |                      |
| $\beta_1$         | 0.9      |    | $-f_\infty$        | 3.0                    |                      |
| $\alpha_2$        | 0.6      |    | $g_\infty$         | $0.70 \times 10^{-10}$ | $0.9 \times 10^{-9}$ |
| $r_0$             | 0.44     |    | Initial Conditions |                        |                      |
| $r_1$             | 450.0    |    | $x_0$              | 10.0                   |                      |
| $d_1$             | 1000.0   |    | $a_0$              | 2.0                    |                      |
| $a_1$             | 1497.691 |    | $c_0^*$            | 0.0                    |                      |
| $\ell_1 + \ell_2$ | 0.13308  |    | $c_{E0}$           | $0.2 \times 10^{-8}$   | $0.5 \times 10^{-7}$ |
| $k_2$             | 0.005    |    |                    |                        |                      |

Figure 3.5.2 compares the stressed and unstressed behavior of the resource-consumer component. We see that the exposed population starts to decline even when bioavailable resource can support a higher population level. Consequently, the decline in the exposed population level cannot be attributed to overcrowding and/or competition for the

resource. This decline in the exposed population level must be a result of toxicant stress.

In Figure 3.5.2, it is shown that the ultimate unexposed population level is higher than the ultimate exposed population level. Moreover, more resource is needed to maintain a lower exposed population level, while less resource is needed to maintain a higher unexposed population level. This last statement seems surprising since we would expect higher population levels to consume more resource than lower population levels. But, if we consider the fact that the presence of a toxicant in the resource and in the environment results in stressed resource-population subsystem, then deviation of the resource-consumer component from an accepted mode of operation or function is to be expected. The above observation indicates the manner by which the resource-consumer subsystem deviates from an accepted standard mode of operation. The significance and possible biological explanation of this observed phenomenon are presented in the next section where we also show that this feature is common to all three resource-consumer-toxicant systems considered and that our analytic results support this observation.

Figure 3.5.3 illustrates the extinction behavior of the population component whose particular parameter values and initial conditions are contained in Table 3.5.1.II. Note that increasing the exogenous toxicant input by an order of magnitude results in extinction of the population.

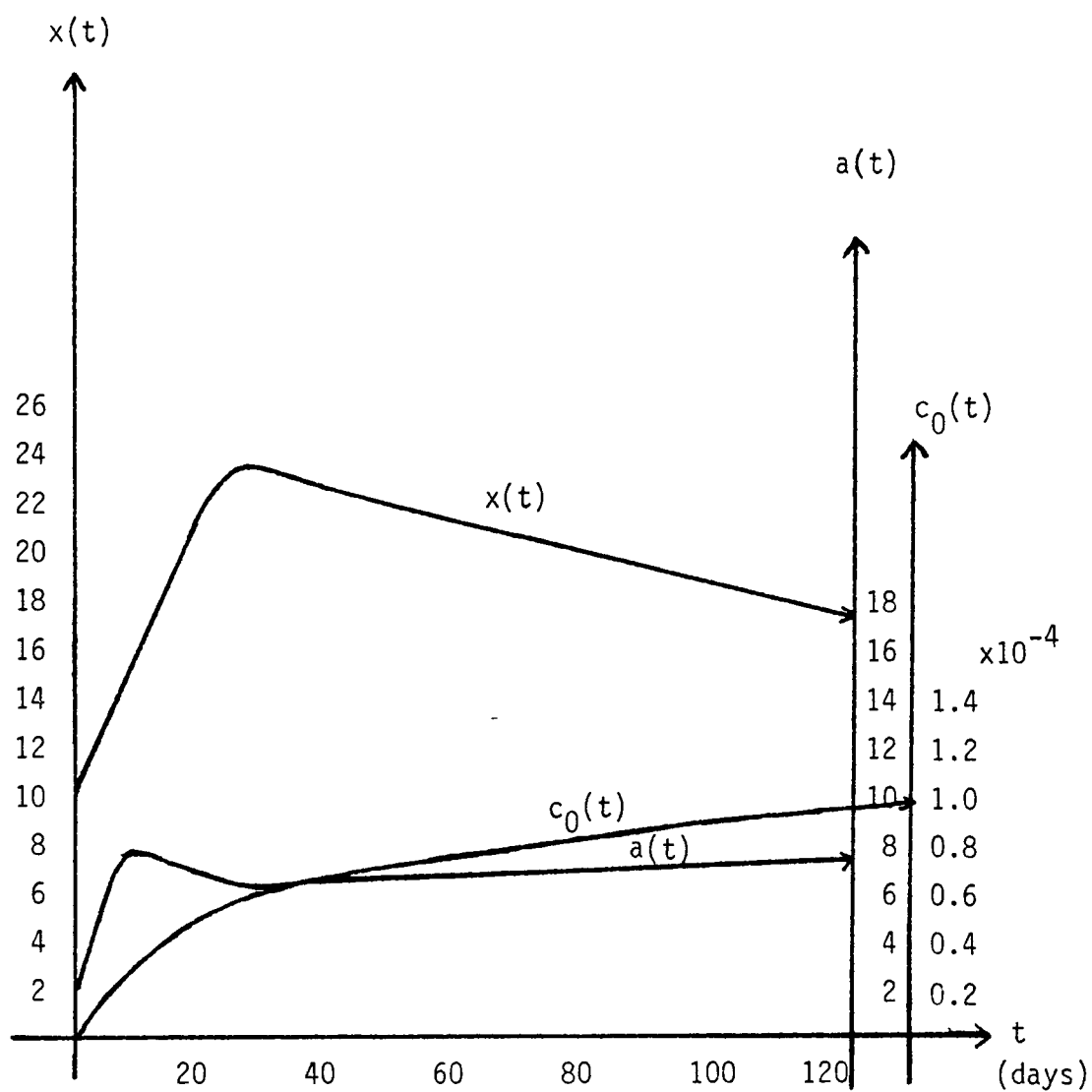


FIGURE 3.5.1. ASYMPTOTIC BEHAVIOR OF THE RESOURCE-CONSUMER COMPONENT OF THE GALLOPIN TYPE MODEL. PARAMETER VALUES AND INITIAL CONDITIONS USED ARE CONTAINED IN TABLE 3.5.2, I.

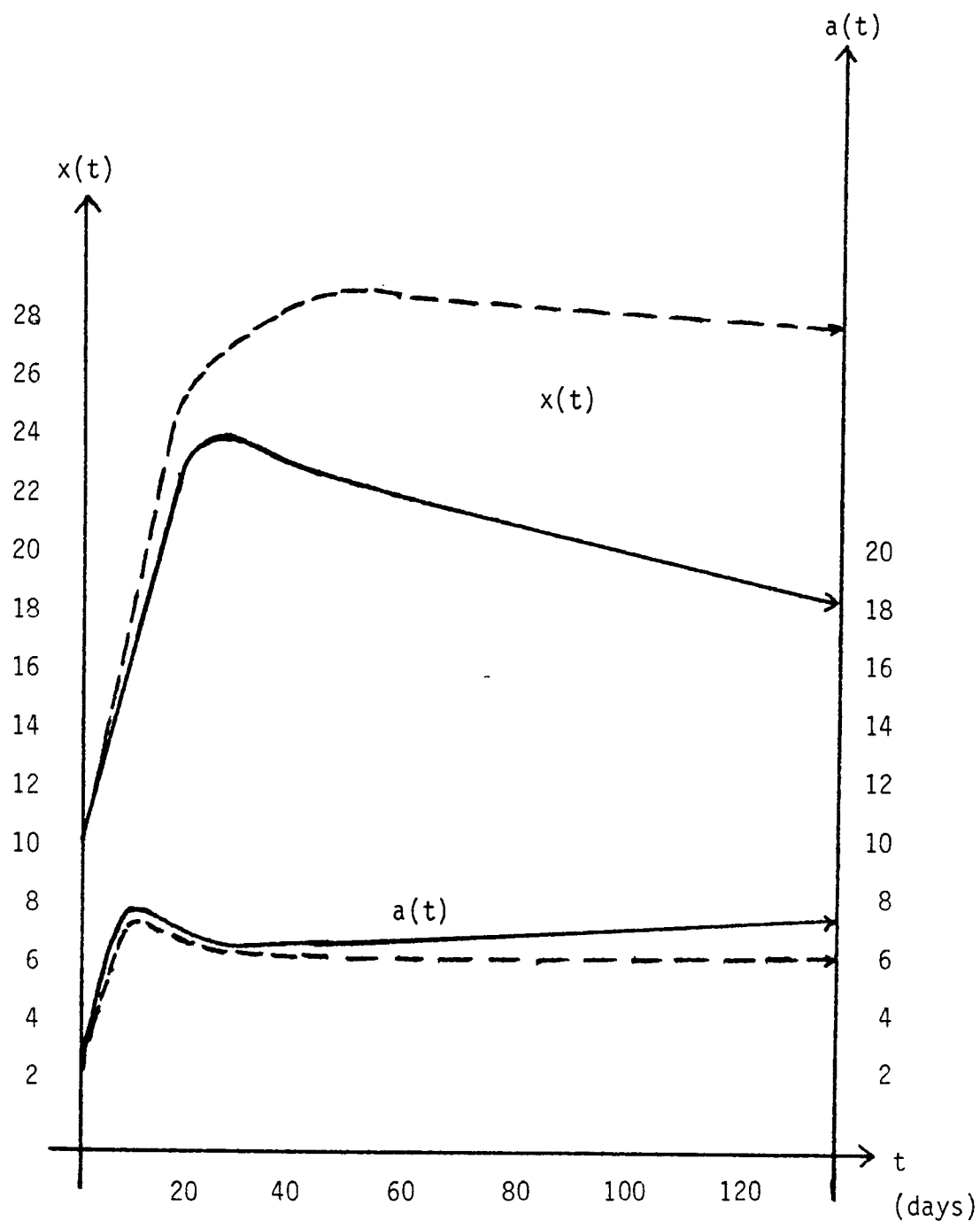


FIGURE 3.5.2. A COMPARISON OF THE RESOURCE-CONSUMER COMPONENT OF THE GALLOPIN TYPE MODEL. DASHED CURVES REPRESENT UNSTRESSED RESOURCE-CONSUMER SUBSYSTEM.

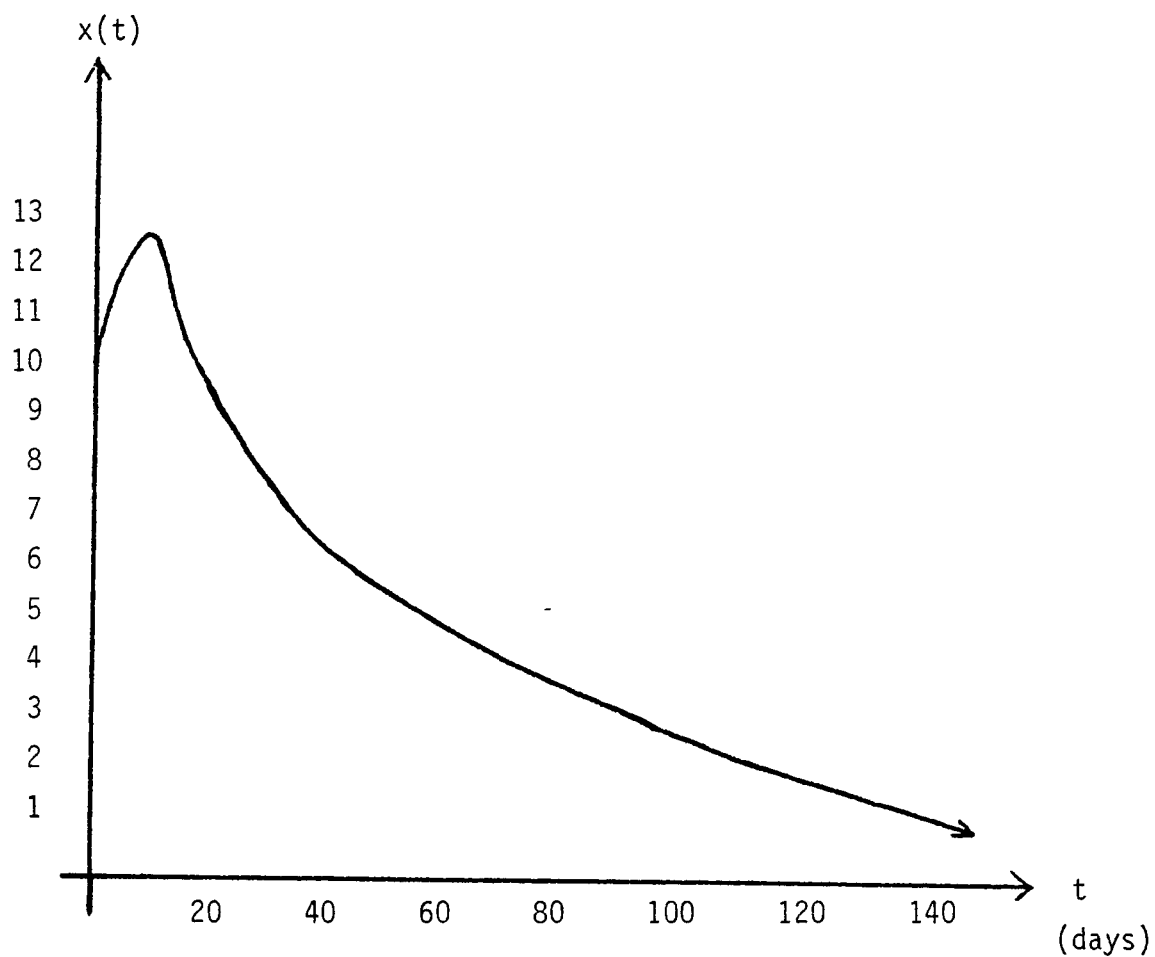


FIGURE 3.5.3. EXTINCTION BEHAVIOR OF THE POPULATION COMPONENT OF THE GALLOPIN TYPE MODEL WITH TOXICANT. PARAMETER VALUES AND INITIAL CONDITIONS USED ARE FOUND IN TABLE 3.5.1, II.

## (ii) Leslie Type Model

The parameter values used in the resource-consumer component of our Leslie type model are obtained from the same component of our Gallopin type model. Parameter values assigned to the toxicant components are determined as in Smith's model in Chapter 2. The solution to the pair of non-linear equations in  $a_1$  and  $l_1 + l_2$  is given by:

$$a_1 = 20.381\sqrt{d_1}, \quad l_1 + l_2 = 0.00182\sqrt{d_1}.$$

In particular, if  $d_1 = 100.0$ , then  $a_1 = 203.81$  and  $l_1 + l_2 = 0.0182$ .

The parameter values and initial conditions used in our computer simulation are summarized in Table 3.5.2.

Figure 3.5.4 shows the asymptotic behavior of the resource-consumer component. The asymptotic behavior of the toxicant component is no longer shown since it is similar to the toxicant component behavior in Gallopin's model. Figure 3.5.5 compares the stressed and unstressed resource-consumer component while Figure 3.5.6 illustrates the extinction behavior of the population component. Parameter values and initial conditions used in Figure 3.5.6 are found in Table 3.5.2, II.

It is significant to note the similarity in the asymptotic behavior of the resource-consumer components of both the Gallopin type and the Leslie type models. In both models, the population level rises above its steady state value; then declines to its equilibrium level when resource consumption and toxicant stress becomes important.

TABLE 3.5.2.

A SUMMARY OF PARAMETER VALUES AND INITIAL CONDITIONS USED  
IN THE LESLIE TYPE MODEL. NON-BLANK ENTRIES IN  
COLUMN II REPRESENT CHANGES IN PARAMETER VALUES  
AND/OR INITIAL CONDITIONS IN COLUMN 1.

| PARAMETERS              | I                    | II | PARAMETERS | I                     | II                    |
|-------------------------|----------------------|----|------------|-----------------------|-----------------------|
| $r_0$                   | 0.44                 |    | $k_2$      | 0.005                 |                       |
| $r_1$                   | 450.00               |    | $f_\infty$ | 10.0                  |                       |
| $\alpha$                | 0.2                  |    | $g_\infty$ | $0.7 \times 10^{-10}$ | $0.80 \times 10^{-9}$ |
| $\gamma$                | 0.6                  |    |            | Initial Conditions    |                       |
| $a_1$                   | 203.81               |    | $x_0$      | 3.0                   |                       |
| $d_1$                   | 100.0                |    | $a_0$      | 2.0                   |                       |
| $\theta$                | $4.2 \times 10^{-6}$ |    | $c_0^*$    | 0.0                   |                       |
| $\beta$                 | 0.1                  |    | $c$        | $0.2 \times 10^{-8}$  | $0.50 \times 10^{-9}$ |
| $\lambda_1 + \lambda_2$ | 0.0182               |    |            |                       |                       |

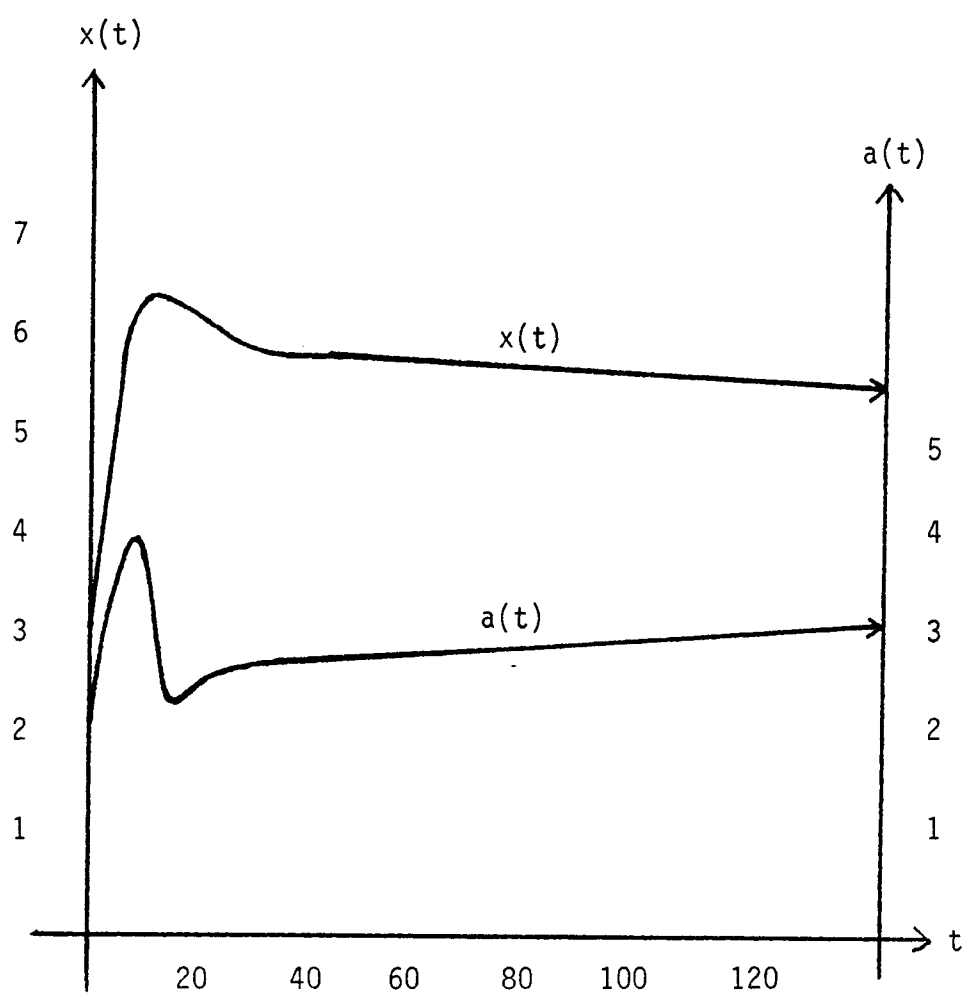


FIGURE 3.5.4. BEHAVIOR OF THE RESOURCE-CONSUMER COMPONENT OF THE LESLIE TYPE MODEL. PARAMETER VALUES AND INITIAL CONDITIONS USED ARE CONTAINED IN TABLE 3.5.2, I.

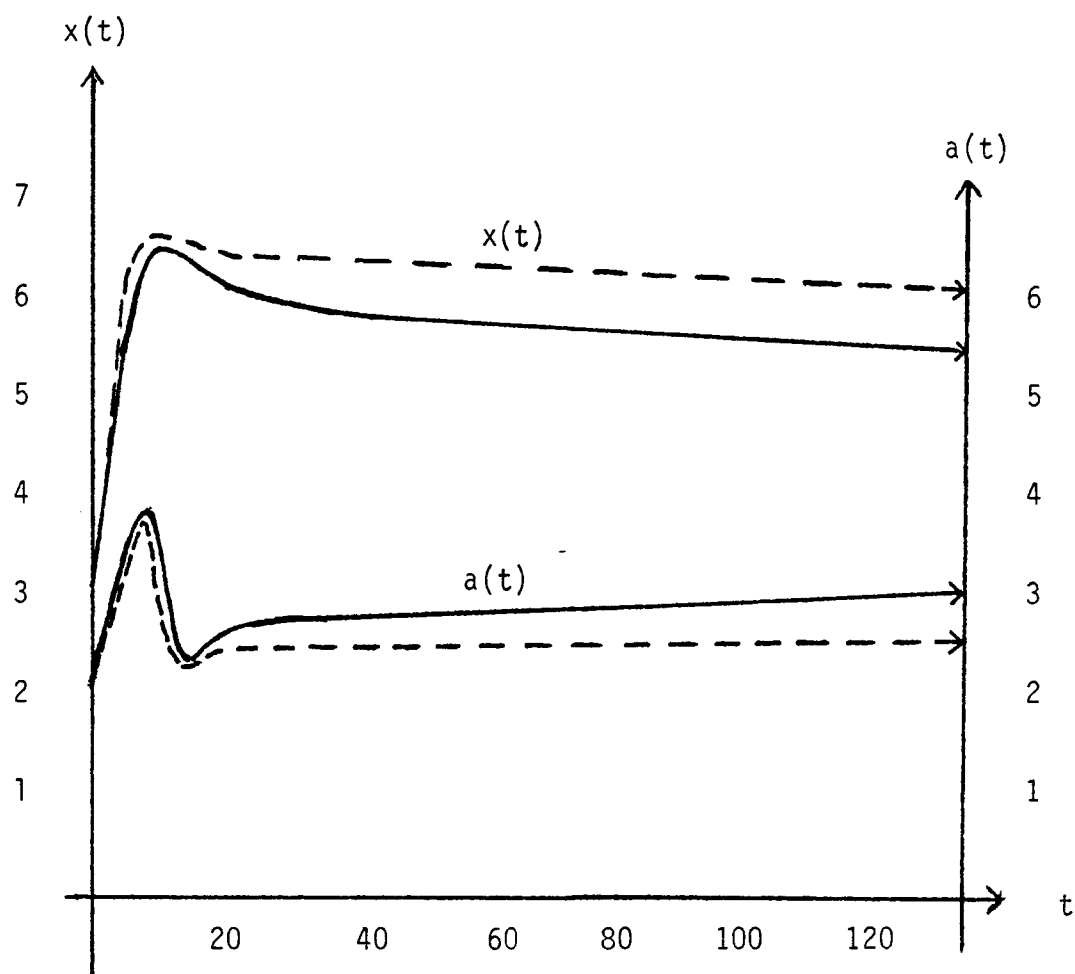


FIGURE 3.5.5. A COMPARISON OF THE RESOURCE-CONSUMER COMPONENT OF THE LESLIE TYPE MODEL. DASHED CURVES REPRESENT UNSTRESSED RESOURCE-CONSUMER SYSTEM. PARAMETER VALUES AND INITIAL CONDITIONS USED ARE CONTAINED IN TABLE 3.5.2, I.

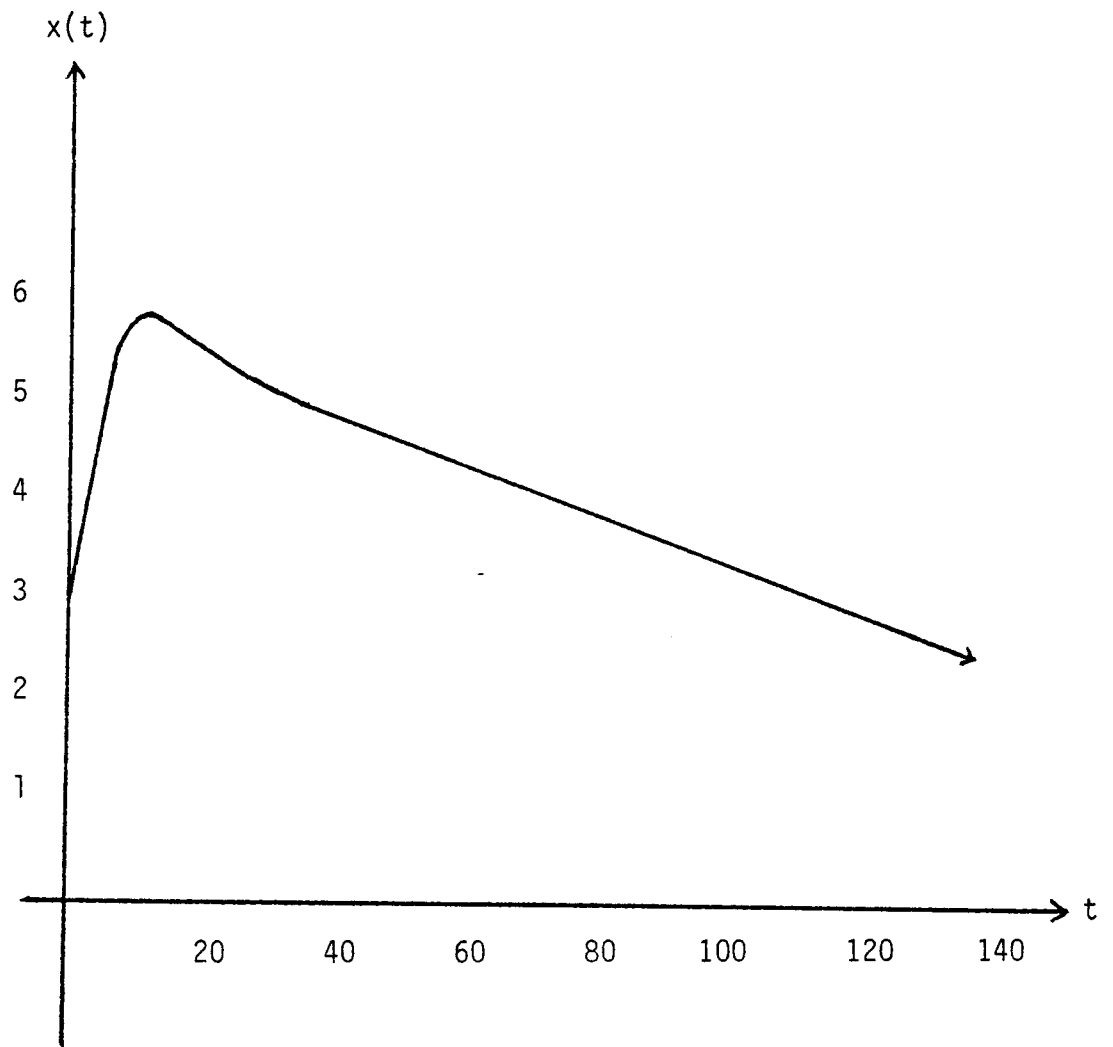


FIGURE 3.5.6. EXTINCTION BEHAVIOR OF THE POPULATION COMPONENT OF THE LESLIE TYPE MODEL. PARAMETER VALUES AND INITIAL CONDITIONS USED ARE CONTAINED IN TABLE 3.5.2, II.

In the Leslie type model however, the stressed population behaves in much the same way as the unstressed population during the early period when resource is abundant and when the body burden is low (see Figure 3.5.5). Such is not the case in the Gallopin type model (see Figure 3.5.2).

### (iii) Continuous Culture Type Model

A summary of parameter values and initial conditions used is shown in Table 3.5.3. Numerical values assigned to parameters in the resource-consumer component are obtained from Herbert et al., (1956) and Holmberg (1981). The parameter values in Table 3.5.3, I, satisfy the inequality

$$\mu_m - D > r_1 c_0^{(\infty)} \quad \text{and} \quad s_R > \frac{K[r_1 c_0^{(\infty)} + D]}{\mu_m - r_1 c_0^{(\infty)} - D},$$

that is, the population survives. The same set of parameter values, but with the exogenous toxicant input increased to  $c_R = 9.28 \times 10^{-10}$  (column II), results in the reversed inequality  $\mu_m - D < r_1 c_0^{(\infty)}$ , that is, the population is driven to extinction.

Simulation results are shown in Figures 3.5.7 through 3.5.9. Note that the overshoot behavior is common to all models considered. Figure 3.5.9 illustrates the extinction behavior of the population component as well as the asymptotic behavior of the resource. In the proof of Theorem 3.4.2, we observed that  $\lim_{t \rightarrow \infty} x(t) = 0$  if and only if  $\lim_{t \rightarrow \infty} s(t) = s_R$ . Our simulation results (Figure 3.5.9) verifies this observation.

TABLE 3.5.3.

A SUMMARY OF PARAMETER VALUES AND INITIAL CONDITIONS  
 USED IN THE CONTINUOUS CULTURE TYPE MODEL.  
 BLANK ENTRIES IN COLUMN II MEAN THE SAME  
 PARAMETER VALUES AS IN COLUMN I.

| PARAMETERS | I                    | II | PARAMETERS         | I                     | II                    |
|------------|----------------------|----|--------------------|-----------------------|-----------------------|
| $\mu_m$    | 0.8                  |    | $\ell_1 + \ell_2$  | 0.0892                |                       |
| K          | 3.0                  |    | $s_R$              | 5.0                   |                       |
| $r_1$      | 330.0                |    | $c_R$              | $1.28 \times 10^{-8}$ | $9.28 \times 10^{-8}$ |
| D          | 0.2                  |    | INITIAL CONDITIONS |                       |                       |
| y          | 0.5                  |    | $x_0$              | 0.3                   |                       |
| $a_1$      | 902.0                |    | $s_0$              | 0.2                   |                       |
| $d_1$      | 300.0                |    | $c_0^*$            | 0.0                   |                       |
| $c_1$      | $4.2 \times 10^{-6}$ |    | $c_{E0}$           | $0.5 \times 10^{-8}$  |                       |

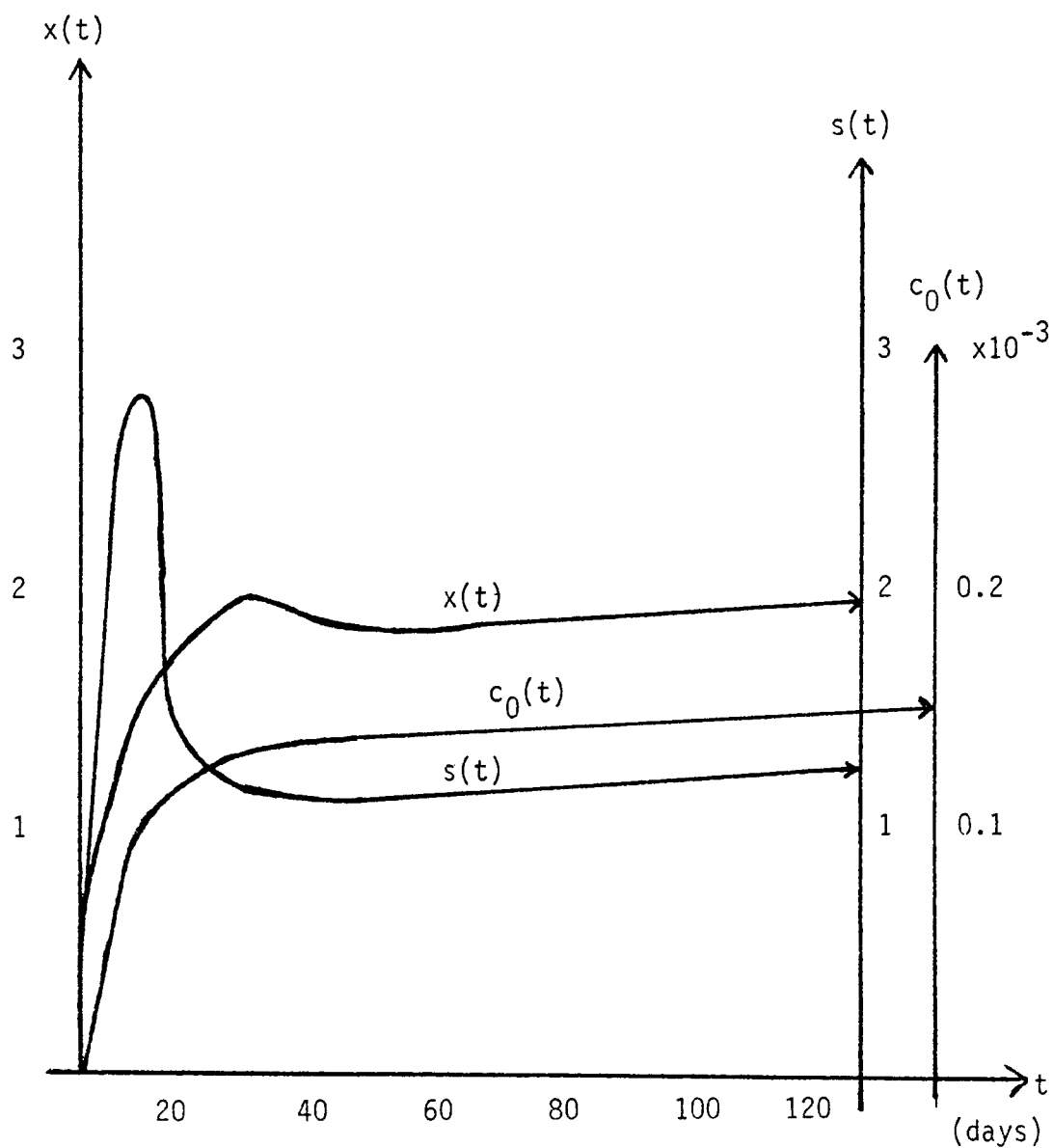


FIGURE 3.5.7. ASYMPTOTIC BEHAVIOR OF THE RESOURCE-CONSUMER COMPONENT OF THE CONTINUOUS CULTURE TYPE MODEL WITH TOXICANT. PARAMETER VALUES AND INITIAL CONDITIONS USED ARE FOUND IN TABLE 3.5.3, I.

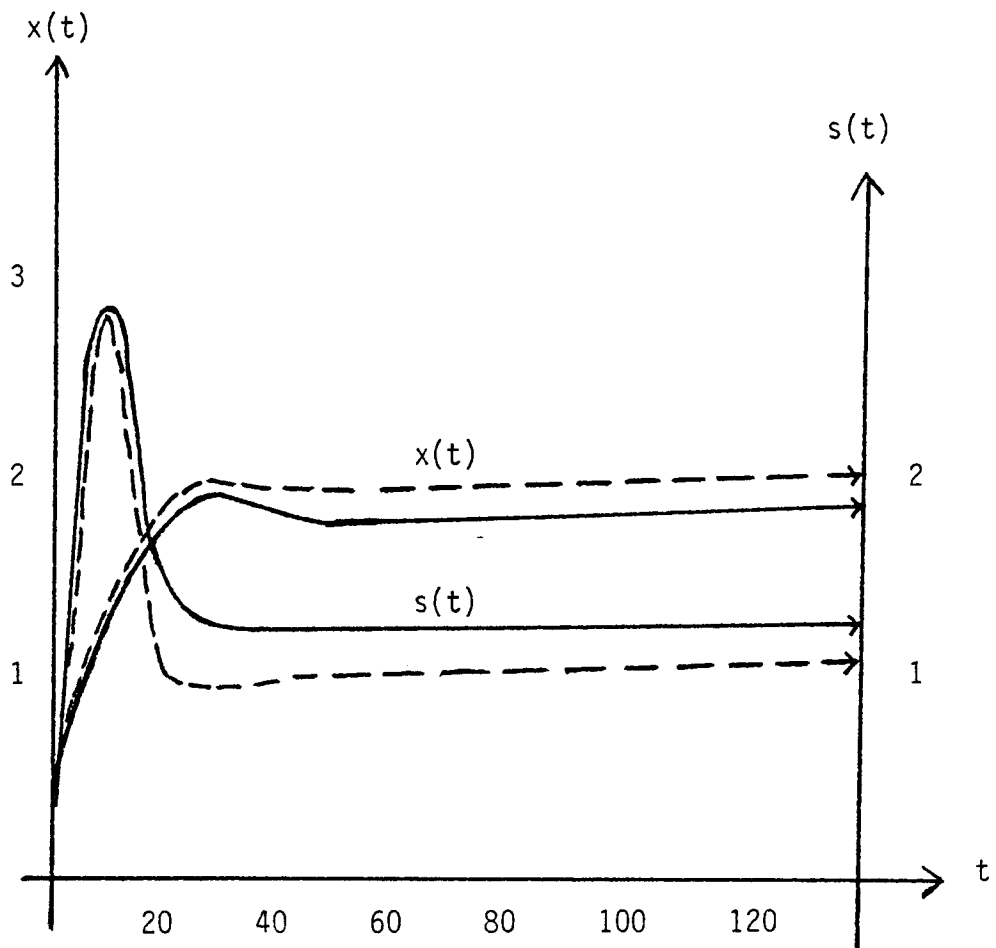


FIGURE 3.5.8. COMPARISON OF THE ASYMPTOTIC BEHAVIOR OF THE RESOURCE-CONSUMER COMPONENTS OF THE CONTINUOUS CULTURE TYPE MODEL WITH AND WITHOUT TOXICANT. DASHED CURVES REPRESENT RESOURCE-CONSUMER COMPONENT WITHOUT TOXICANT.

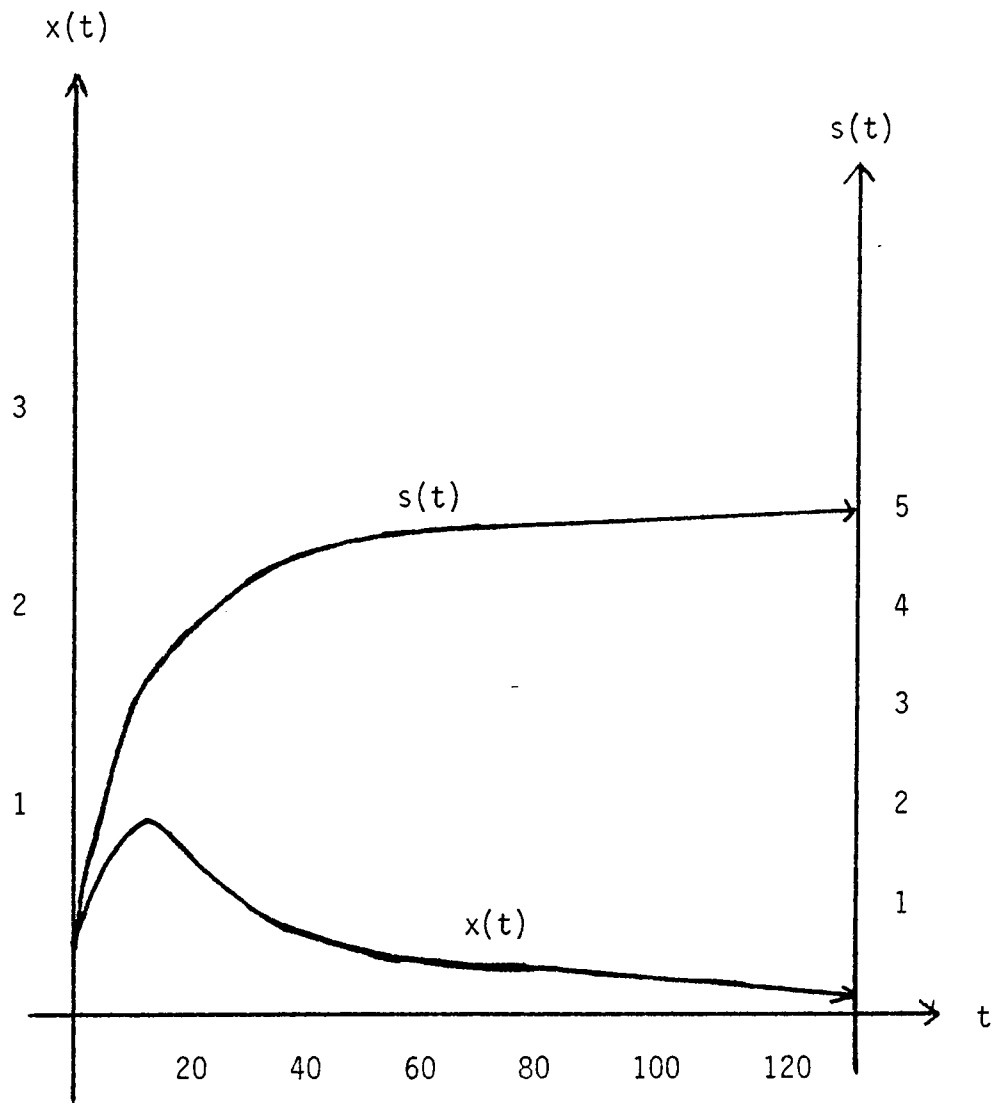


FIGURE 3.5.9. EXTINCTION BEHAVIOR OF THE POPULATION COMPONENT OF THE CONTINUOUS CULTURE TYPE MODEL WITH TOXICANT. PARAMETER VALUES ARE CONTAINED IN TABLE 3.5.3, II.

### 3.6. Discussion

The three resource-consumer-toxicant systems studied here have modelling features that are common and some that are different. But as earlier noted in our simulation results, there is an interesting phenomenon related to ultimate levels of population and resource under stressed and unstressed conditions exhibited by all of the models.

Tables 3.6.1 through 3.6.3 compare equilibria of toxicant stressed and unstressed population and resource models. By comparing each of these situations, we conclude that with toxicant present in the system, higher levels of resource are required to support a lower population level; that is, the equilibrium value of the population with toxicant is less than the  $x$ -equilibrium without toxicant, while the inequality is reversed for the resource equilibrium values. Our numerical results explicitly illustrate this conclusion. This phenomenon suggests that for a stressed population to survive, it must not only consume resource for maintenance and growth but it must also consume to offset losses due to toxicant stress. Thus, the presence of toxicant in these resource-consumer models could be interpreted as resulting in higher maintenance costs and/or lower growth rates. In each case, we have measured the population losses by  $r_1 c_0(\infty)$ . If this loss due to toxicant is high, the ultimate population level is small, but the amount of resource needed to support this population is relatively large.

An attempt will now be made to explain the above mentioned phenomenon. The first part, i.e., lower ultimate population level for

stressed population as compared to unstressed population level, may be explained by the fact that a toxicant increases the death rate of a population. It is well known that the greater the rate at which organisms are removed from the population, the smaller the standing crop the population is capable of maintaining (Slobodkin, 1960).

TABLE 3.6.1  
EQUILIBRIUM VALUES FOR THE LESLIE-TYPE MODEL  
WITH AND WITHOUT TOXICANT PRESENT

|             | NO TOXICANT STRESS                                                | TOXICANT STRESS                                                                       |
|-------------|-------------------------------------------------------------------|---------------------------------------------------------------------------------------|
| $x(\infty)$ | $\left[ \frac{r_0 f_\infty}{\alpha \gamma} \right]^{\frac{1}{2}}$ | $\left[ \frac{f_\infty (r_0 - r_1 c_0(\infty))}{\alpha \gamma} \right]^{\frac{1}{2}}$ |
| $a(\infty)$ | $\left[ \frac{\alpha f_\infty}{\gamma r_0} \right]^{\frac{1}{2}}$ | $\left[ \frac{f_\infty}{\gamma (r_0 - r_1 c_0(\infty))} \right]^{\frac{1}{2}}$        |

TABLE 3.6.2  
EQUILIBRIUM VALUES FOR THE GALLOPIN-TYPE MODEL  
WITH AND WITHOUT TOXICANT PRESENT.

|             | NO TOXICANT STRESS                                                         | TOXICANT STRESS                                                                              |
|-------------|----------------------------------------------------------------------------|----------------------------------------------------------------------------------------------|
| $x(\infty)$ | $\frac{\beta_1 f_\infty}{\alpha_2 \beta_1 - r_0}$                          | $\frac{\beta_1 f_\infty}{\alpha_2 \beta_1 - r_0 + r_1 c_0(\infty)}$                          |
| $a(\infty)$ | $\frac{-x(\infty)}{\alpha_1} \ln\left(\frac{r_0}{\alpha_1 \beta_2}\right)$ | $\frac{-x(\infty)}{\alpha_1} \ln\left[\frac{r_0 - r_1 c_0(\infty)}{\alpha_1 \beta_2}\right]$ |

TABLE 3.6.3.

EQUILIBRIUM VALUES FOR THE CONTINUOUS CULTURE-TYPE  
MODEL WITH AND WITHOUT TOXICANT PRESENT.

|             | NO TOXICANT STRESS              | TOXICANT STRESS                                                     |
|-------------|---------------------------------|---------------------------------------------------------------------|
| $x(\infty)$ | $y[s_R - \frac{KD}{\mu_m - D}]$ | $y[s_R - \frac{K[r_1c_0(\infty) + D]}{\mu_m - r_1c_0(\infty) - D}]$ |
| $s(\infty)$ | $\frac{KD}{\mu_m - D}$          | $\frac{K[r_1c_0(\infty) + D]}{\mu_m - r_1c_0(\infty) - D}$          |

The second part, i.e., relatively large amount of resource needed to support a lower population, is more difficult to justify. For a possible biological explanation, we consider the effects of exposure to toxicant on the energy budget of the population. To do this, we adopt L.B. Slobodkin's (1960) results on population energetics. Since we are concerned with a resource-consumer system at steady state, we limit our discussion of energy budget to the case where the rate of energy accumulation is zero.

Slobodkin has argued that the steady state can be characterized by the fact that it requires energy for its maintenance. Expenditure of potential energy is necessary for

- (i) Movement of the organism's internal parts,
- (ii) Movement of itself in the environment,
- (iii) Production of new protoplasm to compensate for attrition of its own body,
- (iv) Adding new protoplasm to its own body,
- (v) Production of new offspring.

Slobodkin's concern, however, is different from ours. He looks at the concept of efficiency, energy, and entropy as they apply to ecology. On the level of the individual, he limits his discussion only to the last two of the above-listed activities and largely ignores the energy requirements of the first three activities.

In a population-toxicant interaction, however, we feel that the energy requirements of producing new protoplasm to compensate for bodily attrition, i.e., activity (iii), is important. One only has to note that individual organisms, unable to repair toxicant-related injuries, are subjected to premature death.

Let  $I$  be the potential energy ingested per day of a population and  $B$ , the caloric content of the standing crop of a population in the absence of a toxicant;  $P$  represents the potential energy contained in the living protoplasm of the population. In order to maintain living protoplasm, energy must be expended. The greater the energy expenditure  $C$  per calorie of standing crop per time, the smaller will be the value of  $P$  that can be maintained by a given energy income  $I$  per day.

When a population is exposed to a toxicant, it either becomes extinct, or, according to our resource-consumer-toxicant models, it can come to a new steady state population level and consequently, to a new steady state caloric content  $P'$ . Since the new steady state population level is lower than when there was no toxicant, we must have  $P' < P$ .

The energy budget of a population is the sum of the energy requirements for maintenance and growth (Smith, 1963). Since our

population is at steady state, the energy budget for growth is at the zero level. Energy for maintenance is generally used for repair of damages brought about by the usual wear and tear of daily living, for individual growth, and for production of new offsprings needed to replace those organisms that are about to die or are already dead.

Note that when a population is chronically exposed to a toxicant, a new type of maintenance problem comes into existence. Whereas most causes of physical injury, such as fighting, can be eliminated and healing of physical damages could go on unhindered, chronic exposure to toxicant remains as an injury-causing-agent and interrupts any healing activities in an organism. Hence, the organism must continually nourish the injury and must be able to build new tissues at a rate at least as fast as the toxicant kills the target tissues. This continuous tissue-building activity is no doubt energy expensive. The organism must therefore consume more energy to meet this non-ending tissue-building requirement. On top of this, additional energy is needed to metabolize the toxicant in the organism's body. However, in spite of this high level of energy consumption, there is no increase in the amount of the organism's living protoplasm, and so its caloric content is not increased. Thus, the organism's energy expenditure per calorie of standing crop,  $C'$ , is much higher than  $C$ . Much of  $C'$  is used in overcoming the deleterious effects of the toxicants and in metabolizing toxicant in its tissues. This energy expenditure does not result in added protoplasm, and hence may be considered as wasted energy. Consequently, chronic exposure to toxicant of a resource-consumer system in which the population survives results in greater

energy expenditure per calorie of standing crop,  $C'$  ; yet the caloric content of the standing crop  $P'$  is less than  $P$  , the caloric content of the standing crop of the population in the absence of a toxicant, i.e.,

$$C' > C \quad \text{and} \quad P' < P .$$

In terms of resource and population biomass the above inequality may be interpreted as meaning that higher levels of resource are required to support a lower population biomass.

## CHAPTER 4

## PREDATOR-PREY-TOXICANT MODEL

## 4.1. Introduction

It was stated in Chapter 3 that a resource-consumer system may contain some of the complex process in a predator-prey system. In this chapter, we will formulate a predator-prey-toxicant system under conditions similar to those in Chapter 3 and demonstrate that the resulting system can also exhibit the phenomenon observed in our resource-consumer-toxicant system. A modified Lotka-Volterra type predator-prey system is sufficient for this purpose. Following our modelling approach, we will incorporate changes into our basic model and analyze the resulting system. The reduced model is again employed as the concentration dynamics of the predator-prey-toxicant system.

## 4.2. Model Formulation

The Lotka-Volterra type predator-prey system we want to consider consists of the pair of differential equations

$$\frac{dx}{dt} = x[\alpha s - d_0] , \quad (4.2.1)$$

$$\frac{ds}{dt} = s[R_0 - \beta s - \gamma x] \quad (4.2.2)$$

where  $x = x(t)$  is the biomass of the predator population,  $s = s(t)$  is the biomass of the prey population,  $\alpha$  is the birth rate per predator per unit prey consumed,  $d_0$  is the intrinsic death rate

of the predator population,  $R_0$  is the intrinsic growth rate of the prey population,  $\beta$  is the intraspecific coefficient of competition, and  $\gamma$  is the feeding rate per predator per unit prey consumed. Note that this system can be characterized by observing the following:

- (i) The prey population behaves according to the logistic dynamics if left alone. Its specific growth rate is diminished by an amount proportional to the predator density.
- (ii) In the absence of prey, the predator will die out exponentially, but its growth rate is enhanced by an amount proportional to the prey density.

Except for the fact that the prey species behaves according to the logistic dynamics, (i) and (ii) above are the characteristics of a classical Lotka-Volterra model for predator-prey interactions.

In Chapter 3 it is assumed that the resource is relatively unaffected by the toxicant in spite of toxicant accumulation. The parallel situation would be that the prey organisms accumulate toxicants, but are relatively immune to its toxic effects. Thus, we need to assume that the prey population takes up toxic chemicals and goes on living without apparent harm or injury. It is well known in the literature that this situation does occur in nature. Moreover, this apparent immunity from toxic chemical effects can be explained by the science of selective toxicity.

Adrien (1968) defines selective toxicity as the injury of one kind of living matter without harming some other kind with which the first is in intimate contact. The organism that is injured is referred

to as the uneconomic species and the immune organism, as the economic species. There are three mechanisms by which selective toxicity operates, namely (Adrien, 1968),

- (i) The toxicant is accumulated by the uneconomic species;
- (ii) The toxicant is accumulated by both species but the toxicant reacts exclusively with a cytological feature present only in the uneconomic species;
- (iii) The toxicant is accumulated by both species and the toxicant injures a chemical system important for the uneconomic, but not for the economic species.

Let us now assume that at least one of mechanisms (ii) and (iii) is selecting in favor of the prey species, i.e., the prey population is an economic species. Using once more the linear dose-response function without threshold given by (2.4.2), we obtain our predator-prey-toxicant system (P) consisting of the following set of differential equations:

$$\frac{dx}{dt} = x[\alpha s - r_1 c_0 - d_0] \quad (4.2.3)$$

$$\frac{ds}{dt} = s[R_0 - \beta s - \gamma x] \quad (4.2.4)$$

$$\frac{dc_0}{dt} = a_1 c_E + \frac{d_1 \gamma c_1 s}{a_1} - (\ell_1 + \ell_2) c_0 \quad (4.2.5)$$

$$\frac{dc_E}{dt} = g - k_2 c_E . \quad (4.2.6)$$

with initial conditions

$$x(0) = x_0 > 0, s(0) = s_0 > 0, c_0(0) = 0, c_E(0) = c_{E0} > 0.$$

where  $C_1$  is the body burden of an average individual of the prey species.

### 4.3. Asymptotic Analysis of Model (P)

THEOREM 4.3.1. (Extinction Theorem) Assume  $\lim_{t \rightarrow \infty} g(t) = g_\infty$  exists. If  $\lim_{t \rightarrow \infty} s(t) = s(\infty)$  exists and if

$$s(\infty) < \frac{r_1 c_0(\infty) + d_0}{\alpha},$$

then all components have limits and

- (i)  $\lim_{t \rightarrow \infty} c_E(t) = c_E(\infty) = g_\infty/k_2$
- (ii)  $\lim_{t \rightarrow \infty} c_0(t) = c_0(\infty) = \frac{a_1 c_E(\infty) + \frac{d_1 \gamma C_1 s(\infty)}{a_1}}{l_1 + l_2}$
- (iii)  $\lim_{t \rightarrow \infty} x(t) = x(\infty) = 0$
- (iv)  $\lim_{t \rightarrow \infty} s(t) = s(\infty) = R_0/\beta.$

This equilibrium solution is locally asymptotically stable.

PROOF. It is convenient to consider the integral representation of each of the state variables. Equation (4.2.3) is linear in the variable  $x$ ; hence we can write

$$x(t) = x_0 e^{\int_0^t [s(t_1) - r_1 c_0(t_1) - d_0] dt_1} \quad (4.3.1)$$

Equation (4.2.4), on the other hand, is an equation of the Bernoulli type; hence we can write

$$s(t) = \frac{s_0 e^{\int_0^t [R_0 - x(t_1)] dt_1}}{1 + \beta s_0 \int_0^t e^{\int_0^{t_2} [r_0 - x(t_2)] dt_2} dt_1} \quad (4.3.2)$$

Since the last two equations of Model (P) are linear equations, we have

$$c_0(t) = e^{-(\ell_1 + \ell_2)t} \int_0^t \left[ a_1 c_E(t_1) + \frac{d_1 \gamma c_0 s(t_1)}{a_1} \right] \cdot e^{(\ell_1 + \ell_2)t_1} dt_1 \quad (4.3.3)$$

and

$$c_E(t) = e^{-k_2 t} \left[ c_{E0} + \int_0^t g(t_1) e^{k_2 t_1} dt_1 \right] \quad (4.3.4)$$

An application of L'Hopital's Rule to (4.3.3) and (4.3.4) yields

$$\lim_{t \rightarrow \infty} c_0(t) = \lim_{t \rightarrow \infty} \frac{a_1 c_E(t) + \frac{d_1 \gamma c_1 s(t)}{a_1}}{\ell_1 + \ell_2} \quad (4.3.5)$$

and

$$\lim_{t \rightarrow \infty} c_E(t) = \lim_{t \rightarrow \infty} \frac{g(t)}{k_2} . \quad (4.3.6)$$

Since both  $\lim_{t \rightarrow \infty} g(t) = g_\infty$  and  $\lim_{t \rightarrow \infty} s(t) = s(\infty)$  exists by hypothesis, then the limits in (i) and (ii) follow from (4.3.6) and (4.3.5), respectively.

The limit in (iii) follows from (4.3.1) and from the hypothesis that  $s(\infty) < (r_1 c_0(\infty) + d_0)/\alpha$ . Since  $\lim_{t \rightarrow \infty} x(t) = 0$ , then an application of L'Hopital's Rule in (4.3.2) establishes the limit in (iv).

The local asymptotic stability is established by a proof similar to the asymptotic stability proof of Theorem 3.3.2.

THEOREM 4.3.2. (Persistence Theorem) Assume  $\lim_{t \rightarrow \infty} g(t) = g_\infty$

and  $\lim_{t \rightarrow \infty} s(t) > 0$  exists. If

$$\frac{R_0}{\beta} > \frac{r_1 c_0(\infty) + d_0}{\alpha} ,$$

then a feasible equilibrium exists with components

$$\begin{aligned} \text{(i)} \quad \lim_{t \rightarrow \infty} s(t) &= s(\infty) = \frac{r_1 c_0(\infty) + d_0}{\alpha} \\ \text{(ii)} \quad \lim_{t \rightarrow \infty} x(t) &= x(\infty) = \frac{R_0 - \beta s(\infty)}{\gamma} \\ \text{(iii)} \quad \lim_{t \rightarrow \infty} c_0(t) &= c_0(\infty) = \frac{a_1 c_E(\infty) + \frac{d_1 \gamma c_1(\infty)}{a_1}}{\lambda_1 + \lambda_2} \\ \text{(iv)} \quad \lim_{t \rightarrow \infty} c_E(t) &= c_E(\infty) = g_\infty / k_2 . \end{aligned}$$

Moreover, this feasible equilibrium solution is locally asymptotically stable.

PROOF. We first observe from (4.3.5) that since  $\lim_{t \rightarrow \infty} s(t)$  exists, then  $\lim_{t \rightarrow \infty} c_0(t)$  exists. We next claim that the integrand

$\alpha s(t) - r_1 c_0(t) - d_0$  of (4.3.1) is in  $L^1(\mathbb{R}_+)$ . For if we assume for the purpose of contradiction that  $\alpha s(t) - r_1 c_0(t) - d_0$  is not in  $L^1(\mathbb{R}_+)$ , then since  $\alpha s(t) - r_1 c_0(t) - d_0$  has a limit, either

$I = \int_0^\infty [\alpha s(t_1) - r_1 c_0(t_1) - d_0] dt_1$  is  $\infty$  or  $-\infty$ . If  $I$  is  $\infty$ ,

then from (4.3.1), we have  $\lim_{t \rightarrow \infty} x(t) = \infty$ . But from (4.3.2), since

$$s(t) \leq e^{\int_0^t [R_0 - \gamma x(s)] ds},$$

then  $\lim_{t \rightarrow \infty} s(t) = 0$  which contradicts our hypothesis on  $s(\infty)$ . If

$I$  is  $-\infty$ , then from (4.3.1)  $\lim_{t \rightarrow \infty} x(t) = 0$ . An application of

L'Hopital's Rule in (4.3.2) yields  $\lim_{t \rightarrow \infty} s(t) = R_0/\beta$ . Since by

hypothesis

$$\frac{r_1 c_0(\infty) + d_0}{\alpha} < \frac{R_0}{\beta} = s(\infty),$$

it follows from (4.3.1) that  $\lim_{t \rightarrow \infty} x(t) = \infty$ , which again is a

contradiction. Thus,  $\alpha s(t) - r_1 c_0(t) - d_0$  is in  $L^1(\mathbb{R}_+)$ . By

an argument similar to those in the proof of Theorem 3.4.2, it

follows that  $\lim_{t \rightarrow \infty} [\alpha s(t) - r_1 c_0(t) - d_0] = 0$ , or equivalently

$$\lim_{t \rightarrow \infty} s(t) = \frac{r_1 c_0^{(\infty)} + d_0}{\alpha} .$$

The limit in (i) is now established.

To show the limit in (ii), note that from (4.2.4), since

$$\lim_{t \rightarrow \infty} s(t) > 0 , \text{ then } \lim_{t \rightarrow \infty} [R_0 - s(t) - x(t)] = 0 , \text{ or equivalently}$$

$$\lim_{t \rightarrow \infty} x(t) = \frac{R_0 - \beta s^{(\infty)}}{\gamma} .$$

Since

$$s^{(\infty)} = \frac{r_1 c_0^{(\infty)} + d_0}{\alpha} < \frac{R_0}{\beta} ,$$

$$\text{then } \lim_{t \rightarrow \infty} x(t) > 0 .$$

The limits in (iii) and (iv) follow from (4.3.5) and (4.3.6) respectively.

Local asymptotic stability is established by using Hurwitz's criteria and a proof similar to the asymptotic stability proof of Theorem 3.3.2.

REMARKS. From Theorems 4.3.1 and 4.3.2, note that the ultimate prey population satisfies the inequality

$$s^{(\infty)} \leq \frac{r_1 c_0^{(\infty)} + d_0}{\alpha} .$$

We now show that the case

$$s^{(\infty)} > \frac{r_1 c_0^{(\infty)} + d_0}{\alpha} \tag{4.3.7}$$

cannot occur. For if (4.3.7) holds, then it follows from (4.3.1) that  $\lim_{t \rightarrow \infty} x(t) = \infty$ . But from (4.3.2), since we can write

$$s(t) \leq e^{\int_0^t [R_0 - \gamma x(t_1)] dt_1},$$

then  $\lim_{t \rightarrow \infty} s(t) = 0$ , which is a contradiction.

#### 4.4. Discussion and Comments

Table 4.4.1 below is similar to those found in Chapter 3. It compares the equilibrium values of toxicant-stressed and unstressed predator-prey levels.

TABLE 4.4.1.

EQUILIBRIUM VALUES FOR THE LOTKA-VOLTERRA  
TYPE PREDATOR-PREY MODEL WITH AND WITHOUT  
TOXICANT STRESS.

|             | NO TOXICANT STRESS                                             | TOXICANT STRESS                                                                  |
|-------------|----------------------------------------------------------------|----------------------------------------------------------------------------------|
| $x(\infty)$ | $\frac{R_0 - \beta \left[ \frac{d_0}{\alpha} \right]}{\gamma}$ | $\frac{R_0 - \beta \left[ \frac{d_0 + r_1 c_0(\infty)}{\alpha} \right]}{\gamma}$ |
| $s(\infty)$ | $\frac{d_0}{\alpha}$                                           | $\frac{d_0 + r_1 c_0(\infty)}{\alpha}$                                           |

We see in the above table that under the hypothesis of selective toxicity, where the prey population is an economic species, the

phenomenon discussed in our resource-consumer-toxicant models is again demonstrated. In order to survive, the stressed predator population must consume more prey to meet the requirements for maintenance and growth, as well as to offset losses due to toxicant stress. Toxicant in the system again results in higher maintenance costs and lower growth rates.

Let us examine in greater detail the equilibrium values of the predator-prey components in Theorem 4.3.1. Substitution and algebraic manipulation of the limits yield

$$s(\infty) = \frac{d_0 + \frac{r_1 a_1 g_\infty}{k_2(\ell_1 + \ell_2)}}{\alpha - \frac{r_1 d_1 \gamma C_1}{a_1(\ell_1 + \ell_2)}} \quad , \quad (4.4.1)$$

$$\begin{aligned} x(\infty) = & \{R_0 k_2 [\alpha a_1(\ell_1 + \ell_2) - r_1 d_1 \gamma C_1] \\ & - \beta a_1 [d_0 k_2(\ell_1 + \ell_2) + r_1 a_1 g_\infty]\} / \\ & / \{k_2 \gamma [a_1 \alpha(\ell_1 + \ell_2) - r_1 d_1 \gamma C_1]\} \quad . \end{aligned} \quad (4.4.2)$$

Note from (4.4.1) that  $\lim_{t \rightarrow \infty} s(t) > 0$  if and only if

$$\alpha > \frac{r_1 d_1 \gamma C_1}{a_1(\ell_1 + \ell_2)} \quad (4.4.3)$$

From (4.4.2),  $\lim_{t \rightarrow \infty} x(t) > 0$  if and only if (4.4.3) holds and

$$R_0 k_2 [\alpha a_1 (\ell_1 + \ell_2) - r_1 d_1 \gamma C_1] \\ > a_1 \beta [d_0 k_2 (\ell_1 + \ell_2) + r_1 a_1 g_\infty] ,$$

or equivalently,

$$R_0 k_2 \left[ \alpha - \frac{r_1 d_1 \gamma C_1}{a_1 (\ell_1 + \ell_2)} \right] > \beta \left[ d_0 k_2 + \frac{r_1 a_1 g_\infty}{(\ell_1 + \ell_2)} \right] . \quad (4.4.4)$$

An examination of (4.4.3) and (4.4.4) shows that the expression

$$\alpha - \frac{r_1 d_1 \gamma C_1}{a_1 (\ell_1 + \ell_2)} \quad (4.4.5)$$

is an important parameter in the sense that it determines the existence of a feasible equilibrium. We recall that  $\alpha$  is the birth rate per predator per unit prey consumed, that is, the assimilation rate. It is a measure of the rate at which each unit of prey eaten is converted to new predators. The expression

$$\frac{r_1 d_1 \gamma C_1}{a_1 (\ell_1 + \ell_2)}$$

may be interpreted as the amount by which the assimilation rate is diminished due to toxicant coming from feeding on contaminated prey. Thus, (4.4.5) is the new assimilation rate of the predator population.

Inequality (4.4.4) sums up the requirements for survival of the predator population. It is first of all required that (4.4.3) is satisfied. This simply means that there must be a net gain from

predation in spite of predation-toxicant related losses. Inequality (4.4.4) requires that the net gain from predation must exceed losses due to natural causes and losses due to exposure to toxicant in the environment.

Let us rewrite inequality (4.4.4) in the form

$$\alpha > \frac{r_1 d_1 \gamma C_1}{a_1(\ell_1 + \ell_2)} + \frac{\beta d_0}{R_0} + \frac{\beta r_1 a_1 g_\infty}{R_0 k_2(\ell_1 + \ell_2)} \quad (4.4.6)$$

Note that the left hand side of (4.4.6) is the sum of all the loss terms. In particular, the terms

$$\frac{r_1 d_1 \gamma C_1}{a_1(\ell_1 + \ell_2)} \quad \text{and} \quad \frac{r_1 a_1 g_\infty}{R_0 k_2(\ell_1 + \ell_2)}$$

represent rate of loss due to toxicant from predation and from environmental exposure, respectively. It is clear from (4.4.1) and (4.4.2) that the bigger the toxicant related loss terms are, the bigger is  $s(\infty)$  but the smaller is  $x(\infty)$ . Thus, the higher the loss due to toxicant are, the higher is the ultimate prey population level, but the lower is the ultimate predator population level. This last statement could be interpreted as another form of Volterra's Principle.

#### 4.5. Summary and Conclusion

The series of workshops conducted by the Environmental Sciences Division (ESD), Oak Ridge National Laboratory (ORNL) and sponsored by the Office of Toxic Substances, U.S. Environmental Protection Agency

(EPA) (Hammon, 1981) called for the development of mathematical models as a tool for assessing the risk of exposure to harmful chemicals. But ecotoxicological modelling is still too far behind to meet the need for techniques for evaluating the effects of toxic chemicals on the biota. This dissertation is therefore an attempt to make a contribution to this important aspect of ecotoxicological modelling study. Mathematical models that include fate of chemical substances and biological processes directly affected by the toxic materials are formulated and analyzed.

In formulating mathematical models useful in predicting fate and effects of chemicals on natural systems, we start from the hypothesis that the dynamics of the population or of the population-resource system are known before toxic chemicals are introduced into the system. We assume that the population accumulates toxic substances mainly from its diet and from its environment. Appropriate modifications are then introduced on the population dynamics to account for stress effects resulting from bioaccumulation of a toxic chemical.

The process of toxicant uptake from the food and from the environment is still not well understood. In fact, the relative contribution of the environment and of the resource into the body burden of an organism, as well as the question of whether or not these two sources are additive, are the subject of considerable scientific debate. It has been suggested that dietary contribution to the total equilibrium body burden is insignificant, in which case, equilibrium body burden from environmental uptake alone is a precise estimate of the maximum bioaccumulation of a toxic chemical. But as shown in Chapter 2,

Section 1.1, a number of experimental results do not support this argument. Although there are no available explanations as to why dietary contribution in some chemicals is insignificant, while for other toxicant substances the food chain input is important, this aspect of chemical bioaccumulation has been incorporated in our models. We assume that there is a coupling between the uptake rates of different toxicant pathways. In particular, we assume that this feedback between the toxicant accumulation sources depends reciprocally upon the environmental uptake parameter  $a_1$ .

The toxicant dynamics are formulated using the mass balance equation. This results in first order kinetics for the body burden  $c_0$  and environmental concentration  $c_E$ . The population dynamics and the body burden dynamics are coupled through a linear dose-response function without threshold. The resulting systems are analyzed to assess the impact of toxicants on the biota. Our main mathematical results consist of formulating criteria for persistence and extinction. These criteria are intricately related to the exogenous toxicant input  $g$  in Chapter 2, and to both the toxicant input  $g$  and resource input  $f$  in Chapter 3.

A reduced model is introduced and it is argued that the reduced model is an adequate, environmentally sound predictor of threshold values of toxicant concentration as well as persistence level of the population. Numerical solutions are generated to illustrate these conclusions.

The three resource-consumer-toxicant systems studied in Chapter 3 exhibit an interesting behavior related to ultimate levels of

population and resource under stressed and unstressed conditions. We observe that with toxicant present in the system, higher levels of resource are required to support a lower population level. A possible biological explanation is advanced based on population energetics.

In formulating our Leslie-type resource-consumer subsystem, we incorporate some features of a predator-prey system. Using selective toxicity as an hypothesis, where the prey population is an economic species, we have shown in Chapter 4 that a Lotka-Volterra type predator-prey system does exhibit the phenomenon we observe in our resource-consumer-toxicant systems.

Possible improvements and research activities that could enhance the usefulness of our mathematical models can be undertaken. Further research is needed to understand the process of toxicant uptake from the food and from the environment. The problem here is to identify the mechanism that determines additivity or non-additivity of toxicant accumulation from the environment and food pathways. A possible mechanism to investigate is the partitioning of toxicant in an organism's body, i.e., lipids, kidneys, liver, etc..

A possible modification that could make our models useful is to make the toxicant concentration in the resource,  $\theta$ , time dependent. In general, the resource of an organism is also subject to time-varying environmental factors such as pH, temperature, environmental toxicant concentration, solar radiation, etc.; hence  $\theta$  need not be constant. Depending on one's modelling perspective,  $\theta$  may be viewed as an exogenous input to the system, or  $\theta$  can be introduced as another state variable with its own dynamic representation.

Our assumption on the parameter  $\beta$  (the average rate of food intake per unit organismal mass) may limit the applicability of our models to lower trophic levels. In general, the food intake of an organism may depend on its age and/or its size. For lower trophic levels, the average ration size may not differ much between organisms of different age and size. But for higher trophic levels, the average ration size for a younger, smaller organism may be very different from the average ration size for an older, bigger organism. Hence, the average ration size for an average member of the population may be too large or too small for individuals in a particular age or size class. Thus, in order for our models to dynamically represent higher trophic levels,  $\beta$  should be size and/or age dependent. For multi-compartment systems, our models with constant values for  $\beta$  and  $\theta$  may still be used to represent the dynamics of a particular age or size class.

This paper is a response to the need for the development of mathematical models that can predict biological effects of chemical stress. The results of this study indicate that the presence of a toxicant in the resource and in the environment leads to higher maintenance costs and/or lower growth rates.

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