Mathematical models of the physiological mechanisms affecting the adaptation of growing cattle during and after a period of undernutrition.

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Abstract

Grazing animals in the semi-arid tropics are subjected to short or long periods of moderate to severe undernutrition. Many simulation models were developed to represent the mechanisms of ruminant adaptation. These mechanisms include, among others, the differential mobilization of tissues, the recycling of nitrogen to the rumen via the saliva and across the rumen wall, and the relation between intake and animal size. However, most simulation models have attempted to represent the mechanisms for above-maintenance nutritional restrictions.

In this study, a simulation model, a linked rumen and intermediary metabolism model (RUMET), is developed to simulate rumen function and nutrient utilization during continuous growth, undernutrition (submaintenance) and realimentation for growing cattle. The model will represent two mechanisms of adaptation: the differential mobilization of tissues and the recycling of nitrogen to the rumen via the saliva and across the rumen wall. The model consists of a system of ordinary differential equations that simulates the supply of nitrogen and energy to intermediary metabolism and the changes in the weights of ash (bone), muscle, adipose tissue, liver and small intestine and the relative effects of these changes on the maintenance expenditure.

The model shows that during nutritional restriction, the visceral organs (in particular, the small intestine and liver) exhibit quick responses to undernutrition whereas the peripheral tissues (for example the muscle) show a lag in its response to undernutrition. After a nutritional restriction, the enhanced food intake in relation to size and the reduced maintenance expenditure enable the animal to use proportionately more nutrients for growth. The model may be used to predict energy expenditure, liveweight changes and the probability of survival during periods of undernutrition. Voluntary intake, liveweight gains and changes in body composition may be predicted for animals given diets of different composition during and following a period of undernutrition.

The ability to predict the probable short- and long-term consequences of undernutrition will support the planning and evaluation of measures to ameliorate undernutrition on semi-arid and arid rangeland systems.

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Most importantly, I would like to thank my parents who encouraged and supported me despite our very humble beginnings. I especially want to dedicate this thesis to my father who died while I was doing the research for this thesis. Also, I would like to thank Melanie who stood by me and encouraged me unconditionally throughout this thesis.

PART I

INTRODUCTION

Chapter 1

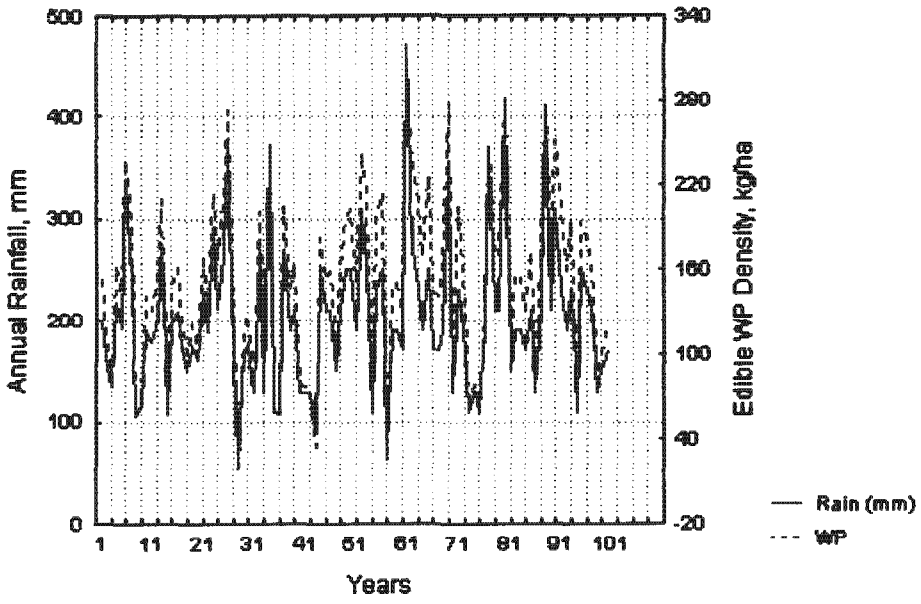
The Undernutrition Problem

1.1 Challenges for managing arid and semi-arid rangeland

The world's steadily expanding population as well as the economic improvements among developing nations has intensified the demand for food, especially animal protein, and made it imperative that food production (including livestock products) be increased. Arid and semi-arid lands cover about one-third of the earth's land surface and nearly two-thirds of the African continent. The majority of African livestock and possibly 30 million livestock-dependent people reside in these zones (Ellis and Swift, 1988). In contrast to intensive systems of animal production which require intensive arable crops and artificial pastures for their support, livestock in arid and semi-arid regions subsist on natural grazing as rainfall is inadequate to support reliable rainfed crop production.

On the semi-arid and arid rangeland, rainfall is seasonal and varies from year to year (for example, see Figure 1.1a,b) and is therefore the driving variable for animal performance in these complex ecosystems. Herbage availability varies with the variation in rainfall (Figure 1.2) and also on soil type and the presence of bush (Dye and Spear, 1982). In addition to herbage quantity, the nutritive value of herbage (nitrogen content and digestibility) varies with season and to a lesser extent between years; the fibre content is slightly lower and digestibility slightly higher in the dry years (Dye, 1984). The nitrogen content of the diet also affects the intake of the animal (Elliot, 1967).

As a consequence of the variation of herbage quantity and quality, grazing animals are subject to short- or long-term periods of moderate to severe undernutrition. Undernutrition in young ruminants affects the chances of their survival and the time required to reach the weight and physiological condition at which they are productive, that is, suitable for slaughter or able to work as draught animals or, if female, to conceive and rear offspring.



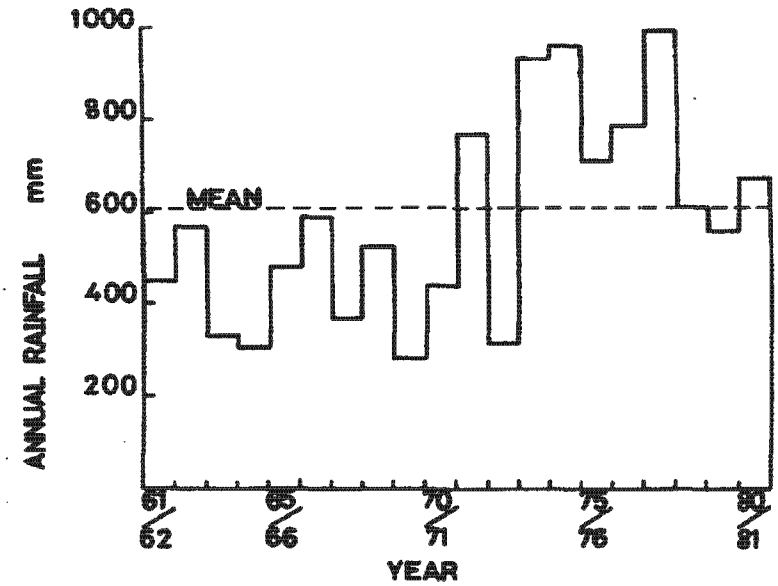


Figure 1.1: (a) Simulation model results showing the variation in rainfall and woody perennials (WP) at Paulshoek, Namaqualand, in South Africa for a 100 year period (Richardson *et al.*, in preparation), **(b)** Histogram of annual rainfall recorded at the Matopos Thornveld site in Zimbabwe (Dye and Spear, 1982).

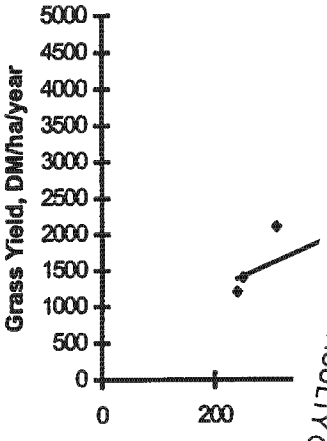


Figure 1.2: Effect of t lity at the Matopos Thornveld site in Zimb e shows the trend of the data)

1.2 The Undern

In both short- and l f an animal falls below its genetic potenti by O'Donovan, 1984) when food subsec celerate and exceed the growth rates of cc ge. However, the response of an animal to ue partly to the complex interactions of t metabolism. The response depends on the ge of the animal when the restriction was d during undernutrition and realimentation stricted animals increase their growth rate b s those animals not restricted. In some case a restricted animal once the restriction is ensation. For example, Allden (1970) four n 15 kg liveweight resulted in significantl The growth curve was displaced in time an

Animals are able to adapt to under- use the food needed to maintain a constant body weight is not only a function of weight but decreases with time in response to low feed intake (Ledger and Sayers, 1977). One of the factors that

contribute to compensatory growth is a reduced energy expenditure of animals during undernutrition and at the early stages of realimentation. This reduction in maintenance increases the energy available for growth and the extent of this contribution depends on the persistence of the reduced maintenance requirement during realimentation, the longer the reduction in maintenance persists the greater the contribution to compensatory growth. When food intake is restricted, the metabolically active tissues, such as the digestive tract and the liver, are likely to be reduced in size and activity (Taylor and Murray, 1991; Burrin *et al.*, 1990).

The management strategies to prevent undernutrition during the dry season have been either to reduce animal numbers per hectare (destocking) or to supplement the diet with protein or urea (nitrogen) during the dry season and nitrogen and energy during drought periods. Reducing the herd size is usually reserved for extended periods of severe forage shortages. A disadvantage of this strategy is that when range conditions are good the forage is often under-utilized, resulting in decreased productivity per hectare. However, during a drought, some pastoralists are reluctant to sell animals because of the belief that keeping their herd increases the probability of them being left with some animals after the drought period. A problem with keeping all the animals arises when there is high mortality of breeding and young females due to limited resources. After the drought these animals are extremely expensive because of the increased demand and a loss of breeding stock makes it difficult for a pastoralist to get back into the system. Therefore, livestock marketing in these environments must be responsive to highly variable levels of supply, both between years and between seasons. Garoian *et al.* (1990) stated that increased efficiency and reduced risk could possibly be achieved through smaller breeding herds and utilization of flexible marketing strategies. However, the production system model of Richardson and Hahn (1991) indicated that herd sizes do not necessarily have to be small. They emphasized the importance of flexible marketing strategies and the need for simulation models to assist in management decisions: for example, a decision has to be taken before the beginning of the dry season as to which animals must be sold.

1.3 How do animals adapt?

The aim of this thesis is not to attempt to understand the dynamics of an entire rangeland system but will address the mechanisms of adaptation of ruminants to submaintenance feeding on arid and semi-arid rangelands. In this section an outline of the current theories of mechanisms of animal adaptation will be described. In the next

section (section 1.4) a general description of managing arid and semi-arid rangeland systems will be outlined.

There are three questions which arise: firstly, what is the growth rate, body composition, including changes in the relative proportions of organs, and weight change of an animal undergoing different degrees of undernutrition during a period of nutritional limitation, secondly, what is the growth rate, body composition and weight change after this period of nutritional restriction when the animal is fed *ad libitum* (realimentation period) and, thirdly, at which body composition, given the various factors affecting compensatory growth (Table 1.1), does the animal survive?

Table 1.1: Factors affecting survival and compensatory growth (modified from Lawrence and Fowler, 1997).

Animal Factors
1. The degree of maturity at the start of undernutrition, that is, the proportion of expected normal mature mass already achieved.
2. The proportion of body weight attributable to adipose deposits at the start of undernutrition.
3. The genotype.
4. Changes in metabolic rate.
5. Changes in both energy and nitrogen metabolism.
Nutritional factors
1. The severity of the undernutrition, that is what fraction or multiple of the maintenance energy required is eaten on a mean daily basis.
2. The duration of the period of undernutrition.
3. The nutrient density of the food during undernutrition.
4. Food intake during rehabilitation.
5. Intake of rumen degradable protein.

To understand how animals adapt we will attempt to understand the exogenous and endogenous factors affecting undernutrition. There are three major causes of reduced growth rate which lead to the same end result. Two of these are the intake of sub-optimum amounts of good quality food and free access to food limited in one or more essential nutrients (O'Donovan, 1984). These causes of undernutrition can best be illustrated by an example from studies in a semi-arid area of Zimbabwe where the deficiencies of three major nutrients usually lead to sub-optimal production. Firstly, a

shortage of energy (food) occurs when the grass is sparse due to drought or overstocking or both. Secondly, during the dry season the protein content of available herbage is low and this limits animal intake and consequently, production, even if grass is plentiful. This is because ruminants eat small amounts of low protein roughages. However, the provision of small quantities of protein-rich concentrates or non-protein nitrogen, such as urea or biuret, increases the rate of digestion, stimulates dry matter intake and improves the animal's energy status (Elliot, 1967). Thirdly, a deficiency of phosphorus may occur throughout the year (Ward, 1968) and adversely affect performance since phosphorus plays an important role in energy metabolism. We assume for the purpose of this thesis that phosphorus is not deficient simply because phosphorus supplementation is relatively inexpensive and easy to administer.

Another factor which contributes to undernutrition can be explained in terms of the variation in animal maintenance energy. In order to understand this phenomenon, a scheme (Figure 1.3) is depicted of the partition of dietary energy as used by an animal. The utilization of ingested food energy (Gross Energy Intake, GE) by an animal involves several kinds of losses. Part of the GE is lost through excretion of energy in faeces, urine and combustible gases. The remaining part of the GE intake, the metabolizable energy (ME), is used firstly for the supply of the energy requirements for maintenance. The part of the ME used for maintenance (ME_m) is used to produce the ATP required for sustaining primary life processes and the heat increment of utilization for maintenance. This part of maintenance is fully dissipated as heat. If GE intake (Relative Feeding Level, RFL: RFL = 1 at maintenance) is higher than the maintenance requirement, the ME available for production (ME_g), is retained in the body as energy (Energy Retention, ER).

During growth, part of the available energy for production is lost as heat due to the inefficiency of use of ME. Thus, ER is the difference between ME and the total heat production. If GE intake is below the maintenance requirement, energy reserves from the body are mobilized (negative ER) to cover the deficit in ME for sustaining primary life processes. If an animal has no intake then it will still produce heat because the metabolic processes continue. This is known as fasting metabolism (F_m). Following McDonald *et al.* (1981),

$$ER = k_g\left[ME - \frac{F_m}{k_m}\right] \tag{1.1}$$

$$\text{where} \quad k_m = 0.503 + 0.35 \times q \tag{1.2}$$

$$\text{and} \quad k_g = 0.006 + 0.78 \times q \tag{1.3}$$

$$\text{and} \quad q = \frac{ME}{GE}. \tag{1.4}$$

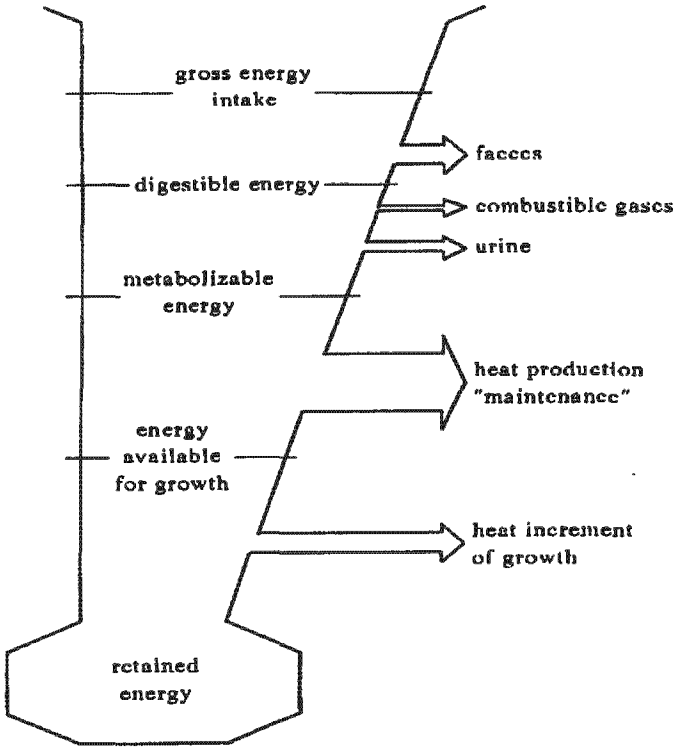


Figure 1.3: Partitioning of dietary energy (Young, 1975).

In most ME based models, the ME requirement of an animal is calculated as the sum of ME required for maintenance, growth, lactation and other activities. ME requirements for maintenance (ME_m) are usually calculated from body weight (W, kg) as $ME_m = a \times \frac{W^{0.75}}{k_m}$ where the coefficient, a, might range from 0.25 to 0.50 for animals on a low plane of nutrition and 0.50 for lactating animals and k_m (i.e., the efficiency of ME utilization for maintenance) might range from 0.65 to 0.75 depending on the ME content of the feed (Baldwin and Sainz, 1995). Similarly, the ME requirement of the gain (ME_g) would be calculated as $ME_g = \frac{RE}{k_g}$ where RE is the energy retained in the gain (MJ/kg) and k_g is the efficiency of ME utilization for gain. k_g might range from 0.33 to 0.65 depending on the relative content of fat and protein in the expected gain and ME content of the diet (Baldwin and Sainz, 1995).

These factors determine the nutrient requirements of an animal for unconstrained growth and accounts for the animal's potential growth in terms of both overall rate and composition. However, before the energy ingested can be used for the synthesis of new tissue, certain essential demands to maintain existing tissues must be met. This theory led to the development of feeding systems in which animal demands for maintenance are calculated separately from those for production. These systems have

assumed that maintenance costs are constant, relative to current animal weight, but this assumption is increasingly being questioned. Consequently, two main criticisms of the maintenance concept will be considered. The first is that models of ER assumes that ME_m is a function of weight only (in practice the basal energy costs are influenced by recent and current growth rates, that is, not just liveweight). The second group takes the same theme further and questions the validity of separating energy requirements for maintenance from those for growth (Gill and Oldham, 1993). Arguments relating to whether maintenance is solely a function of current state have been reviewed in detail by Turner and Taylor (1983).

Experimental findings show that animals either losing or maintaining liveweight over a considerable time, appear to have lower feed or energy requirements (Ledger and Sayers, 1977). Foot and Tulloh (1977) showed that the daily dry matter intake of the same diet by steers maintained at constant weight declined over time from 5.9 kg to 4.4 kg over a 100 day period. Also, Graham and Searle (1975) estimated that the energy requirements in lambs were reduced when they were given a constant feed intake. Some workers have less confidence in the validity of retaining the concept of an empirical value for maintenance (Milligan and Summers, 1986). They point out that the costs of some functions, which are normally accounted for as maintenance, vary continuously from low to high planes of nutrition and therefore the change in efficiency of ME utilization at zero energy balance is artificial. The adaptability of metabolism to stabilize at a new energy level is the basis of compensatory growth, which occurs after periods of undernutrition as the animal returns to its genetically determined growth pattern (O'Donovon, 1984).

The phenomenon whereby the animal increases its growth rate during realimentation is termed compensatory growth (Bohman, 1955). More factors assumed to affect compensatory growth are listed in Table 1.1 and explained in Lawrence and Fowler (1997).

1.4 Mathematical Models

The need to evaluate management strategies for rangelands resulted in the model of Jones and Sandland (1974) which was based on empirical relations between animal production and stocking rate. Subsequently, the influence of rainfall and stocking rate have been incorporated into the model (Danckwerts, 1984). There are, however, limitations to these models based on empirical relations in that they provide accurate predictions only for conditions closely similar to those under which the original data

was collected. A more flexible production model was developed by Richardson and Hahn (1991) to assist development agencies and agricultural planners to evaluate alternative production strategies (Figure 1.4). The management options include changing the stocking rate, milk off-take and sales policy. However, the equations of the animal submodel assumed that the composition of body weight change (protein content of gain, fat content of gain) of the animal is a function of the weight of the animal only.

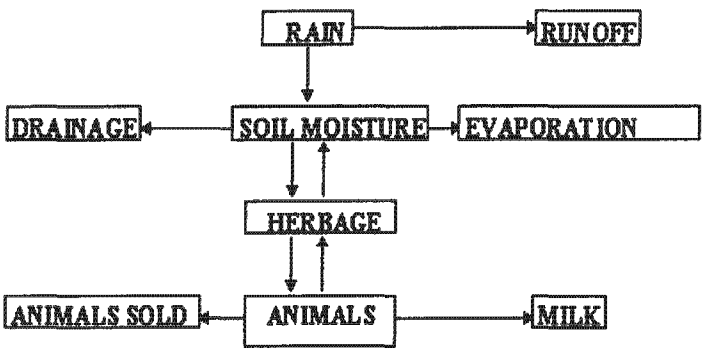


Figure 1.4: Model structure of a rangeland system (Richardson and Hahn, 1991).

In order to predict the response during and following a period of nutritional limitation, there is a need to develop a model to include more detailed components (Figure 1.5). The appropriate degree of detail with which to represent the animal in mathematical models of growth will depend on the objective of the model. Detailed mechanistic models of components of the system have largely contributed to our understanding of parts of the system but have not satisfied our aim to predict the outputs of the system. An example of such models would be the detailed model of the mammary gland by Freetly *et al.* (1993). The model gives us insight into the biochemical processes involved in the mammary gland but has not attempted to give ways of increasing the milk output relative to environmental and physiological conditions. Another example are the detailed intermediary metabolism models of France *et al.* (1987) and Gill *et al.* (1984). These models provide valuable information and aid our understanding of the partitioning of energy during growth of an animal but do not predict the effects of undernutrition and realimentation on body composition.

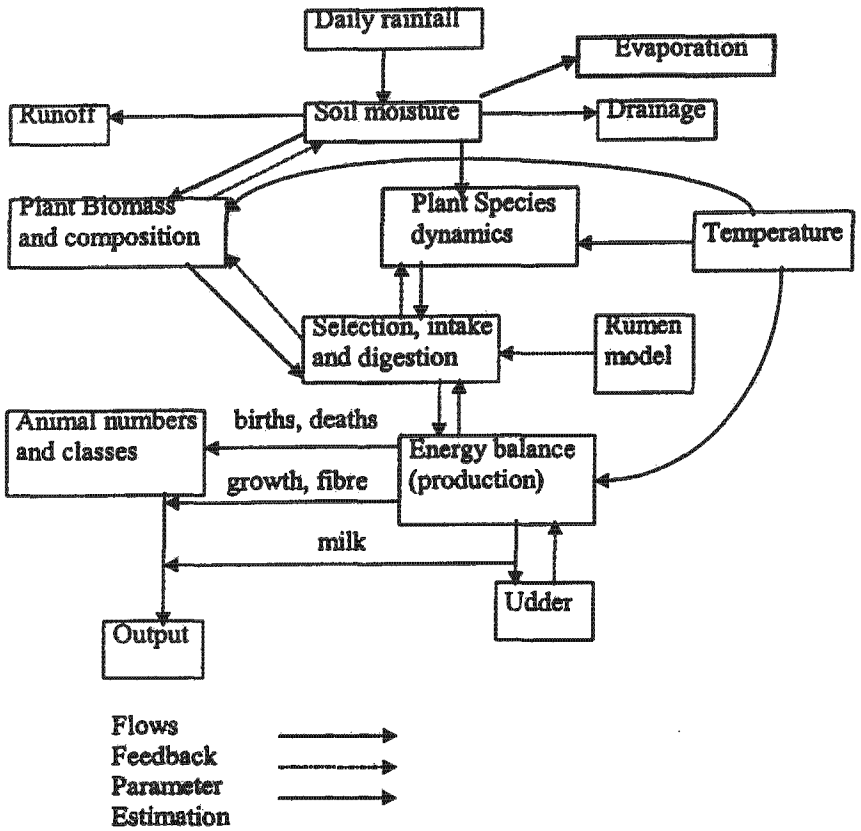


Figure 1.5: Components of a rangeland model (Richardson *et al.*, 2000).

Over the past decade there has been considerable progress in the modelling of both rumen and intermediary metabolism due to the increased availability of data and power of computers, but more importantly, due to the improved concepts that have been formulated as knowledge of the subject has increased. However, two problems still occur (Witten and Richardson, 2000):

1. Most mathematical models are developed to simulate the continuous growth process and do not adequately predict animal responses to limited (sub -maintenance) feeding.
2. Although suggested by France *et al.* (1987) that the modelling focus should also be on the integration of components to increase adequate predictions, the integration of models of the whole digestive process has been slow.

Therefore, the general aim of this thesis is to develop a mathematical model to simulate the effects of undernutrition and to account for some of the factors (such as relative organ weights) that influence maintenance energy requirements over time.

1.5 Managing arid and semi-arid rangelands under uncertainty

Conventional approaches (as outlined in Scoones, 1994) to managing arid and semi-arid rangeland systems will not be sufficient in providing effective solutions. A variety of problems are characterized by unpredictable conditions, potentially resulting in qualitative changes in system dynamics. Uncertainty poses a challenge to managing such systems and results in the need for a flexible and adaptive response (Behnke and Scoones, 1993). For example, on arid and semi-arid rangeland, rainfall, forage production and animal productivity all vary between years, between regions and within landscapes (Richardson *et al.*, 1997). Such systems are characterized by the heterogeneity of their components, which provides the variability on which selection can act, typically through non-linear interactions among those components. Consequently, these systems become hierarchically arranged into structures that are determined and reinforced by the flows and interactions among the parts (Levin, 1999).

In order to practically adopt a flexible management approach, it is required to understand the relation between the structure and functioning of an ecosystem and to understand the variation and interactions of processes; for example, patterns of nutrient flux, trophic structure, diversity-productivity relations, and how these processes feed back to influence the subsequent development of those processes and interactions between those processes. In addition, elucidating these processes and their interactions across temporal and spatial scales is fundamental in understanding ecosystem functioning (Levin, 1999). However, in general, it may be difficult to detect signals of change early enough to motivate effective solutions and such signals, even when detected, are likely to be displaced in time and space from the cause. For example, the variation and timing of the rain must be considered.

The initial goal of managing such systems in the past has been directed toward high short-term productivity at the expense of long-term sustainability of the system (Scoones, 1994). Developing sustainable approaches to system use implies understanding which processes, both across and within scales, determine what maintains system resilience (Holling, 1992) and how human interventions, for example, might affect it. By resilience we mean that a system tends to maintain its "integrity" when subject to disturbance (Holling, 1973). Even though natural ecosystems are complex and adaptive, heavily managed systems are not and are vulnerable to single perturbations, for example, pest outbreaks.

Ironically, the mechanisms that provide short-term resilience may also impose a rigidity of structure that erodes the capacity to respond to disturbances over long time scales. In addition, an important aspect is to elucidate the thresholds for the change of processes and their interactions across scales (Walker, 1993). For example, during a drought, one would like to know which animals would survive, the size and composition of the herd eventually affecting vegetation composition (Witten and Richardson, 2000).

1.6 Research motivation and thesis outline

In the previous sections the factors affecting the compensatory growth of cattle on arid and semi-arid rangelands and the possible role that mathematical models can play in integrating the components of the system have been outlined. Mathematical models can also assist in elucidating the thresholds for the change of processes, which is required to understand the mechanisms involved during undernutrition and realimentation of animals on arid and semi-arid rangeland. The objectives of this thesis are to develop dynamic mechanistic elements representative of the growth process that will enable (1) the simulation of the change of body weight and composition of an animal during and following a period of sub-maintenance feeding (2) the simulation of body composition changes of animals fed to maintain a constant body weight and (3) the understanding of the mechanisms involved during undernutrition and realimentation.

In Chapter 2 the theoretical background of models currently in use in modelling digestion and metabolism processes in ruminants will be described and a modelling framework from which to develop an integrated model to satisfy the modelling objectives will be presented. Chapter 3 describes the development and evaluation of a rumen model based on a rumen model developed by Dijkstra *et al.* (1996). In Chapter 4 a chemostat-type model of three aggregated functional classes of microbe species in the rumen is proposed. The aim of Chapter 4 is to understand the general qualitative behaviour of the model: the effects of changing substrate composition and quantity, the effect of nitrogen (ammonia) supplementation, and the effect of a periodic input of substrate on the abundance of microbial populations in the rumen. In Chapter 5 the mechanistic model of intermediary metabolism of France *et al.* (1987) is modified to simulate the effects that changes in relative feeding level (RFL) have on the weights of liver and small intestine. In Chapter 6, four mechanistic mathematical models, three of which are based on different assumptions of growth, are compared when simulating different feeding patterns. The objective of Chapter 7 is to modify the linked rumen

and metabolism models described in chapter 3 and chapter 5 to examine the recycling of nitrogen to the rumen via the saliva and diffusion across the rumen wall. Finally, Chapter 8 provides a general discussion of the results of this thesis and important implications for future research.

Chapter 2

The Modelling Approach

2.1 Introduction

The utilization of food by an animal depends on the quantity and quality of food ingested, and the subsequent processes of digestion, absorption and metabolism. In vivo studies are required to quantify nutrients absorbed, while the processes of metabolism have been studied through experiments conducted at the level of the whole animal, the tissue and the individual cell, with the aim of understanding component parts of the overall process (Gill *et al.*, 1989). As experimental techniques used by researchers and the quality and quantity of the data collected have improved, the desire to integrate this knowledge into a conceptual framework of the whole system has grown. In order to interrelate the component parts, an attractive framework exists via the development of mathematical models. However, the tremendous increase in the quality and quantity of data presents a challenge to anyone who wishes to develop a mathematical model of a given system. At what point does the model cease to have explanatory value, having become too complex to do anything more than to simulate a variety of parameters values and initial conditions for a system? In part, the answer to this question depends on the objectives of a particular modelling exercise. Models proposed often have many parameters and many of these parameters may not be known for the particular system studied. Furthermore, the complexity of the models makes it difficult to study sensitivity to parameters and initial conditions, even on fast computers. This difficulty is magnified when the systems, or parts thereof, that are simulated are inherently stochastic. In addition to the computational difficulties and the incomplete knowledge of parameters, there may be a lack of experimental data to evaluate model output of particular systems, for example, for animals feeding at or below maintenance.

Simply integrating the component parts of a particular system is a necessary condition to understand system behaviour but is not sufficient. One needs to consider the interactions among components and how they give rise to macroscopic system properties and how, in turn, these properties feed back to influence the subsequent development of those interactions. For example, nitrogen recycling from the blood plasma to the rumen may be an important mechanism to aid the ruminant to survive during periods of food shortages. Nitrogen is recycled to aid the growth of rumen

microorganisms, which are an essential source of nitrogen for the host animal. Also the system optimum is not the sum of the local optima. For example, manipulating rumen processess has attempted to optimize rumen output with little or no consideration of the needs of host animal metabolism (Øskov, 1977). The multiple time and spatial scales in biological systems forces one to, in general, focus on some particular level, often using heuristic approximations for the finer detail, which are neglected. Because one goal of this thesis is to predict body composition, there is a need to integrate models of rumen digestion and metabolism. The models developed in this thesis will provide such a framework where improvements to the models can be made within this framework.

2.2 Classification of Models

The following scheme has been proposed by France and Thornley (1984) for the classification of models:

Dynamic	or	Static
Deterministic	or	Stochastic
Mechanistic	or	Empirical.

Even though this terminology was adopted by the animal science modelling community, there remain ambiguities which will be discussed later. An empirical equation is one that has been fitted to experimental data in order to describe a relation which has actually been observed between two or more variables (Riggs, 1963). Such equations imply nothing about the underlying reason for the relation and thus, although a particular set of data may be faithfully described, it cannot be safely applied beyond the range of values for independent variables upon which the model is based (Baldwin, 1995). Many models currently in use in animal science are empirical (including most of the models that form the basis of feeding standards) and these models must be applied with care to ensure that recommendations are not made for conditions where the data used in formulating the equations do not apply.

In contrast, mechanistic models are derived from theory or hypothesis and formulated on the basis of concepts regarding underlying functions in an attempt to account for variations in experimental data. France and Thornley (1984) also presented the following formulation of the organizational hierarchy of biological systems as related to levels of aggregation, the formulation of models and further definition of the terms "empirical" and "mechanistic":

Level	Description of the Level
$i+1$	herd of animals
i	animal
$i-1$	organs
$i-2$	tissues
$i-3$	cells

According to the hierarchy, an empirical model is an equation or set of equations formulated at a given level of organisation based on data gathered at that level; for example, input/output data gathered at the level of the animal (i) are used to describe or model animal level functions. In contrast, mechanistic models imply that both equation forms and parameter values arose from studies of lower level processes ($i-1, i-2, i-3$,..) (Baldwin, 1995). In general, the empirical modeller formulates equations at the given level of organisation based upon data gathered at that level, e.g. input/output data gathered at the animal level (i) are used to describe or model animal level functions (Baldwin, 1995). Regression analysis is the most common approach used in formulating such models. Riggs (1963) remarked that the only constraint that need to be imposed upon the form of a regression equation is that it best fits the data. However, regression equations can be formulated on the basis of concepts developed from studies of lower level processes ($i-1, i-2, i-3$, etc.), for example, the lactation curve model of Pollott (2000).

Furthermore, definition of the terms "theoretical" and "mechanistic" imply that both equation forms and parameter values in mechanistic models arose from studies of lower level processes. However, this is a difficult criterion to satisfy (Baldwin, 1995). For example, there are empirical models derived from underlying ($i-1$, $i-2$, $i-3$, etc.) processes, such as the model of Blaxter and Boyne (1978), which describes the efficiency of utilization of gross energy for maintenance and for body gain. These types of models may be referred to as *phenomenological*. Deterministic behaviour means simply that the future state of the system is predictable based on its current state and future input. In contrast, stochastic behaviour has an intrinsic (probabilistic) uncertainty about the future state of the system even if the current state and future inputs are completely known (Baldwin, 1995).

2.2.1 Empirical models: example of equations describing animal growth

The growth process is generally considered from two aspects; firstly, the increase of body mass as a function of time, usually described by the liveweight growth curve for

the whole animal, and secondly, changes in the form of the animal resulting from differences in the relative growth rates of the component parts of the body (Fowler, 1968). The simplest models of growth describe weight (W) as a function of time (t): $W = f(t)$. These equations are unrealistic because they assume that food is never limiting. The unconstrained growth curve is sigmoidal and thus f, which is required to be representative of potential growth from birth to maturity, must generate a curve of this nature. Thus, growth is a rate and many growth functions are obtained by considering the rate of growth ($\frac{dW}{dt}$) as a function of W: $\frac{dW}{dt} = h(W)$.

The most common empirical growth equations used in animal science are the Gompertz equation, logistic equation and Richards equation. The former two are special cases of the Richards equation. Richards (1959) proposed a flexible empirical model for describing different patterns of animal growth in which the rate of growth is given by the differential equation

$$\frac{dW}{dt} = \frac{k \times W}{(1-m)}\left[\left[\frac{Wmax}{W}\right]^{1-m} - 1\right] \qquad (2.1)$$

where $Wmax$ is the maximum attainable size and k is a rate constant. Flexibility is achieved by varying the parameter m. The solution to the differential equation is $W(t) = Wmax(1 - Ae^{-kt})^{\frac{1}{(1-m)}}$. The following are two of the most commonly used cases of Richards equation in animal science.

Logistic or autocatalytic (case $m = 2$):
The logistic equation assumes that growth is proportional to weight and to the amount of substrate available for growth (Gill and Oldham, 1993). For this model the relative growth rate declines linearly with size. The growth curve is symmetrical about its point of inflection at $W(t) = 0.5 \times Wmax$ where the growth rate is a maximum.

Gompertz equation (case m = 1):
The equation assumes that growth is proportional to weight and that substrate is nonlimiting. It also assumes that the fractional growth rate decreases exponentially with time. The equation generates a sigmoidal curve, but non-symmetrical, with the inflection point occurring at $\frac{1}{e}$ times the final weight (Gill and Oldham, 1993).

2.2.2 Mechanistic model development: Compartment models

A mechanistic approach to predicting animal productivity would be to simulate animal performance based on studies conducted at the tissue, cellular, and organ levels. Moreover, the parameters used in the equations must, if possible, be derived

from data obtained at the lower levels of organisation. Significant progress towards this end has been made in recent years, and it appears that mechanistic elements are currently in use in feeding systems to increase the accuracy of prediction and general applicability. For example, a mechanistic element currently used in feeding systems is the estimation of available (absorbed) amino acids from the protein degradation rate and microbial synthesis in the rumen.

A challenge is to develop a model that can trace through time the effects of submaintenance feeding on animal performance and the response to energy and protein. To meet this requirement, specific nutrients (e.g., glucose, amino acids, lipid, etc.) must be included as state variables since their pattern of use as energy sources and for productive functions affect productivity. In addition, factors such as relative organ weights that influence maintenance energy requirements over time must be explicitly represented.

For this purpose, compartmental systems have been used to represent biological concepts. A compartment is an amount of some material that is kinetically homogeneous (Godfrey, 1983; Jacquez, 1972). By kinetically homogeneous it is meant that the material of a compartment is at all times homogeneous; any material entering it is instantaneously mixed with the material of the compartment. The box in Figure 2.1 represents the *i*th compartment of an *n* compartment system. Let X_i represent the mass of compartment *i*. The arrows in Figure 2.1 represent the sum of the flows into and out of the compartment.

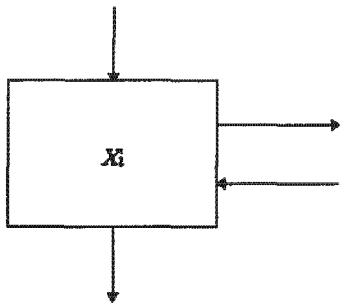


Figure 2.1: One compartment of a compartmental system.

The flows into the compartment from outside the system, or input, are represented by $In_i \geq 0$, the transfers from the *ith* to *jth* compartment by F_{ij}, the outflows to the environment and, therefore, out of the system by F_{io}. All the $F_{hk's}$ are ≥ 0.

The general equations for such a system are obtained by writing the instantaneous mass balance equations for each compartment:

$$\frac{dX_i}{dt} = \sum_{i=1}^{n} \mathrm{In}_i - \sum_{j \neq i} (F_{ij} - F_{ji}) - \sum_{i=1}^{n} F_{io}. \tag{2.2}$$

The inputs, In_i, are always nonnegative and are generally constant or functions of time only, though occasionally there may be systems in which the inflows are functions of X as well. The functions F_{ij}, F_{ji} and F_{io} can be functions of $X_1,...,X_n$. and possibly t. By definition, $F_{ii} \equiv 0$. All flows are defined to be nonnegative quantities; the signs in equation 2.2 take care of the direction of flows. Since there cannot be negative masses, the amounts X_i must always be nonnegative. Thus, when $X_i = 0$, $X_i \geq 0$ whatever the values of X_j for $j \neq i$.

The total flow out of a compartment over any interval of time must be bounded by the amount that was initially present plus the amount that flowed into the compartment during the interval. The following analytical properties summarize these physical constraints:

(1) $F_{ij} \geq 0, I_i \geq 0$ and $F_{io} \geq 0 \ \forall i, j, t.$

(2) If $X_i = 0$, then $F_{io} = 0$ and $F_{ij} = 0 \forall j$, so that $X_i \geq 0$.

Theorem 1: If F is k-times continuously differentiable, that is, C^k, then the first part of condition (2) implies that we can write each $F_{ij}(X)$ as $F_{ij}(X) \equiv f_{ij}(X). X_i$ for some function $f_{ij}(X)$, which is at least C^{k-1}.

Proof: Let $F(x) \geq 0$ be C^r, $r \geq 1$ and $F(0) = 0$. Consider the function $F(xt)$. $\frac{dF(xt)}{dt} = xF'(xt)$. (A)

Integrating in t, from zero to 1,

$$F(x) - F(0) = x\int_0^1 F'(xt)\,dt \tag{B}$$

The integral in (B) is a function of x, $f(x)$, and using $F(0) = 0$,

$$F(x) = xf(x).$$

From the derivation, if $F(x)$ is C^r, $f(x)$ *is* C^{r-1}. We will usually want $f(x)$ to be at least C^1. □

As a result, equation 2.2 can be written as

$$\frac{dX_i}{dt} = \underbrace{\text{In}_i}_{\text{Input}} + \underbrace{\sum_{j \neq i} f_{ji} \cdot X_i}_{\text{Production}} - \underbrace{f_{io}}_{\text{Output}} - \underbrace{\sum_{j \neq i} f_{ij}}_{\text{Utilization}} \qquad (2.3)$$

The f_{ij}'s are called the fractional transfer coefficients. In general, they will be functions of X and t. If the fractional transfer coefficients are constants or functions of time only, such systems are called linear systems. If each f_{ji} is a function of only X_j and some $X_j \mapsto f_{ji}(X_j)$ is not constant, system (2.3) is called nonlinear donor controlled.

The forms of the equations for the input, production, utilization and output can take many different forms depending on the biological mechanisms being described.

General reaction kinetics

Four principles guide the reaction kinetics (Pettigrew *et al.*, 1992). First, the kinetics apply to the entire transactions (pathways) in the model and not to individual reactions within these pathways. Second, the rate of a transaction is a function of the state of the animal. Specifically, the rates are determined by the concentration of substrates, and sometimes by the concentration of inhibitors and hormones. Third, model transactions are saturable with substrate, and the kinetics follow established patterns of saturable systems (Mahler and Cordes, 1971). Tissue catabolism transactions are the exception to this rule. Fourth, most rates are expressed per unit of tissue constituting the reaction site, to reflect differences in body size. These principles lead to the generalized Michaelis-Menten equation for a reaction velocity involving n substrates of concentration S_n;

$$P_{ij} = \frac{V_{max} \times X_i}{1 + \sum_{i=1}^{n} \frac{K_i}{S_i} + \sum_{i=1}^{n} \frac{I_i}{J_i}} \qquad (2.4)$$

where V_{max} is the maximum velocity per unit tissue and K_i is the affinity constant for substrates with concentration S_i, and I_i is the affinity constant for an inhibitor with concentration J_i. A low affinity constant produces a relatively high rate of reaction when substrate is limiting. Therefore, the priority of one reaction over another for a limiting substrate is produced in the model by assigning a smaller affinity constant to the high priority transaction. According to France *et al.* (1987), V_{max} is described by a modified logistic form which affects the rate of the production reaction, P_{ij}:

$$V_{max_i} = k_i \left[1 - \left[\frac{X_i}{X_{max_i}} \right]^{\theta_i} \right] \qquad (2.5)$$

where k_i is a rate constant, X_i is a variable describing the weight of a particular body component, X_{max_i} is the maximum value of X_i and θ_i is a steepness parameter of the

ith reaction. Therefore, a generalized production of a substance from the ith compartment to the j th compartment is

$$\text{Production} = P_{ij} = \frac{k_i \times X_i \times \left[1 - \left[\frac{X_i}{Xmax}\right]^{\theta_i}\right]}{1 + \sum_{i=1}^{n} \frac{K_i}{S_i} + \sum_{i=1}^{n} \frac{I_i}{J_i}} \tag{2.6}$$

For catabolic transactions, the utilization (U_i) of the i^{th} tissue follows simple linear kinetics:

$$U_i = v_i X_i \tag{2.7}$$

where v_i is a rate constant of the ith reaction. Figures 2.2(a) shows the effect of changing the value of the affinity constant (K_i) on the response of transaction rate to substrate concentration and Figure 2.2(b) shows the effect of changing the value of the steepness parameter on the response of transaction rate to substrate concentration (Pettigrew *et al.*, 1992).

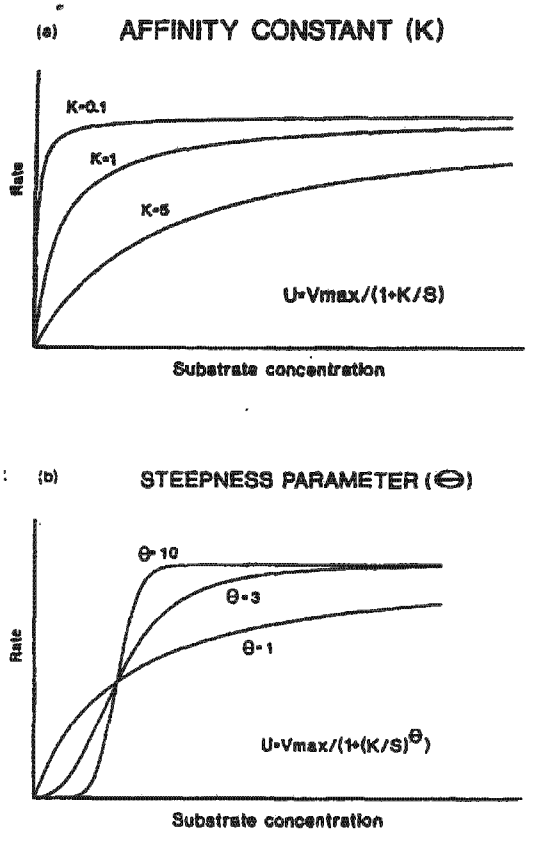


Figure 2.2: **(a)** The effect of changing the value of the affinity constant (K_i) on the response of transaction rate to substrate concentration and **(b)** the effect of changing the value of the steepness parameter (θ) on the response of transaction rate to substrate concentration.

2.3 Framework for integrating system components

2.3.1 Aggregating the system

Most mechanistic models in ruminant digestion have dealt with parts of the whole digestive system largely because of the increased number of differential equations with increasing model complexity which results in tedious computation. Bywater (1990) suggested that the aggregation of detailed explanatory models (which represent mechanisms of underlying biological processes) provides a suitable method of establishing models at an appropriate resolution for inclusion in whole farm studies. He also suggested that knowledge derived from detailed models can be used to identify and select factors to be included in aggregate models. Models established by such a procedure will tend to predict performance across differing environments better than empirical models or models which attempt to provide a mechanistic representation of biological processes, but which have not formally identified the relative importance of modelled processes.

In this section, a systems approach to the development of mathematical models is provided. The driving force behind the creation of models is the admission that "Truth is elusive, but we can gradually approximate it by creating better and better representations" (Yeargers *et al.*, 1996). One reason why the truth is so elusive in real systems is because the universe is extremely complex. No living system operates in isolation; it is part of a living network arranged in a hierarchical fashion. Miller (1978) proposed a general approach to the analysis of living systems in which the presence of seven hierarchical levels were postulated; namely, supranational, national, organization, group, organism, organ and cell. The level at which we study biological systems in animal nutrition is at the level of the organism and organ. We have eliminated the cell as a distinct level and have aggregated the levels above the organism level and refer to it as the *environment.* Therefore, our model of a living system is composed of three interconnected levels in the following order from top to bottom; 1) environment, 2) organism, 3) tissue/organ.

In addition to dividing living systems horizontally, Miller (1978) also divided them vertically; namely, matter/energy and information processing systems. Such a division cannot be made for two reasons; firstly, all matter/energy processing systems have information processing subsystems incorporated in them for control purposes and separating the two systems on this level is practically impossible, even though it is possible on a subsystem level (Jaros *et al,* 1988). Secondly, information processing in

itself is often achieved by the handling of matter/energy. A good example is the endocrine system, in which hormones are physically circulated for control purposes. Jaros *et al.* (1979) proposed to divide an organism vertically into two major groups of processes: *endodynamic* processes and *exodynamic* processes (Figure 2.3). Although such a grouping is made, none of these processes can proceed in isolation. In fact, they interact with one another at various levels. The division simply signifies that the interactions within the groups are much stronger than between groups (Jaros *et al.*, 1988). However, the main reason for the division is the similarity in the way they function and to which their purposes are aimed.

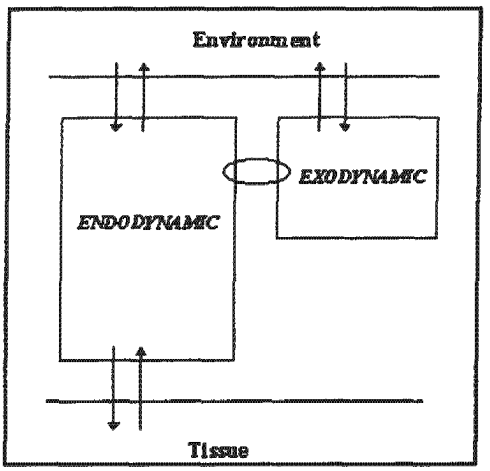


Figure 2.3: Diagram showing the relationship between the three hierarchical levels and the enodynamic and exodynamic processes.

Endodynamic processes are directed towards a lower level of the hierarchy. On the level of the organism, endodynamic processes are directed towards the maintenance of the tissue. The processes in this category are mainly concerned with the transfer of matter/energy between the environment and the tissues and vice versa. The substances involved in the transfer are nutrients from the diet and waste products. Matter/energy transfer can take place in both an upward and a downward direction through the organism. Exodynamic processes are aimed towards the external environment of the hierarchical level under observation. The reason for the existence of these processes is to ensure the continued existence of the organism within its environment. An example would be reproduction. The subdivision of the whole system into endodynamic and exodynamic processes helps to sort available knowledge into smaller compartments.

2.3.2 Endodynamic Processes

At the level of the organism, endodynamic processes can be defined as the study of processes involved in the conservation of optimal tissue function (Jaros *et al.*, 1988). Although these processes are concerned primarily with the processing of matter and energy, each of them requires a considerable amount of information processing for control purposes. The proposed framework (Figure 2.4) is adapted for models in nutrition from Jaros *et al.* (1988).

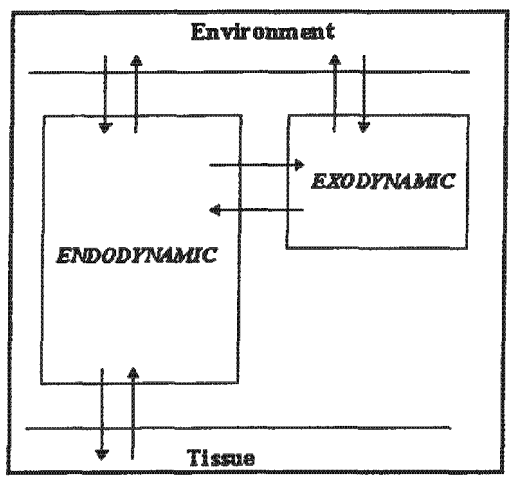


Figure 2.4: A framework for endodynamic processes.

The framework divides endodynamic processes into two major types of subsystems, namely, matter/energy subsystems and co-ordination (information processing) subsystems. The matter/energy subsystems fulfil the endodynamic purpose of providing a link between the environment and the tissue and between tissue and tissue, while the co-ordination processes can be regarded as accessory to ensure optimal functioning.

2.3.2.1 Matter/Energy subsystem

There are similarities between the various matter/energy and coordination processes. Both the matter/energy and coordination systems can be divided into four types of subsystems (Figure 2.5): (1) Input/output subsystems, (2) Distribution subsystems, (3) Processing subsystems, and (4) Tissue subsystems.

Figure 2.6 gives a scheme of the proposed RUMET (RUmen and METabolism) model developed in this thesis.

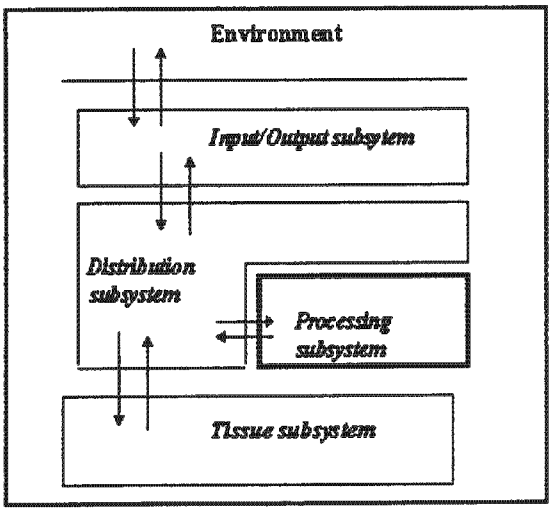


Figure 2.5: A diagram of matter/energy processing subsystems.

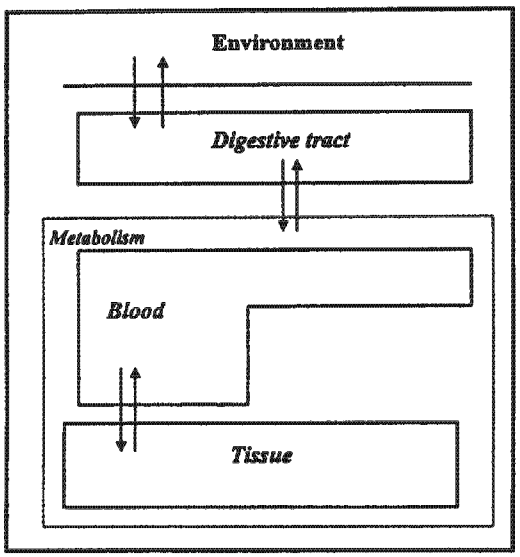


Figure 2.6: A scheme of the RUMET model.

a) Input/Output subsystems

The matter or energy needed by the tissues is obtained from the environment. The input/output system is situated between the environment and the organism, and is involved in the exchange of matter or energy between the two levels in both directions. This divide could be the feed intake of the organism. An example of a subsystem in this group may be the *rumen*. The rumen is part of the digestive tract of the ruminant. Of central importance to the nutrition of ruminant animals is the existence of a large microbial population within the rumen which serves to digest ingested nutrients with the associated production of volatile fatty acids (VFA) and provides a substantial proportion of the total protein absorbed from the small intestine,

through the ruminal synthesis and subsequent small intestinal digestion of microbial biomass. The rumen also plays an important role in the control of intake.

b) Distribution subsystems

The function of these subsystems is to transport substances between the various parts of the organism. There are several ways that transport can take place. We are specifically concerned with the transport of nutrients or substances via the blood but it may also be, for example, via the lymphatic system etcetera. In many cases more than one method is utilized. An example of a subsystem in this group may be the *circulatory system.* It is the most important distribution subsystem. The heart provides the power for the circulation of blood, which acts as the distributing medium, while the blood vessels are the conduits. Matter/energy enters and leaves it via the capillary vessels in the organs. In animal nutrition, models have assumed the blood to be static, that is, models have not accounted for the flow of blood from compartment to compartment, with the exception of one model by Cant *et al*, (2001). Dynamic models may include the flow of blood especially to account for basal energy costs.

c) Processing subsystem

Substances taken up by the input/output subsystem cannot always be utilized immediately or in the same form as they entered the organism. The processing subsystems modify the substances for use in intermediary metabolism. An example of a subsystem in this group may be the *liver.* It is the most important single processing subsystem, and its position in series with the gastrointestinal tract (GIT), and more significantly, between the GIT and the peripheral circulation, bears witness to its importance. It is the major organ where chemical transformation processes occur and also serves to balance the nutrients in metabolism. In addition, it also serves as a storage organ for different substances such as protein, glycogen, iron and various vitamins.

d) Tissue subsystem

These substances are the important sources of deposition of the end-products of digestion and metabolism of nutrients. The dynamics within these tissues are important. Examples of a subsystem in this group may be the *mammary gland* and *muscle.* The mammary gland of the ruminant is important in the conversion of nutrients into milk and the muscle is the biggest store of protein in an animal and important for posture and movement (and has other functions).

2.3.2.2 Co-ordination processes

Endodynamic processes are complex and depend on the contributions from many organs and tissues. The main point to remember is that there is a complex, interdependent and continuous sequence of reactions, flows, and time constants, all of which contribute to the process. Each of the components must function properly in order to ensure the efficiency of the entire process. Therefore, a stringent control must be exercised on the components. In our model of animal nutrition we are mainly concerned with the endocrine system via hormones and assume that the autonomic nervous system has little effect on the utilization of the nutrients. This may, however, be found to be important because of the increasing interest in the neural effects on hormones (Lawrence and Fowler, 1997).

PART II

MODEL DEVELOPMENT

Chapter 3

Modelling rumen function: the effects of different N supplements on low quality roughages

3.1 Challenges for rumen models

Understanding rumen function, with respect to nutrient digestion and ultimately nutrient supply, requires an understanding of rumen microbial dynamics (see chapter 4), rumen metabolism (energy and protein interactions) and the degradation of substrate and formation of endproducts (Figure 3.1). The importance of energy and protein interactions within the rumen is well established and research effort has mainly been directed towards qualitative descriptions of rumen processes and how such processes may be manipulated in order to improve overall efficiency of ruminant livestock production (Chalupa, 1977). However, due to the development of improved experimental techniques, including automated techniques, the quantity and quality of data has increased tremendously leading to an increase in quantitative descriptions of rumen processes. This facilitated the development of mechanistic models as a framework to integrate knowledge of rumen processes in order to understand and/or predict aspects of rumen function. Initiated by the pioneering work of Baldwin *et al.* (1970), several mathematical models of whole rumen function were developed in order to integrate rumen processes. These models do not necessarily share common objectives and the evaluation of these models depends on an appraisal of the total effort in relation to the objectives of the modelling exercise (France and Thornley, 1984).

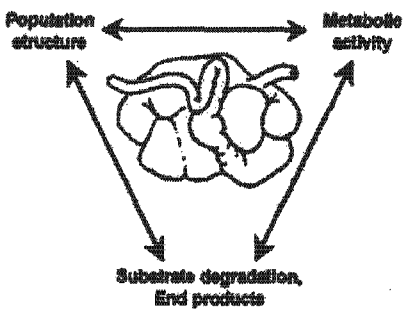


Figure 3.1: Understanding rumen function requires understanding rumen microbial dynamics, rumen metabolism (energy and protein interactions) and the degradation of substrate and formation of endproducts.

France *et al.* (1982) were the first to develop a rumen model based on the rate:state formalism. Most of the rumen models developed subsequently followed their approach. Baldwin *et al.* (1987) developed a model of rumen function in dairy cattle.

Their objective was to predict the rate and pattern of nutrient absorption in the dairy cow. The limited evaluation against in vivo data in high producing dairy cattle was satisfactory for most diets, with the exception of the volatile fatty acid (VFA) production rates. Some of the suggestions to improve the model were addressed by Argyle and Baldwin (1988) and Baldwin (1995). Dijkstra *et al.* (1992) extended the models of Baldwin *et al.* (1987) primarily by improving the representation of the microbial population. The objective of their modelling exercise was to examine the effect of supplementation of forage diets on the profile of nutrients available for absorption in cattle. Specific areas of improvement included representing a combination of microbial substrate preference, differential outflow and microbial composition, and the recycling of microbial matter. This model was tested thoroughly on a range of diets and generally provided reasonable predictions, with the exception of VFA molar proportions (Neal *et al.*, 1992). However, the model tries to be too general and simulates a wide range of possible diets. It was shown that the representation of protozoan activities needed more attention and this was subsequently addressed by Dijkstra (1994), in which the objective was to provide a quantitative understanding of protozoan dynamics. This mathematical integration of dietary, bacterial and protozoal factors improved the understanding of factors involved in the contribution of protozoa to fibre degradation (Dijkstra and Tamminga, 1995) and microbial recycling (Dijkstra *et al.*, 1998) in the rumen. Dijkstra *et al.* (1996) also constructed a simple mechanistic model of digestion of sugarcane in cattle. The aggregation level and mathematical approach were based on the model of France *et al.* (1982). The primary objective of their rumen model was to predict nutrient supply to the host animal from intake of sugarcane and supplements as a means of indicating pre-experimentally which combinations of available supplements are most likely to enhance animal production.

Progress towards the prediction of rumen output has been encouraging, however, Dijkstra and France (1996) listed some of the major limitations that remain: examining the effect of discontinuous feeding regimes and outflow of material from the rumen, the effects of microbial distribution in the rumen on fermentation and degradation of substrate, the effect of manipulating microbial interactions, examining the factors determining VFA profile, and examining the factors affecting the amino acid composition of microbial matter and undegraded food.

3.2 Nitrogen supplementation for cattle on rangelands

During the dry winter months on arid and semi-arid rangelands, the crude protein content of the dry herbage is low (10-30g/kg), while the percentage of crude fibre range from 400-450g/kg (Elliott *et al.*, 1965). A protein (nitrogen) deficiency reduces

feed intake by limiting the rate of microbial growth and, therefore, the rate of digestion of organic matter and hence the clearance of digesta from the rumen. Consequently, microbial protein synthesis is limited and smaller quantities of amino acids are available in the intestine to meet the animal's tissue demand for protein. Initially, a lack of herbage nitrogen sources for the microbes in the rumen can be overcome with nitrogen supplements of protein or non-protein nitrogen that are degradable in the rumen. If the resulting increased microbial protein flow to the intestine is not sufficient to meet the tissue requirements for amino acids after supplementation, the short-fall can be met by supplements of protein not subjected to rumen degradation. Research has aimed to predict rumen microbes protein requirement so that the maximum intake of roughage can be achieved for the minimal input of protein supplements.

For these reasons, the rumen model developed by Dijkstra *et al.* (1996) was modified and used to examine the effects of different nitrogen supplements on a low-quality roughage diet. The rumen model was modified in three ways: firstly, to predict voluntary food intake by cattle grazing tropical rangeland, secondly, to account for the delay due to the attachment of microorganisms to food particles and thirdly, by adding a routine to calculate the molar proportions of VFA from the data of Reed *et al.* (1968). Notation and symbols are defined in Table 3.1 and Table 3.2, respectively, and follows the notation and symbols used by Dijkstra *et al.* (1996).

3.3 Rumen model changes

This section will describe the three changes made to the rumen model developed by Dijkstra *et al.* (1996).

3.3.1 Adding an intake component to predict voluntary food intake

The total dry matter intake, $DMItot$ (g/day), can either be limited by intake controlled by the rate of digestion (DMI, g/day) or by the rate at which the animal eats ($BiDMI$, g/day), described by Poppi *et al.* (1994). The model combines these two mechanisms of total food intake in the following way:

$$DMItot = min[DMI, BiDMI]. \tag{3.1}$$

Firstly, intake (DMI) may be limited by the digestive system, which is modelled as a function of the rate of output of faecal dry matter and the digestibility of the diet selected. Rumen volume is related to body weight and is at a maximum when the animal's weight is 0.75 of its mature weight (Butterfield, 1988). In animals weighing less than 0.75 of their mature weight, rumen volume is assumed not to decrease with a

decrease in liveweight. Following Richardson *et al.* (2000), a routine was added to ensure that the rumen volume does not decrease if an animal of less than 0.75 mature size loses weight. This is because *ad libitum* intakes of animals that have lost weight are greater than in animals of the same weight that have grown continuously (Saudibet and Verde, 1976). Therefore, intake (DMI) is estimated from the rumen volume, $Rvol$ (L), maximum concentration of dry matter in the rumen, $cDMRu$ (g/L), and the fractional rate of disappearance of dry matter from the rumen, $kRuEx$ (Richardson *et al*, 2000):

$$DMI = kRuEx \times cDMRu \times RVol. \tag{3.2}$$

The fractional rate of disappearance of dry matter from the rumen, $kRuEx$, is calculated as the proportion of the total loss of dry matter from the rumen by absorption, passage and as gas, $UDMRu$ (equation 3.32 in Appendix 3.1), and the total weight of dry matter in the rumen, $RumenDM$ (equation 3.33 in Appendix 3.1): $kRuEx = \frac{UDMRu}{RumenDM}$. The concentration of dry matter in the rumen ($cDMRu$) was assumed to be 80.06 g/L (Czerkawski, 1986). The rumen volume is related to liveweight by the equation of Butterfield (1988):

$$Yrvol = 3.0 \times Xwt + (1 - 3.0) \times (Xwt)^2 \tag{3.2a}$$

$$\text{where } Yrvol = \frac{Rvol}{Rvolmat} \tag{3.2b}$$

$$Xwt = \frac{Weight}{Wmax} \tag{3.2c}$$

$Rvol$	volume of the rumen (litres)
$Rvolmat$	volume of the rumen at maturity (assumed to be 75 litres)
$Weight$	weight of the animal (kg)
$Wmax$	mature weight of the animal, kg (assumed to be 650kg)

Secondly, intake ($BiDMI$) can be limited by the rate at which the animal is able to eat herbage:

$$BiDMI = Bbite \times maxbite \times AdjDM. \tag{3.3}$$

This intake ($BiDMI$) depends on the size of the mouth, herbage density and the maximum number of bites per day, $maxbite$. The maximum number of bites per day, $maxbite$, increases from zero at birth to 38 000 at 16 weeks. Maximum bite size is a function of arcade breadth, Arc, which is related to the peak weight (highest weight so far attained by the animal) already attained by an animal (Taylor *et al.*, 1987):

$$Arc = 9.1 \times Weight^{0.29} \times Wmax^{0.07}. \quad (3.3a)$$

Peak weight is used as arcade breadth does not decrease if an animal loses weight. $Bbite$ is the amount eaten in one bite if forage density is optimal. It is estimated to be 0.00035 kg per individual bite. $Bbite$ is also a function of arcade breadth, Arc:

$$Bbite = Abite \times Arc. \quad (3.3b)$$

We have simulated animals over a relatively narrow weight range (200-250 kg) and have therefore assumed $Abite$ to be constant, $Abite = 0.0000041$.

The intake ($BiDMI$) is adjusted for herbage density using the following adjustment factor, $AdjDM$, based on the approach of Johnson and Parsons (1985):

$$AdjDM = \frac{\left(\frac{shoot}{kshoot}\right)^3}{1+\left(\frac{shoot}{kshoot}\right)^3}. \quad (3.3c)$$

where shoot is the density of the herbage in $\frac{g}{m^2}$ and kshoot is the maximum density of the herbage (assumed to be $120\frac{g}{m^2}$).

3.3.2 Delay of microorganism attachment to food particles

When animals eat tropical forages, the food entering the rumen consists of a high proportion of course fibrous particles that affect the pattern of digestion in the rumen and passage of matter to the duodenum. When fibrous particles enter the rumen, microbes must become attached to them before fermentation can begin. In addition, the size or specific gravity of digesta particles must reach a critical value before they can pass from the rumen. The delay in the attachment of microbes does not affect digestibility (Allen and Mertens, 1988) but contributes to rumen fill and so reduces intake. The delay in the onset of fermentation has been demonstrated experimentally by Pienaar and Roux (1989).

For these reasons, the undegradable protein (Pu) and insoluble degradable protein (Pd) pools and the undegradable fibre (Fu) and degradable fibre (Fd) pools, were divided into forage and non-forage components. The fibre referred to is crude fibre. The non-forage components consists of a single pool and is exactly the same as the undegradable protein (Pu) and insoluble degradable protein (Pd) and undegradable fibre (Fu) and degradable fibre (Fd) pools described in Dijkstra *et al.* (1996). However, for each forage undegradable protein and insoluble degradable protein and forage undegradable fibre and degradable fibre pools, three pools (unavailable

unescapable pool, available unescapable pool, available escapable pool) were added so that the lag at the start of fermentation and selective passage of forage particles may be simulated. Therefore, twelve additional forage pools were added to the model developed by Dijkstra *et al.* (1996). The representation of the delay components of both forage protein and forage fibre is based on model III of Allen and Mertens (1988), but the action of rumen micro-organisms is represented explicitly. The forage fibre and protein components are transferred from the pool where it is not available to microorganisms and cannot escape from the rumen (unavailable unescapable pool, UaUe) to the pool where it is available for microoganisms but not able to escape from the rumen (available unescapable pool, AvUe) and then to the pool where it is available to the microorganisms and able to escape from the rumen (available escapable pool, AvEs). As an example, Figure 3.2 gives the scheme of the three forage degradable fibre pools. The input to the forage protein and fibre pools is from food component 1 (D_i, see, for example, equation 3.8) and the input to the non-forage pools is from food component 2 (D_i, see, for example, equation 3.8).

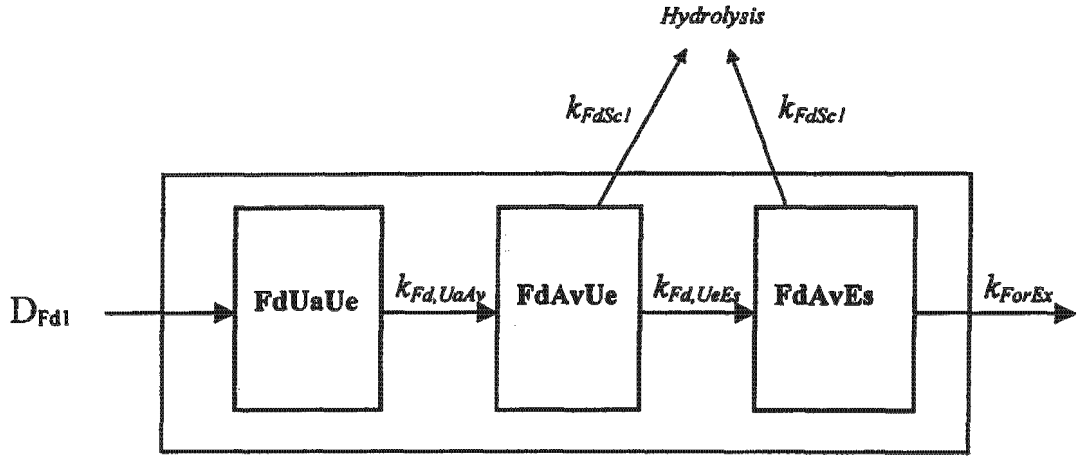


Figure 3.2: Sub-model of forage degradable fibre.

The sub-model of forage fibre and protein in the rumen is included in the equations in Appendix 3.1. The notation follows that of Dijkstra *et al.* (1996). Consequently, the values of $k_{Pu,UaAv}$, $k_{Pd,UaAv}$, $k_{Fd,UaAv}$ and $k_{Fu,UaAv}$ are the same fractional rate as the digesta particles (12/day). The values of $k_{Pd,UeEs}$, $k_{Pu,UeEs}$, $k_{Fd,UeEs}$, $k_{Fu,UeEs}$ are assumed to be the same (6.0/day).

3.3.3 Molar proportion of volatile fatty acids

A routine was included to calculate the molar proportions of VFA from the data of Reed *et al.* (1968):

$$A_{Ac} = FrAc \times A_{VFA} \quad (3.4a)$$

$$A_{Pr} = FrPr \times A_{VFA} \quad (3.4b)$$

$$A_{Bu} = FrBu \times A_{VFA} \quad (3.4c)$$

where A_{Ac}, A_{Pr}, A_{Bu} are the absorbed acetic, proprionic and butyric acid (in moles) respectively, and FrAc, FrPr, FrBu are the fractions of absorbed acetic, propionic and butyric acid estimated to be 0.63, 0.23 and 0.14, respectively.

3.4 General model structure

This scheme is based on the model developed by Dijkstra *et al.* (1996) with the modifications already described. The scheme adopted for the model is given in Figure 3.3, except that the forage fibre and protein pools have been divided, as shown in the example in Figure 3.2, and is indicated by broken boxes (- - -) in Figure 3.3. Principal model symbols are defined in Table 3.1 and model notation is listed in Table 3.2. The rumen model comprises 23 state variables represented as solid and broken boxes in Figure 3.3. Four zero pools are defined for the hindgut which represents nutrients available for absorption and is represented as dotted boxes (...) in Figure 3.3. The food components are represented as three major fractions: a nitrogen-containing fraction, a carbohydrate containing fraction and a fatty acid fraction. The nitrogen-containing fractions in the rumen include non-forage undegradable protein (Pu), non-forage insoluble degradable protein (Pd), three forage undegradable protein (PuUaUe, PuAvUe, PuAvEs), three forage degradable protein (PdUaUe, PdAvUe, PdAvEs), soluble protein (Ps) and ammonia (Am). The carbohydrate fractions in the rumen include non-forage undegradable fibre (Fu), non-forage degradable fibre (Fd), three forage undegradable fibre (FuUaUe, FuAvUe, FuAvEs), three forage degradable fibre (FdUaUe, FdAvUe, FdAvEs), insoluble starch (Si) and soluble starch and sugars (Sc). The fatty acid fractions in the rumen are long chain fatty acids (Ld) and volatile fatty acids (Va). A single pool representing an aggregated microbial DM (Mi) is included in the model.

All pools are expressed in grams except for the volatile fatty acids that are expressed in moles, volume is expressed in litres and time in days. Because the time scale of the rumen model developed by Dijkstra *et al.* (1996) is in hours and the desired time scale is days, all the rate constants in the model were multiplied by twenty four. This could be done because the models on the daily and hourly time scales gave similar output. The concentration of each entity (C_i) is the pool size divided by the rumen volume. General model assumptions are that the growth of microbes is related to the availability of energy (Sc) and nitrogen (either Ps or Am) and it is assumed that energy derived from protein fermentation is not significant relative to the energy derived from carbohydrate fermentation (Russel *et al.*, 1983). The composition of Mi and the amounts of carbohydrates and Am or Ps needed for growth are adopted from Reichl and Baldwin (1975), assuming a yield of 4 mol ATP/mol glucose.

The subscripts used in the hydrolysis rates (for example, k_{FdSc_1} and k_{FdSc_2}) refers to the components of the diet, that is, component 1 or 2.

Table 3.1*: Symbols used in the model.

Symbol	Description
Aa	Post-rumen amino acids
Ab	Absorption
Am	Ammonia
Ex	Exit from rumen
Fd	Non-Forage rumen degradable fibre
Fl	Rumen fluid phase
Fu	Non-Forage rumen undegradable fibre
Gl	Post-rumen glucose
Ld	Rumen long chain fatty acid
Li	Post-rumen long chain fatty acids
Mi	Microbial dry matter
N	Nitrogen
Pd	Non-Forage rumen insoluble, degradable protein
Ps	Rumen soluble protein
Pu	Non-forage rumen undegradable protein
Sc	Rumen soluble starch and sugars
Si	Rumen insoluble starch
So	Rumen solid phase
Ur	Urea
Va	Rumen volatile fatty acids
Vf	Post-rumen volatile fatty acids
VFA	Total absorbed volatile fatty acids
PuUaUe	Forage undegradable protein, unavailable and unescapable
PuAvUe	Forage undegradable protein, available and unescapable
PuAvEs	Forage undegradable protein, available and escapable
PdUaUe	Forage degradable protein, unavailable and unescapable
PdAvUe	Forage degradable protein, available and unescapable
PdAvEs	Forage degradable protein, available and escapable
FuUaUe	Forage undegradable fibre, unavailable and unescapable
FuAvUe	Forage undegradable fibre, available and unescapable
FuAvEs	Forage undegradable fibre, available and escapable
FdUaUe	Forage degradable fibre, unavailable and unescapable
FdAvUe	Forage degradable fibre, available and unescapable
FdAvEs	Forage degradable fibre, available and escapable

*Notation and symbols are adopted from Dijkstra *et al.* (1996)

Table 3.2*: Notation used in the model.

Notation	Translation	Units
A_i	Absorption rate of *i*	g/day
C_i	Concentration of *i*	g/L
D_i	Dietary input of *i*	g/day
k_{ij}	Fractional rate constant for *i*-*j* transaction	/day
$k_{i,jk}$	Fractional rate constant of *i* in *j*-*k* transaction	/day
$J_{i,jk}$	Inhibition constant for *j*-*k* transaction with respect to *i*	g*i*/L
$M_{i,jk}$	Affinity constant for *j*-*k* transaction with respect to *i*	g*i*/L
$P_{i,jk}$	Rate of production of *i* in *j*-*k* transaction	g*i*/day
Q_i	Quantity of *i*	g *i*
$R_{i,jk}$	Requirement for *i* in *j*-*k* transaction	g*i*/g*j*
t	Time	days
$U_{i,jk}$	Rate of utilization of *i* by *j*-*k* transaction	g*i*/day
v_{ij}	Velocity for *i*-*j* transaction	/day
$Y_{i,jk}$	Yield of *i* for *j*-*k* transaction	g*i*/g*j*

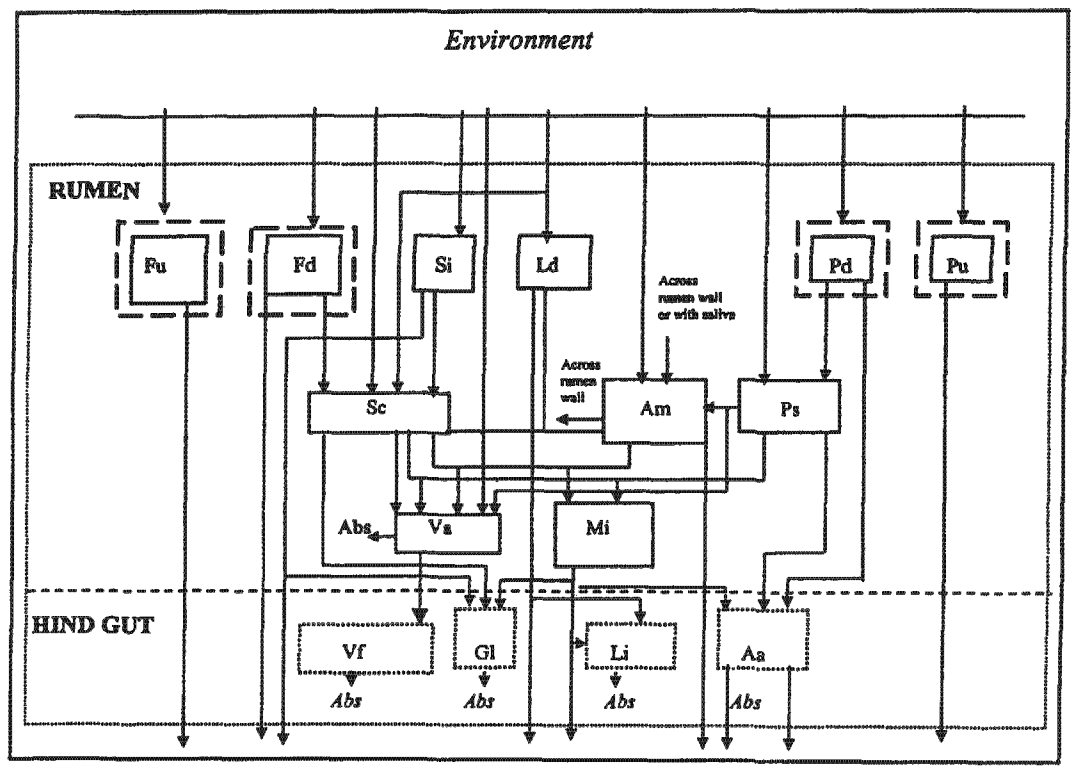


Figure 3.3: A modified rumen model

*Notation and symbols are adopted from Dijkstra *et al.* (1996)

The nutrients absorbed in the intestines are amino acids and small peptides (A_{Aa}), glucose (A_{Gl}), long chain fatty acids (A_{Li}) and volatile fatty acids (A_{Vf}). Fermentation of fibre and starch not absorbed from the small intestine is represented as well, these fermentation products are assumed to be absorbed as Vf from the large intestine.

Model Input

Three different food components ($DMI_j, 1 \leq j \leq 3$), constituting the diet, can be used as input to the model by specifying the proportion of each component ($frDMI_j$) multiplied by the total dry matter intake (DMI_{tot}):

$$DMI_j = frDMI_j \times DMI_{tot}. \tag{3.5}$$

The input can either be a specified amount of food (as in equation 3.6 below) or an adlib intake (as in equation 3.2).

$$DMI_{tot} = food + bint \times days, \tag{3.6}$$

where $food$ is the amount of food in grams and $bint$ is a constant for the frequency of feeding per day. The $j = 1$ and 2 components of the diet are made of ten (10) constituents which are estimated from experimental data as far as possible (for example, see Table 3.3).

$$D_i = C_i \times DMI_j \qquad i = 1 \text{ to } 10, \quad j = 1 \text{ to } 2. \tag{3.7}$$

In addition, for example, we can write the total dietary Fd as the sum of the Fd constituent from food component 1 (D_{Fd_1}) and from food component 2 (D_{Fd_2}) as

$$D_{Fd} = D_{Fd_1} + D_{Fd_2} \tag{3.8}$$

The $j = 3$ component is used for a nitrogen supplement only and therefore only contains one constituent (usually ammonia).

3.5 Description of the model

The parameters and variables for each pool will be described now. The differential equation will be given after the description of each pool and all equations are listed in Appendix 3.1 and all parameter values are given in Appendix 3.2.

Forage Undegradable Fibre

Forage unavailable unescapable undegradable fibre, Q_{FuUaUe} (g).

There is one input to this pool from the food (D_{Fu_1} or $P^*_{Fu,InFu}$: equation 3.9a) and the outflow is to the available unescapable undegradable fibre pool ($U_{Fu,UaAv}$: equation 3.9b).

$$\frac{dQ_{FuUaUe}}{dt} = \underbrace{P^*_{Fu,InFu}}_{\text{diet input}} - \underbrace{U_{Fu,UaAv}}_{\substack{\text{flow to available} \\ \text{unescapable undegradable fibre}}} \tag{3.9}$$

Forage available unescapable undegradable fibre, Q_{FuAvUe} (g).

There is one input to this pool from the unavailable unescapable undegradable fibre pool ($P_{Fu,UaAv}$: equation 3.10a) and the outflow is to the available escapable undegradable fibre pool ($U_{Fu,UeEs}$: equation 3.10b).

$$\frac{dQ_{FuAvUe}}{dt} = \underbrace{P_{Fu,UaAv}}_{\substack{\text{inflow from unavailable} \\ \text{unescapable undegradable fibre}}} - \underbrace{U_{Fu,UeEs}}_{\substack{\text{outflow to available escapable} \\ \text{undegradable fibre}}} \tag{3.10}$$

Forage available escapable undegradable fibre, Q_{FuAvEs} (g).

There is one input to this pool from the available unescapable undegradable fibre pool ($P_{Fu,UeEs}$: equation 3.11a) and the outflow is out of the rumen ($U_{Fu,EsEx}$: equation 3.11b).

$$\frac{dQ_{FuAvEs}}{dt} = \underbrace{P_{Fu,UeEs}}_{\substack{\text{inflow from available} \\ \text{unescapable undegradable fibre}}} - \underbrace{U_{Fu,EsEx}}_{\text{outflow with solid material}} \tag{3.11}$$

Non-Forage Undegradable fibre pool, Q_{Fu} (g).

There is one input to this pool from the food (D_{Fu_2} or $P_{Fu,InFu}$: equation 3.12b) and the outflow is with the solid material ($U_{Fu,FuEx}$: equation 3.12c).

$$\frac{dQ_{Fu}}{dt} = \underbrace{P_{Fu,InFu}}_{\text{diet input}} - \underbrace{U_{Fu,FuEx}}_{\text{outflow with solid material}} \tag{3.12}$$

Forage Degradable Fibre

Forage unavailable unescapable degradable fibre, Q_{FdUaUe} (g).

There is one input to this pool from the food (D_{Fd_i} or $P^*_{Fd,InFd}$: equation 3.13a) and the outflow is to the available unescapable degradable fibre pool ($U_{Fd,UaAv}$: equation 3.13b).

$$\frac{dQ_{FdUaUe}}{dt} = \underbrace{P^*_{Fd,InFd}}_{\text{diet input}} - \underbrace{U_{Fd,UaAv}}_{\substack{\text{flow to available} \\ \text{unescapable degradable fibre}}} \tag{3.13}$$

Forage available unescapable degradable fibre, Q_{FdAvUe} (g).

There is one input to this pool from the unavailable unescapable degradable fibre pool ($P_{Fd,UaAv}$: equation 3.14a). There are two outputs from this pool: the hydrolysis of Fd to Sc by the rumen microorganisms ($U_{Fd,UeSc}$: equation 3.14b) and the outflow to the available escapable degradable fibre pool ($U_{Fd,UeEs}$: equation 3.14c). The hydrolysis rate (k_{FdSc_i}) can be calculated from the relevant experimental input data chosen and C_{refMi} is a reference value (12.5 g/L) of the concentration of microbial DM in the rumen based on data reported by Robinson *et al.* (1987).

$$\frac{dQ_{FdAvUe}}{dt} = \underbrace{P_{Fd,UaAv}}_{\substack{\text{inflow from unavailable} \\ \text{unescapable degradable fibre}}} - \underbrace{U_{Fd,UeSc}}_{\text{hydrolysis to Sc}} - \underbrace{U_{Fd,UeEs}}_{\substack{\text{outflow to available escapable} \\ \text{degradable fibre}}} \tag{3.14}$$

Forage available escapable degradable fibre, Q_{FdAvEs} (g).

There is one input to the available escapable degradable fibre pool ($U_{Fd,UeEs}$: equation 3.15a). There are two outputs from this pool: the hydrolysis of Fd to Sc by the rumen microorganisms ($U_{Fd,EsSc}$: equation 3.15b) and the outflow to the hindgut ($U_{Fd,EsEx}$: equation 3.15c). The hydrolysis rate (k_{FdSc_i}) can be calculated from the relevant experimental input data chosen and C_{refMi} is a reference value (12.5 g/L) of the concentration of microbial DM in the rumen based on data reported by Robinson *et al.* (1987).

$$\frac{dQ_{FdAvEs}}{dt} = \underbrace{P_{Fd,UeEs}}_{\substack{\text{inflow from available} \\ \text{unescapable degradable fibre}}} - \underbrace{U_{Fd,EsSc}}_{\text{hydrolysis to Sc}} - \underbrace{U_{Fd,EsEx}}_{\text{outflow}} \tag{3.15}$$

Non-Forage Degradable fibre pool, Q_{Fd} (g).

There is one input to this pool from the food (D_{Fd_2}: equation 3.16b). There are two outputs from this pool: the hydrolysis of Fd to Sc by the rumen microorganisms ($U_{Fd,FdSc}$: equation 3.16c) and the outflow to the hindgut with the solid material ($U_{Fd,FdEx}$: equation 3.16d). The hydrolysis rate (k_{FdSc_2}) can be calculated from the relevant experimental input data chosen and C_{refMi} is a reference value (12.5 g/L) of the concentration of microbial DM in the rumen based on data reported by Robinson *et al.* (1987).

$$\frac{dQ_{Fd}}{dt} = \underbrace{P_{Pd,InFd}}_{\text{diet input}} - \underbrace{U_{Fd,FdSc}}_{\text{hydrolysis to Sc}} - \underbrace{U_{Fd,FdEx}}_{\text{outflow with solid material}} \qquad (3.16)$$

Insoluble starch pool, Q_{Si} (g).

There is one input to this pool from the food ($P_{Si,InSi}$: equation 3.17b). There are two outputs from this pool: the hydrolysis of Si to Sc by microorganisms ($U_{Si,SiSc}$: equation 3.17c) and the outflow of Si to the hindgut with the solid material ($U_{Si,SiEx}$: equation 3.17d).

$$\frac{dQ_{Si}}{dt} = \underbrace{P_{Si,InSi}}_{\text{diet input}} - \underbrace{U_{Si,SiSc}}_{\text{hydrolysis to Sc}} - \underbrace{U_{Si,SiEx}}_{\text{outflow with solid material}} \qquad (3.17)$$

Soluble starch and sugars pool, Q_{Sc}.

There are four inputs to this pool: from the food ($P_{Sc,InSc}$: equation 3.18b), the hydrolysis of dietary lipid ($P_{Sc,InLd}$: equation 3.18c), the hydrolysis of Fd ($P_{Sc,FdSc}$: equation 3.18d) and the hydrolysis of Si ($P_{Sc,SiSc}$: equation 3.18e). The yield of Sc from the hydrolysis of dietary lipid ($Y_{Sc,InLd}$) is estimated based on the assumption that the hydrolysis of 1 kg of feed lipid is assumed to give 150 g glycerol, 800 g Ld and 50 g of other compounds (Reichl and Baldwin, 1975), assuming that the total input from the Ld pool is 80% of the lipid intake. Therefore, $Y_{Sc,InLd} = \frac{0.15}{0.80} = 0.19\,\text{kg Sc / kg Ld}$. The yield of Sc from Fd ($Y_{Sc,FdSc}$) and the yield of Sc from Si ($Y_{Sc,SiSc}$) are set at unity (Dijkstra *et al.*, 1996), assuming no loss in the respective transactions.

There are four outputs from this pool: Sc is required for microbial growth with ammonia (Am) ($U_{Sc,AmMi}$: equation 3.18f) and with soluble protein (Ps) ($U_{Sc,PsMi}$: equation 3.18g), for microbial non-growth purposes (U_{Sc,ScV_n}: equation 3.18h) and the outflow of Sc with the fluid phase ($U_{Sc,ScEx}$: equation 3.18i). The requirement factors, $R_{Sc,AmMi}$ and $R_{Sc,PsMi}$, were calculated by Dijkstra *et al.* (1996) from Reichl and Baldwin

(1975) as 5.28 gSc/gAm and 3.15 gSc/gPs, respectively. The utilization of Sc by microbes for non-growth purposes ($U_{Sc,ScVa}$: equation 3.21h) is divided into a maintenance requirement part and an energy spilling part due to a lack of Ps. The rate at which Sc is used for maintenance ($v^{(1)}_{ScVa}$) is set at 2.4 g Sc/ g Mi/day (Russel and Baldwin, 1979). The maximum utilization rate of Sc not related to growth or maintenance ($v^{(2)}_{ScVa}$) is calculated from the maximal potential use of Sc for growth with Ps, minus the maximal use for growth with Am. Therefore, $v^{(2)}_{ScVa} = 12$ g Sc/g Mi/day. The affinity and inhibition constants, $M_{Sc,ScVa}$ and $J_{Ps,ScVa}$, were assigned values of 12.08 and 0.230 g/L (Dijkstra *et al.*, 1996).

$$\frac{dQ_{Sc}}{dt} = \underbrace{P_{Sc,InSc}}_{\text{diet input}} + \underbrace{P_{Sc,InLd}}_{\text{Input from Ld}} + \underbrace{P_{Sc,FdSc}}_{\text{hydrolysis from Fd}} + \underbrace{P_{Sc,SiSc}}_{\text{hydrolysis from Si}} - \underbrace{U_{Sc,AmMi}}_{\substack{\text{microbial growth} \\ \text{with ammonia}}} - \underbrace{U_{Sc,PsMi}}_{\substack{\text{microbial growth} \\ \text{with soluble protein}}} - \underbrace{U_{Sc,ScVa}}_{\text{microbial non-growth}} - \underbrace{U_{Sc,ScEx}}_{\text{outflow with fluid}} \quad (3.18)$$

Forage Undegradable Protein

Forage unavailable unescapable undegradable protein, Q_{PuUaUe} (g).

There is one input to this pool from the food (D_{Pu_1} or $P^{*}_{Pu,InPu}$: equation 3.19a) and the outflow is to the available unescapable undegradable protein pool ($U_{Pu,UaAv}$: equation 3.19b).

$$\frac{dQ_{PuUaUe}}{dt} = \underbrace{P^{*}_{Pu,InPu}}_{\text{diet input}} - \underbrace{U_{Pu,UaAv}}_{\substack{\text{flow to available} \\ \text{unescapable undegradable protein}}} \quad (3.19)$$

Forage available unescapable undegradable protein, Q_{PuAvUe} (g).

There is one input to this pool from the unavailable unescapable undegradable protein pool ($P_{Pu,UaAv}$: equation 3.20a) and the outflow is to the available escapable undegradable protein pool ($U_{Pu,UeEs}$: equation 3.20b).

$$\frac{dQ_{PuAvUe}}{dt} = \underbrace{P_{Pu,\,UaAv}}_{\substack{\text{inflow from unavailable}\\ \text{unescapable undegradable protein}}} - \underbrace{U_{Pu,UeEs}}_{\substack{\text{outflow to available escapable}\\ \text{undegradable protein}}} \tag{3.20}$$

Forage available escapable undegradable protein, $Q_{Pu\,AvEs}$ (g).

There is one input to this pool from the available unescapable undegradable protein pool ($P_{Pu,UeEs}$: equation 3.21a) and the outflow is out of the rumen ($U_{Pu,EsEx}$: equation 3.21b).

$$\frac{dQ_{PuAvEs}}{dt} = \underbrace{P_{Pu,UeEs}}_{\substack{\text{inflow from available}\\ \text{unescapable undegradable protein}}} - \underbrace{U_{Pu,EsEx}}_{\text{outflow}} \tag{3.21}$$

Non-Forage undegradable protein pool, Q_{Pu} (g).

There is one input to this pool from the food (D_{Pu_2} or $P_{Pu,InPu}$: equation 3.22b) and the outflow is with the solid material ($U_{Pu,PuEx}$: equation 3.22c).

$$\frac{dQ_{Pu}}{dt} = \underbrace{P_{Pu,InPu}}_{\text{diet input}} - \underbrace{U_{Pu,PuEx}}_{\text{outflow with solid material}} \tag{3.22}$$

Forage Degradable Protein

Forage unavailable unescapable degradable protein, Q_{PdUaUe} (g).

There is one input to this pool from the food (D_{Pd_1} or $P^{*}_{Pd,InPd}$: equation 3.23a) and the outflow is to the available unescapable degradable protein pool ($U_{Pd,UaAv}$: equation 3.23b).

$$\frac{dQ_{PdUaUe}}{dt} = \underbrace{P^{*}_{Pd,InFd}}_{\text{diet input}} - \underbrace{U_{Pd,UaAv}}_{\substack{\text{flow to available}\\ \text{unescapable degradable protein}}} \tag{3.23}$$

Forage available unescapable degradable protein, Q_{PdAvUe} (g).

There is one input to this pool from the unavailable unescapable degradable protein pool ($P_{Pd,UaAv}$: equation 3.24a). There are two outputs from this pool: the hydrolysis of Pd to Ps by the rumen microorganisms ($U_{Pd,UeSc}$: equation 3.24b) and the outflow to the

available escapable degradable protein pool ($U_{Pd,UeEs}$: equation 3.24c). The hydrolysis rate (k_{PdPs_1}) can be calculated from the relevant experimental input data chosen and C_{refMi} is a reference value (12.5 g/L) of the concentration of microbial DM in the rumen based on data reported by Robinson *et al.* (1987).

$$\frac{dQ_{PdAvUe}}{dt} = \underbrace{P_{Pd,UeAv}}_{\text{inflow from unavailable unescapable degradable protein}} - \underbrace{U_{Pd,UeSc}}_{\text{hydrolysis to Ps}} - \underbrace{U_{Pd,UeEs}}_{\text{outflow to available escapable degradable protein}} \quad (3.24)$$

Forage available escapable degradable protein, Q_{PdAvEs} (g).

There is one input to the available escapable degradable protein pool ($U_{Pd,UeEs}$: equation 3.25a). There are two outputs from this pool: the hydrolysis of Pd to Ps by the rumen microorganisms ($U_{Pd,EsSc}$: equation 3.25b) and the outflow to the hindgut ($U_{Pd,EsEx}$: equation 3.25c). The hydrolysis rate (k_{PdPs_1}) can be calculated from the relevant experimental input data chosen and C_{refMi} is a reference value (12.5 g/L) of the concentration of microbial DM in the rumen based on data reported by Robinson *et al.* (1987).

$$\frac{dQ_{PdAvEs}}{dt} = \underbrace{P_{Pd,UeEs}}_{\text{inflow from available unescapable degradable protein}} - \underbrace{U_{Pd,EsSc}}_{\text{hydrolysis to Ps}} - \underbrace{U_{Pd,EsEx}}_{\text{outflow}} \quad (3.25)$$

Non-Forage insoluble degradable protein pool, Q_{Pd} (g).

There is one input to this pool from the food ($P_{Pd,InPd}$: equation 3.26b) and two outputs: the hydrolysis of Pd to Ps by proteolytic enzymes produced by rumen microorganisms ($U_{Pd,PdPs}$: equation 3.26c) and the washout with the solid material ($U_{Pd,PdEx}$: equation 3.26d). The hydrolysis rate (k_{PdPs_2}) can be calculated from the relevant experimental input data chosen and C_{refMi} is a reference value (12.5 g/L) of the concentration of microbial DM in the rumen based on data reported by Robinson *et al.* (1987).

$$\frac{dQ_{Pd}}{dt} = \underbrace{P_{Pd,InPd}}_{\text{diet input}} - \underbrace{U_{Pd,PdPs}}_{\text{hydrolysis to Ps}} - \underbrace{U_{Pd,PdEx}}_{\text{outflow with solid material}} \quad (3.26)$$

($R_{Am,PsMi}$ = 38 mg Am/g Ps) was calculated from Reichl and Baldwin (1975). The average maximum relative growth in vitro of five microbial bacterial species was 0.8/h, when preformed monomers were supplied (Russel and Baldwin, 1979) and the growth with Am as the sole N source is set at half this maximum growth rate (Maeng *et al.*, 1976). These values were used to calculate the maximum uptake rates of Ps and Am (v_{PsMi} = 10.464 g Ps/g Mi/day; v_{AmMi} = 1.224 g Am/g Mi/day). The affinity constant for Am uptake ($M_{Am,AmMi}$) is set at 26 mg/L (Hespell and Bryant, 1979). The affinity constant for Ps uptake for microbial growth ($M_{Ps,PsMi}$ = 230 mg/L) was calculated by Dijkstra *et al.* (1996) from bacterial growth data as affected by amino acid availability (Russel *et al.* 1983). The inhibition constant ($J_{Ps,AmMi}$) is estimated to be equal to $M_{Ps,PsMi}$. The values of the affinity constants, $M_{Sc,AmMi}$ and $M_{Sc,PsMi}$ are equal, because the ratio of Am versus Ps utilization by bacteria is not affected by the availability of carbohydrate (Russel *et al.*, 1983), and is calculated below. The voluntary DMI of unsupplemented sugarcane and the passage rates, k_{FIEx} and k_{SoEx}, are 16g DM/kg LW, 1.921/day and 0.72/day, respectively (Leng and Preston, 1976; Owens and Goetsch, 1986). The rumen protozoal biomass with sugarcane diets is up to 40% of total microbial biomass (Leng and Preston, 1976). The fractional outflow of protozoa is taken to be $\frac{1}{2}k_{SoEx}$ (Faichney *et al.*, 1989). The attached bacteria are assumed to be 75% of the total (Forsberg and Lam, 1976; Craig *et al.*, 1987) and are washed out with the solids, whilst the non-attached bacteria flow out of the rumen with the liquid phase. Hence, the fractional outflow rate of microorganisms (which in steady state is equal to the net fractional growth rate) is 0.0315/h by Dijkstra *et al.* (1996). The affinity constants $M_{Sc,AmMi}$ and $M_{Sc,PsMi}$ (= 12.08 g/L) were then calculated using this net growth rate and equations 3.30d and 3.31f, assuming for a reference diet that C_{Am} = 26 mg/L (Leng and Preston, 1976), C_{Ps} = 6 mg/L (Wright and Hungate, 1967) and C_{Sc} = 600 mg/L (Clapperton and Czerkawski, 1969). Finally, $M_{Ld,PsMi}$ and $M_{Ld,AmMi}$ are assigned low values (1.0 mg/L) in order to ensure that microbial Ld uptake is smaller than the amount available. The fractional absorption rate in equation 3.28g (k_{AmAb}) is allocated the value of 12/day (Siddons *et al.*, 1985).

$$\frac{dQ_{Am}}{dt} = \underbrace{P_{Am,InAm}}_{\text{diet input}} + \underbrace{P_{Am,UrAm}}_{\text{transport of urea}} + \underbrace{P_{Am,PsAm}}_{\text{fermentation of Ps}} - \underbrace{U_{Am,PsMi}}_{\text{microbial growth}} - \underbrace{U_{Am,AmMi}}_{\text{microbial growth}} - \underbrace{U_{Am,AmAb}}_{\text{absorption across rumen wall}} - \underbrace{U_{Am,AmBx}}_{\text{outflow with fluid}} \quad (3.28)$$

there are numerous factors that affect microbial protein synthesis and efficiency: for example, nitrogen concentrations in the rumen, nitrogen sources, rates of nitrogen and carbohydrate degradation, carbohydrate sources, the ratio of forage to concentrate in the diet, dry matter intake, synchronisation of nitrogen and simultaneous release of energy. Other factors, such as the rates of solid and liquid passage, must also be considered (Hoover and Stokes, 1991). If nitrogen is not limiting, then the yield of microbial protein can be assumed to be energy dependent (Hume *et al.*, 1970). However, this is not the case for ruminants on arid and semi-arid rangelands.

Gomes *et al.* (1994) found that the supplementation of barley straw with 15.5 or 31% of a 1.2 to 1.0 mixture of maize and barley increased microbial protein synthesis from 12.8 to 14.1 and 17.5g N/kg DOMI. Their study implied that with low quality straw, supplementation of the diet with 31% concentrate increased both voluntary intake and microbial protein synthesis per unit of DOMI, apparently due to increased liquid and solid outflow rates when starch was included in the diet. This study also indicated that the addition of readily fermentable carbohydrates into the rumen increased microbial growth. However, increasing the energy level beyond an optimal level was shown not to increase microbial growth further (McAllan *et al.*, 1994).

The simulation model of rumen function described will be used to examine the effects of different nitrogen supplements on a low-quality roughage (Autumn grass*) diet. The composition and degradation rates of the diet were estimated and given in Table 3.3. The model was used to examine the effects of supplementing the roughage diet with a constant amount of nitrogen: 338g/day of Cottonseed Meal (CM1: 23g N), 675g/day of Cottonseed Meal (CM2: 46g N), 100g/day of urea only (UR: 46g N), and 292g/day of a mixture of 50% Cottonseed Meal and 50% Maize Grain (CMMG: 41g N). The composition of all the supplements are also given in Table 3.3.

The roughage diet alone has a slow rate of rumen digestion and therefore the residence time of particulate matter in the rumen will be long and the rate of physical breakdown of particles would play a major role in regulating rumen clearance. Supplementing the roughage diet with the CM1 (23g N) and CM2 (46g N) supplements was done to examine the effects of an increase in the nitrogen content in the rumen on absorbed nutrients from the rumen. It has been shown experimentally that supplementing with urea alone does not result in a significant increase of the intake and nitrogen content of the rumen liquor, however, supplementing with both a carbohydrate source and nitrogen source has a huge effect on the N content of the

*mature grass harvested at the end of the rainy season

rumen liquor, amount of microbial DM in the rumen and consequently, on the absorbed nutrients (Elliott *et al.*, 1965). It will then be of particular interest to compare the absorbed nutrients for animals given the UR (46g N) supplement and the CM2 (46g N) supplement.

A sensitivity analysis of some of the key parameters identified in the model development stage was also examined. In particular, the lack of soluble protein in the rumen limits microbial growth (Hennessy *et al.*, 2000) and it will be of particular interest to examine the effects of changing the amount of soluble protein recycled to the rumen via the saliva (Psal) on the nitrogen content of the rumen and the absorbed amino acids. The rates at which ammonia (v_{AmMi}) and soluble protein (v_{PsMi}) are incorporated into microbial biomass are essential processes that limit microbial growth and, consequently, the degradation of organic matter. These rates and the rate of ammonia absorption across the rumen wall (k_{AmAb}) will also be examined.

3.7 Model results and discussion

The effects of supplementing a low quality roughage diet with four supplements on nutrient intake, rumen nutrient concentrations and the absorption of nutrients is shown in Table 3.4. To obtain the initial values in Table 3.4 the model was run for 20 days ($\Delta t = 1$ day) with a 50% roughage and 50% concentrate as continuous input *ad libitum*.

Using the 50% roughage and 50% concentrate diet as input, the model simulated a decrease in the absorption of amino acids, glucose, long chain fatty acids and volatile fatty acids. This was expected because of the low nitrogen content of the roughage diet (31.7g/day). When the roughage diet was supplemented with CM1 (23g N), the DM intake of roughage increased substantially (by about 909g) from the DM intake on the roughage diet only. When the roughage diet was supplemented with CM2 (46g N), the DM intake also increased but only by 379g more than the DM intake on the CM1 (23g N) supplement (Figure 3.4a). Nolte *et al.* (2000) demonstrated that digestible OM intake decreased linearly with the addition of increasing proportions of urea. This could be due to the decrease in NDF digestibility observed at higher urea levels because of the shorter retention time of the digesta. They also showed that microbial protein increased between 25% and 50% urea inclusion, after which a plateau was reached, even though NH_3-N concentrations in the rumen kept increasing. Increasing the quantity of the Cottonseed Meal supplement increased the concentration of ammonia and microbial DM in the rumen, which increased the degradation of neutral detergent fibre (NDF) and starch and soluble sugars.

Table 3.3: Feed Composition and hydrolysis rates of feed components of a roughage basal diet and supplements.

Feed	Feed Composition (g/kg)										Hydrolysis rate (/day)		
	Fd	Fu	Sc	Si	Ps	Pd	Pu	Am	Ld	Ash	Fd	Pd	Si
Autumn grass	536	204	160	0	7	25	10	0	8	5	0.09	0.00	0.84
Cottonseed Meal	263	135	47	13	57	359	8	0	50	0	1.56	1.82	3.00
50% Cottonseed Meal + 50% Maize Grain	181	73	125	273	37	211	6	0	42	0	1.39	1.98	1.33
Urea	0	0	0	0	0	0	0	549	0	0	0.00	0.00	0.00

Fd = Degradable fiber, Fu = Undegradable fiber, Sc = Soluble sugars and starch, Si = Insoluble starch and sugars, Ps = Soluble protein, Pd = Degradable protein, Pu = Undegradable protein, Am = Ammonia, Ash, Ld = Fat.

Table 3.4: Inputs and simulated nutrient profile of a low quality roughage diet and four supplements: 338g/kg of Cottonseed Meal (CM1: 23g N), 675g/kg of Cottonseed Meal (CM2: 46g N), 100g/kg of urea only (UR: 46g N), 292g/kg of a mixture of 50% Cottonseed Meal and 50% Maize Grain (CMMG: 23g N).

	Initial	Autumn Grass	CM1 (23g N)	CM2 (46g N)	CMMG (23g N)	UR (46g N)
General						
Days	0	50	50	50	50	50
Weight (kg)	201	193	221	235	233	224
Gross Energy (MJ/day)	149.65	65.58	96.60	110.64	112.62	114.24
Rumen volume (Litres)	58.9	59.1	63.2	65.8	65.5	63.7
Intake:						
Roughage DM (g/day)	6515	4720	5629	6008	5654	7061
Supplement (gDM/day)	0.0	0.0	338	676	568	100
Starch and soluble sugars (g/day)	34.2	755.2	1165.1	621.9	805.7	1129.8
NDF (g/day)	5563.5	3493.0	3202.7	3418.7	2809.9	3784.8
Total N (g/day)	94.95	31.72	232.84**	269.99**	156.43**	450.2
Non-Urea N (g/day)	94.95	31.72	209.84	223.99	133.43	0.00*
Long chain fatty acids (g/day)	29.74	37.80	163.23	174.24	141.35	56.49
Rumen fluid concentration of:						
Ammonia (mg/L)	21.4	1.7	2.2	12.5	15.2	20.0
Volatile fatty acid (mmole/L)	55.7	27.7	38.3	40.2	42.5	48.1
Microbial DM (g/L)	32.9	8.4	17.5	22.2	19.8	16.7
Absorption of						
Amino acids (mole/day)	7.0	2.1	4.1	5.4	5.0	3.9
Glucose (mole/day)	3.1	1.9	1.7	1.7	1.9	2.2
Long chain fatty acids (mole/day)	0.2	0.06	0.1	0.1	0.1	0.09
Volatile fatty acids (mole/day)	36.2	18.1	26.7	29.2	30.7	33.8
Energy (MJ/day)	79.07	35.00	51.31	58.8	59.79	60.40

* Roughage non-urea nitrogen ** Includes a constant amount of urea across the rumen wall (Dijkstra *et al.*, 1996)

The DM intake of roughage on the CMMG supplement (23g N) was almost the same as the DM intake of roughage on the CM1 supplement (23g N), however, the urea supplement (UR: 46g N) increased the DM intake of roughages substantially (by 2341g). Interestingly, the model predicted that the absorbed nutrients on the UR (46g N) supplement was similar to the absorbed energy with the CM1 (23g N) supplement.

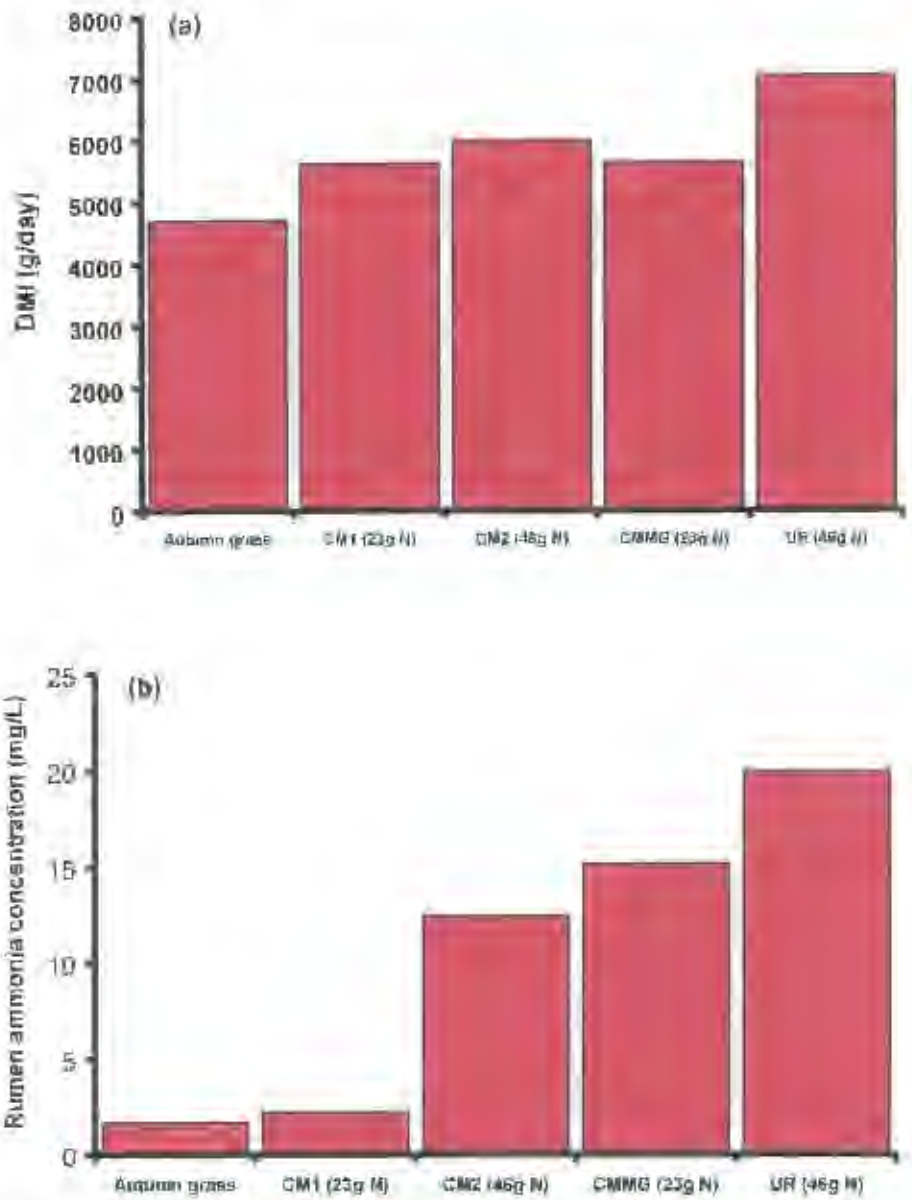


Figure 3.4: The effect of a roughage diet alone, the CM1 (23g N), CM2 (46g N),CMMG (23g N) and UR (46g N) supplement on (a) the dry matter intake, g/day, and (b) rumen ammonia concentration, mg/L.

It has been shown experimentally that supplementing a low quality roughage diet with nitrogen and carbohydrate has a greater effect on the N content of the rumen liquor than supplementing with nitrogen or carbohydrate separately (Elliott *et al.*, 1965). The

model predicts that the effect of a N supplement which includes a carbohydrate source has a large effect on the ammonia concentration and that supplementing with urea only has the largest effect on the rumen ammonia concentration (Figure 3.4b).

Sensitivity Analysis

Table 3.6 shows the effects of increasing nine parameters (v_{AmMi}, v_{PsMi}, k_{AmAb}, k_{FlEx}, k_{SoEx}, $k_{Pd,CaNr}$, $k_{Fd,CaNr}$, $Y_{Am,UrAm}$, Psal) independently by 50% of their estimated value on the absorbed nutrients and key variables. A description and values of these parameters are given in Table 3.5. The metric used for parameter sensitivity is the absolute difference between the initial value of the variable and its value after 10 days of simulation time. The sensitivity analysis was done on the roughage diet with the CM1 (23g N) and CM2 (46g N) supplements.

The effect of changing the parameters had a greater effect on the absorption of long chain fatty acid, amino acids and volatile fatty acids when supplemented with CM1 (23g N) than when the roughage diet was supplemented with CM2 (46g N) (Table 3.6). However, there was very little effect of changing the parameters on the absorbed glucose. Figure 3.5 shows the effect of changing the parameters on DM intake.

Table 3.5: Description of parameters used in the sensitivity analysis

Parameter	Description of the parameter	Value	Units
v_{AmMi}	Rate of ammonia incorporation in microbial biomass	1.224	per day
v_{PsMi}	Rate of soluble protein incorporation in microbial biomass	10.464	per day
k_{AmAb}	Rate at which ammonia is absorbed across the rumen wall	12	per day
k_{FlEx}	Rate at which fluid flows out of the rumen	1.92	per day
k_{SoEx}	Rate at which solid material flows out of the rumen	0.72	per day
$k_{Fd,CaNr}$	Rate at which Fd entering the rumen becomes available for hydrolysis	12	per day
$k_{Pd,CaNr}$	Rate at which Pd entering the rumen becomes available for hydrolysis	12	per day
$Y_{Am,UrAm}$	Yield of rumen ammonia from plasma urea across the rumen wall	0.971	g Am/g Ur
Psal	Amount of soluble protein in saliva	5.0	g

Increasing the parameter values had a greater effect on the DM intake (Figure 3.5) and generally on the absorbed nutrients from the rumen (Table 3.6) when the basal roughage diet was supplemented with CM1 (23g N).

Table 3.6: Effect on increasing nine parameters by 50% of their estimated values on the absorbed nutrients from the rumen.

Parameter	A_{GI}		A_{LI}		A_{As}		A_{VF}	
	CM1	CM2	CM1	CM2	CM1	CM2	CM1	CM2
V_{AmAb}	1.535	1.529	0.105	0.078	2.987	1.949	10.710	9.147
V_{PsIR}	1.528	1.514	0.105	0.079	3.016	1.958	10.859	9.513
k_{AmAb}	1.529	1.522	0.105	0.079	3.049	1.953	10.831	9.309
k_{Fles}	1.160	1.279	0.106	0.078	3.061	1.808	13.122	10.505
k_{Gmex}	1.375	1.369	0.093	0.067	2.584	1.444	7.165	5.790
$k_{Pd,UaAv}$	1.505	1.498	0.104	0.078	2.982	1.908	10.221	8.748
$k_{Pd,UaAv}$	1.530	1.522	0.105	0.078	3.023	1.952	10.769	9.263
$Y_{Am,UrAm}$	1.531	1.518	0.104	0.079	2.977	1.944	10.578	9.185
Psal	1.530	1.520	0.105	0.079	2.992	1.951	10.672	9.232

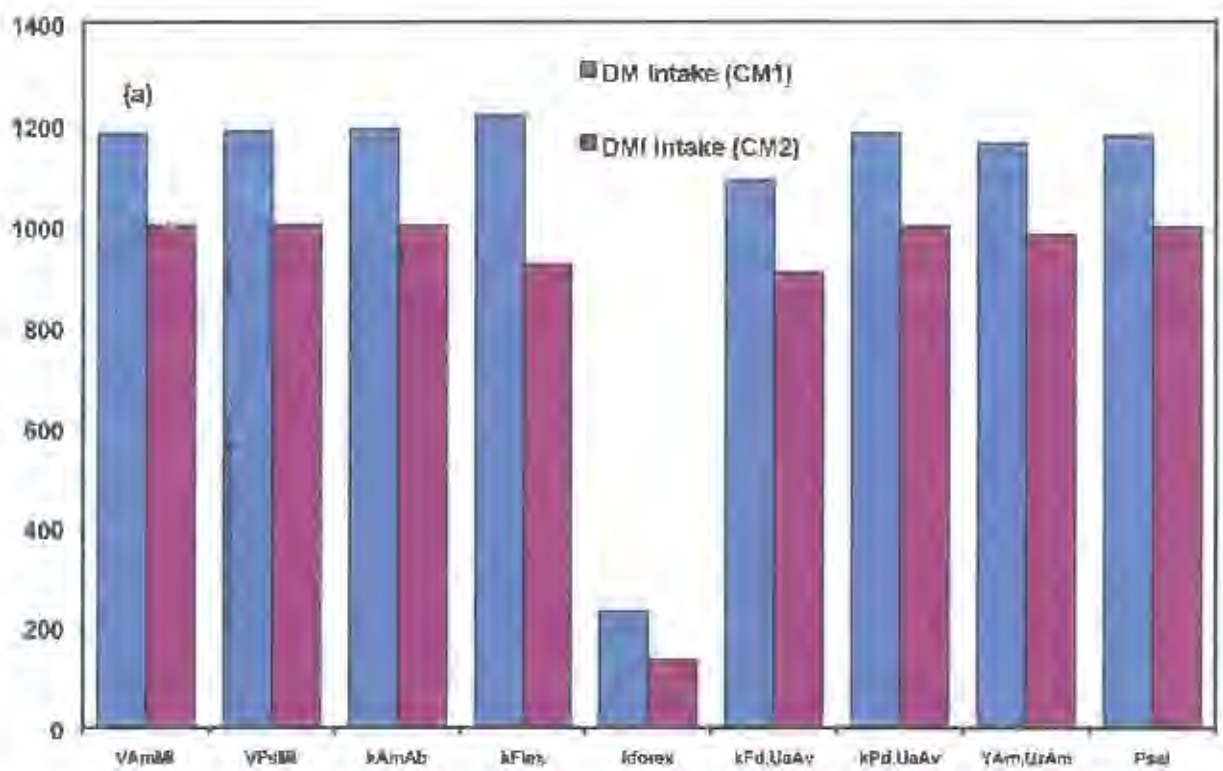


Figure 3.5: The effect of increasing nine parameters on the DM intake of growing cattle.

3.8 Conclusions

Mature fibrous forages are inadequate sources of nutrients for both the rumen microorganisms and the host animal. Therefore, their nutritional value could be dramatically improved by supplementing with key nutrients to the rumen to produce protein by the microbes and energy from VFA. A model of the digestion and absorption of low quality roughage diets was used to evaluate various supplements for growing cattle on arid and semi-arid rangeland. The objective of the model was to predict dry matter intake and absorbed nutrients for the host animal in order to indicate which supplements were most likely to improve the utilization of low quality roughage. Because of the very low nitrogen level of the roughage, a nitrogen supplement is usually added to improve the nitrogen availability in the rumen. The model shows that feeding the CMMG supplement resulted in the highest rumen ammonia concentration, however, the CM2 (46g N) supplement produced about the same amount of absorbed VFA (58.84 vs 59.79) but more amino acids were absorbed from the rumen (5.36 vs 5.04). Also, even though the urea supplement (UR) gave the largest increase of roughage intake, the absorbed nutrients with the UR supplement was not much different to the absorbed nutrients on the other supplements. The effect of urea-N recycling to the rumen via the saliva and diffusion across the rumen wall will be examined in chapter 7.

APPENDIX 3.1

MODEL EQUATIONS

$$DMItot = min[BiDMI, DMI]. \tag{3.1}$$

$$DMI = kRuEx \times cDMRu \times RVol. \tag{3.2}$$

$$Yrvol = 3.0 \times Xwt + (1 - 3.0) \times (Xwt)^2 \tag{3.2a}$$

where $Yrvol = \frac{Rvol}{Rvolmat}$ (3.2b)

$$Xwt = \frac{Weight}{Wmax} \tag{3.2c}$$

$$BiDMI = Bbite \times maxbite \times AdjDM. \tag{3.3}$$

$$Arc = 9.1 \times Weight^{0.29} \times Wmax^{0.07}. \tag{3.3a}$$

$$Bbite = Abite \times Arc. \tag{3.3b}$$

$$AdjDM = \frac{\left(\frac{shoot}{kshoot}\right)^3}{1+\left(\frac{shoot}{kshoot}\right)^3}. \tag{3.3c}$$

$$A_{ac} = FrAc \times A_{Va} \tag{3.4a}$$

$$A_{pr} = FrPr \times A_{Va} \tag{3.4b}$$

$$A_{bu} = FrBu \times A_{Va} \tag{3.4c}$$

$$DMI_j = frDMI_j \times DMI_{tot}. \tag{3.5}$$

$$DMI_{tot} = food + bint \times days \tag{3.6}$$

$$D_i = C_i \times DMI_j \qquad i = 1 \text{ to } 10, \quad j = 1 \text{ to } 2. \tag{3.7}$$

$$D_{Fd} = D_{Fd_1} + D_{Fd_2} \tag{3.8}$$

Forage Undegradable Fibre

Forage unavailable unescapable undegradable fibre, Q_{FuUaUe} (g).

Input: $$P^{*}_{Fu,InFu} = D_{Fu_1} \tag{3.9a}$$

Output: $$U_{Fu,UaAv} = k_{Fu,UaAv} \times Q_{FuUaUe} \tag{3.9b}$$

Differential Equation:

$$\frac{dQ_{FuUaUe}}{dt} = P^{*}_{Fu,InFu} - U_{Fu,UaAv} \tag{3.9}$$

Forage available unescapable undegradable fibre, Q_{FuAvUe} (g).

Input: $$P_{Fu,UaAv} = U_{Fu,UaAv} \tag{3.10a}$$

Output: $$U_{Fu,UeEs} = k_{Fu,UeEs} \times Q_{FuAvUe} \tag{3.10b}$$

Differential Equation:

$$\frac{dQ_{FuAvUe}}{dt} = P_{Fu,UaAv} - U_{Fu,UeEs} \tag{3.10}$$

Forage available escapable undegradable fibre, Q_{FuAvEs} (g).

Input: $$P_{Fu,UeEs} = U_{Fu,UeEs} \tag{3.11a}$$

Output: $$U_{Fu,EsEx} = k_{ForEx} \times Q_{FuAvEs} \tag{3.11b}$$

Differential Equation:

$$\frac{dQ_{FuAvEs}}{dt} = P_{Fu,UeEs} - U_{Fu,EsEx} \tag{3.11}$$

Non-Forage Undegradable Fibre, Q_{Fu} (g).

Concentration: $$C_{Fu} = \frac{Q_{Fu}}{Rvol} \tag{3.12a}$$

Input: $$P_{Fu,InFu} = D_{Fu_2} \tag{3.12b}$$

Output: $$U_{Fu,FuEx} = k_{SoEx} \times Q_{Fu} \tag{3.12c}$$

Differential Equation:

$$\frac{dQ_{Fu}}{dt} = P_{Fu,InFu} - U_{Fu,FuEx} \tag{3.12}$$

Forage Degradable Fibre

Forage unavailable unescapable degradable fibre, Q_{FdUaUe} (g).

Input: $$P^{*}_{Fd,InFd} = D_{Fd_1} \quad (3.13a)$$

Output: $$U_{Fd,UaAv} = k_{Fd,UaAv} \times Q_{FdUaUe} \quad (3.13b)$$

Differential Equation:

$$\frac{dQ_{FdUaUe}}{dt} = P^{*}_{Fd,InFd} - U_{Fd,UaAv} \quad (3.13)$$

Forage available unescapable degradable fibre, Q_{FdAvUe} (g).

Input: $$P_{Fd,UaAv} = U_{Fd,UaAv} \quad (3.14a)$$

Output: $$U_{Fd,UeSc} = k_{FdSc_1} \times (\frac{C_{Mi}}{C_{refMi}}) \times Q_{FdAvUe} \quad (3.14b)$$

$$U_{Fd,UeEs} = k_{Fd,UeEs} \times Q_{FdAvUe} \quad (3.14c)$$

Differential Equation:

$$\frac{dQ_{FdAvUe}}{dt} = P_{Fd,UaAv} - U_{Fd,UeSc} - U_{Fd,UeEs} \quad (3.14)$$

Forage available escapable degradable fibre, Q_{FdAvEs} (g).

Input: $$P_{Fd,UeEs} = U_{Fd,UeEs} \quad (3.15a)$$

Output: $$U_{Fd,EsSc} = k_{FdSc_1} \times (\frac{C_{Mi}}{C_{refMi}}) \times Q_{FdAvEs} \quad (3.15b)$$

$$U_{Fd,EsEx} = k_{ForEx} \times Q_{FdAvEs} \quad (3.15c)$$

Differential Equation:

$$\frac{dQ_{FdAvEs}}{dt} = P_{Fd,UeEs} - U_{Fd,EsSc} - U_{Fd,EsEx} \quad (3.15)$$

Non-Forage Degradable fibre pool, Q_{Fd} (g).

Concentration: $$C_{Fd} = \frac{Q_{Fd}}{Rvol} \quad (3.16a)$$

Input: $$P_{Fd,InFd} = D_{Fd_2} \quad (3.16b)$$

Output: $$U_{Fd,FdSc} = k_{FdSc_2} \times Q_{Fd} \times (\frac{C_{Mi}}{C_{refMi}}) \quad (3.16c)$$

$$U_{Fd,FdEx} = k_{SoEx} \times Q_{Fd} \quad (3.16d)$$

Differential Equation:

$$\frac{dQ_{Fd}}{dt} = P_{Pd,InFd} - U_{Fd,FdSc} - U_{Fd,FdEx} \tag{3.16}$$

Insoluble starch pool, Q_{Si} (g).

Concentration: $$C_{Si} = \frac{Q_{Si}}{Rvol} \tag{3.17a}$$

Input: $$P_{Si,InSi} = D_{Si_1} + D_{Si_2} \tag{3.17b}$$

Output: $$U_{Si,SiSc} = k_{SiSc_2} \times Q_{Si} \times (\frac{C_{Mi}}{C_{refMi}}) \tag{3.17c}$$

$$U_{Si,SiEx} = k_{SoEx} \times Q_{Si} \tag{3.17d}$$

Differential Equation:

$$\frac{dQ_{Si}}{dt} = P_{Si,InSi} - U_{Si,SiSc} - U_{Si,SiEx} \tag{3.17}$$

Soluble starch and sugars pool, Q_{Sc} (g).

Concentration: $$C_{Sc} = \frac{Q_{Sc}}{Rvol} \tag{3.18a}$$

Input: $$P_{Sc,InSc} = D_{Sc_1} + D_{Sc_2} \tag{3.18b}$$

$$P_{Sc,InLd} = Y_{Sc,InLd} \times (D_{Ld_1} + D_{Ld_2}) \tag{3.18c}$$

$$P_{Sc,FdSc} = Y_{Sc,FdSc} \times (U_{Fd,FdSc} + U_{Fd,UeSc} + U_{Fd,EaSc}) \tag{3.18d}$$

$$P_{Sc,SiSc} = Y_{Sc,SiSc} \times U_{Si,SiSc} \tag{3.18e}$$

Output: $$U_{Sc,AmMi} = R_{Sc,AmMi} \times U_{Am,AmMi} \tag{3.18f}$$

$$U_{Sc,PsMi} = R_{Sc,PsMi} \times U_{Ps,PsMi} \tag{3.18g}$$

$$U_{Sc,ScVa} = V^{(1)}_{ScVa} \times Q_{Mi} + \frac{V^{(2)}_{ScVa} \times Q_{Mi}}{1 + (\frac{M_{Sc,ScVa}}{C_{Sc}}) + (\frac{C_{Ps}}{J_{Ps,ScVa}})} \tag{3.18h}$$

$$U_{Sc,ScEx} = k_{FlEx} \times Q_{Sc} \tag{3.18i}$$

Differential Equation:

$$\frac{dQ_{Sc}}{dt} = P_{Sc,InSc} + P_{Sc,InLd} + P_{Sc,FdSc} + P_{Sc,SiSc} - U_{Sc,AmMi} - U_{Sc,PsMi} - U_{Sc,ScVa} - U_{Sc,ScEx} \tag{3.18}$$

Forage Undegradable Protein

Forage unavailable unescapable undegradable protein, Q_{PuUaUe} (g).

Input: $$P^{*}_{Pu,InPu} = D_{Pu_1} \tag{3.19a}$$

Output: $$U_{Pu,UaAv} = k_{Pu,UaAv} \times Q_{PuUaUe} \tag{3.19b}$$

Differential Equation:

$$\frac{dQ_{PuUaUe}}{dt} = P^{*}_{Pu,InPu} - U_{Pu,UaAv} \tag{3.19}$$

Forage available unescapable undegradable protein, Q_{PuAvUe} (g).

Input: $$P_{Pu,UaAv} = U_{Pu,UaAv} \tag{3.20a}$$

Output: $$U_{Pu,UeEs} = k_{Pu,UeEs} \times Q_{PuAvUe} \tag{3.20b}$$

Differential Equation:

$$\frac{dQ_{PuAvUe}}{dt} = P_{Pu,UaAv} - U_{Pu,UeEs} \tag{3.20}$$

Forage available escapable undegradable protein, Q_{PuAvEs} (g).

Input: $$P_{Pu,UeEs} = U_{Pu,UeEs} \tag{3.21a}$$

Output: $$U_{Pu,EsEx} = k_{ForEx} \times Q_{PuAvEs} \tag{3.21b}$$

Differential Equation:

$$\frac{dQ_{PuAvEs}}{dt} = P_{Pu,UeEs} - U_{Pu,EsEx} \tag{3.21}$$

Non-Forage undegradable protein, Q_{Pu} (g).

Concentration: $$C_{Pu} = \frac{Q_{Pu}}{Rvol} \tag{3.22a}$$

Input: $$P_{Pu,InPu} = D_{Pu_2} \tag{3.22b}$$

Output: $$U_{Pu,PuEx} = k_{SoEx} \times Q_{Pu} \tag{3.22c}$$

Differential Equation:

$$\frac{dQ_{Pu}}{dt} = P_{Pu,InPu} - U_{Pu,PuEx} \tag{3.22}$$

Forage Degradable Protein

Forage unavailable unescapable degradable protein, Q_{PdUaUe} (g).

Input: $$P^{*}_{Pd,InPd} = D_{Pd_1} \tag{3.23a}$$

Output: $$U_{Pd,UaAv} = k_{Pd,UaAv} \times Q_{PdUaUe} \tag{3.23b}$$

Differential Equation:

$$\frac{dQ_{PdUaUe}}{dt} = P^{*}_{Pd,InPd} - U_{Pd,UaAv} \tag{3.23}$$

Forage available unescapable degradable protein, Q_{PdAvUe} (g).

Input: $$P_{Pd,UaAv} = U_{Pd,UaAv} \tag{3.24a}$$

Output: $$U_{Pd,UeSc} = k_{PdPs_1} \times (\frac{C_{Mi}}{C_{refMi}}) \times Q_{PdAvUe} \tag{3.24b}$$

$$U_{Pd,UeEs} = k_{Pd,UeEs} \times Q_{PdAvUe} \tag{3.24c}$$

Differential Equation:

$$\frac{dQ_{PdAvUe}}{dt} = P_{Pd,UaAv} - U_{Pd,UeSc} - U_{Pd,UeEs} \tag{3.24}$$

Forage available escapable degradable protein, $Q_{Pd,AvEs}$ (g).

Input: $$P_{Pd,UeEs} = U_{Pd,UeEs} \tag{3.25a}$$

Output: $$U_{Pd,EsSc} = k_{PdPs_1} \times (\frac{C_{Mi}}{C_{refMi}}) \times Q_{PdAvEs} \tag{3.25b}$$

$$U_{Pd,EsEx} = k_{ForEx} \times Q_{PdAvEs} \tag{3.25c}$$

Differential Equation:

$$\frac{dQ_{PdAvEs}}{dt} = P_{Pd,UeEs} - U_{Pd,EsSc} - U_{Pd,EsEx} \tag{3.25}$$

Non-Forage insoluble degradable protein pool, Q_{Pd} (g).

Concentration: $$C_{Pd} = \frac{Q_{Pd}}{Rvol} \tag{3.26a}$$

Input: $$P_{Pd,InPd} = D_{Pd_2} \tag{3.26b}$$

Output: $$U_{Pd,PdSc} = k_{PdPs_2} \times Q_{Pd} \times (\frac{C_{Mi}}{C_{refMi}}) \tag{3.26c}$$

$$U_{Pd,PdEx} = k_{SoEx} \times Q_{Pd} \tag{3.26d}$$

Differential Equation:

$$\frac{dQ_{Pd}}{dt} = P_{Pd,InFd} - U_{Pd,PdSc} - U_{Pd,PdEx} \tag{3.26}$$

Soluble Protein Pool, Q_{Ps} (g).

Concentration: $$C_{Ps} = \frac{Q_{Ps}}{Rvol} \tag{3.27a}$$

Input: $$P_{Ps,InPs} = D_{Ps_1} + D_{Ps_2} \tag{3.27b}$$

$$P_{Ps,PdPs} = Y_{Ps,PdPs} \times (U_{Pu,PdPs} + U_{Pu,UaAv} + U_{Pu,UcEs}) \tag{3.27c}$$

Output: $$U_{Ps,PsMi} = [v_{PsMi} \times Q_{Mi}]/[1 + (\frac{M_{Ps,PsMi}}{C_{Ps}}) + (\frac{M_{Sc,PsMi}}{C_{Sc}}) + (\frac{M_{Ld,PsMi}}{C_{Ld}})] \tag{3.27d}$$

$$U_{Ps,PsAm} = [v_{PsAm} \times Q_{Mi}]/[1 + (\frac{M_{Ps,PsAm}}{C_{Ps}}) + (\frac{C_{Sc}}{J_{Sc,PsAm}})] \tag{3.27e}$$

$$U_{Ps,PsEx} = k_{FlEx} \times Q_{Ps} \tag{3.27f}$$

Differential Equation:

$$\frac{dQ_{Ps}}{dt} = P_{Ps,InPs} + P_{Ps,PdPs} - U_{Ps,PsMi} - U_{Ps,PsAm} - U_{Ps,PsEx} \tag{3.27}$$

Ammonia pool, Q_{Am} (g).

Concentration: $$C_{Am} = \frac{Q_{Am}}{Rvol} \tag{3.28a}$$

Input: $$P_{Am,InAm} = D_{Am_1} + D_{Am_2} + D_{Am_3} \tag{3.28b}$$

$$P_{Am,UrAm} = \frac{Y_{Am,UrAm} \times D_{Ni}}{1 + \frac{C_{Am}}{J_{Am,UrAm}}} \tag{3.28c}$$

where $$D_{Ni} = 0.16 \times (D_{Pu_1} + D_{Pd_1} + D_{Ps_1} + D_{Pu_2} + D_{Pd_2} + D_{Ps_2} + 0.82 \times (D_{Am_1} + D_{Am_2} + D_{Am_3})$$

$$P_{Am,PsAm} = Y_{Am,PsAm} \times U_{Ps,PsAm} \tag{3.28d}$$

Output: $$U_{Am,PsMi} = R_{Am,PsMi} \times U_{Ps,PsMi} \tag{3.28e}$$

$$U_{Am,AmMi} = [v_{AmMi} \times Q_{Mi}]/[1 + (\frac{M_{Am,AmMi}}{C_{Am}}) + (\frac{C_{Ps}}{J_{Ps,AmMi}}) + (\frac{M_{Sc,AmMi}}{C_{Sc}}) + (\frac{M_{Ld,AmMi}}{C_{Ld}})] \tag{3.28f}$$

$$U_{Am,AmAb} = k_{AmAb} \times Q_{Am} \tag{3.28g}$$

$$U_{Am,AmEx} = k_{FlEx} \times Q_{Am} \tag{3.28h}$$

Differential Equation:

$$\frac{dQ_{Am}}{dt} = P_{Am,InAm} + P_{Am,UrAm} + P_{Am,PsAm} - U_{Am,PsMi} - U_{Am,AmMi} - U_{Am,AmAb} - U_{Am,AmEx} \quad (3.28)$$

Long chain fatty acid pool, Q_{Ld} (g).

Concentration: $$C_{Ld} = \frac{Q_{Ld}}{Rvol} \quad (3.29a)$$

Input: $$P_{Ld,InLd} = D_{Ld_1} + D_{Ld_2} \quad (3.29b)$$

Output: $$U_{Ld,AmMi} = R_{Ld,AmMi} \times U_{Am,AmMi} \quad (3.29c)$$

$$U_{Ld,PsMi} = R_{Ld,PsMi} \times U_{Ps,PsMi} \quad (3.29d)$$

$$U_{Ld,LdEx} = k_{SoEx} \times Q_{Ld} \quad (3.29e)$$

Differential Equation:

$$\frac{dQ_{Ld}}{dt} = P_{Ld,InLd} - U_{Ld,AmMi} - U_{Ld,PsMi} - U_{Ld,LdEx} \quad (3.29)$$

Microbial pool, Q_{Mi}.

Concentration: $$C_{Mi} = \frac{Q_{Mi}}{Rvol} \quad (3.30a)$$

Input: $$P_{Mi,AmMi} = Y_{Mi,AmMi} \times U_{Am,AmMi} \quad (3.30b)$$

$$P_{Mi,PsMi} = Y_{Mi,PsMi} \times U_{Ps,PsMi} \quad (3.30c)$$

Outflow: $$U_{Mi,MiEx} = [0.2 \times k_{SoEx} + 0.45 \times k_{SoEx} + 0.15 \times k_{FlEx}] \times Q_{Mi} \quad (3.30d)$$

Differential Equation:

$$\frac{dQ_{Mi}}{dt} = P_{Mi,AmMi} + P_{Mi,PsMi} - U_{Mi,MiEx} \quad (3.30)$$

Rumen volatile fatty acid pool, Q_{Va} (mol).

Concentration: $$C_{Va} = \frac{Q_{Va}}{Rvol} \quad (3.31a)$$

Input: $$P_{Va,InVa} = D_{Va} \quad (3.31b)$$

$$P_{Va,AmMi} = Y_{Va,AmMi} \times U_{Sc,AmMi} \quad (3.31c)$$

$$P_{Va,PsMi} = Y_{Va,PsMi} \times U_{Sc,PsMi} \quad (3.31d)$$

$$P_{Va,ScVa} = Y_{Va,ScVa} \times U_{Sc,ScVa} \quad (3.31e)$$

$$P_{Va,PsAm} = Y_{Va,PsAm} \times U_{Ps,PsAm} \quad (3.31f)$$

Outflow:

$$U_{Va,VaAb} = k_{VaAb} \times Q_{Va} \quad (3.31g)$$

$$U_{Va,VaEx} = k_{FlEx} \times Q_{Va} \quad (3.31h)$$

Differential Equation:

$$\frac{dQ_{Va}}{dt} = P_{Va,InVa} + P_{Va,AmMi} + P_{Va,PsMi} + P_{Va,ScVa} + P_{Va,PsAm} - U_{Va,VaAb} - U_{Va,VaEx} \quad (3.31)$$

$$\begin{aligned} UDMRu = {} & U_{Pd,EsEx} + U_{Pu,EsEx} + U_{Fu,EsEx} + U_{Fd,EsEx} + U_{Pu,PuEx} + U_{Pd,PdEx} + U_{Ps,PsEx} \\ & + U_{Am,AmEx} + U_{Ld,LdEx} + U_{Fu,FuEx} + U_{Fd,FdEx} + U_{Si,SiEx} + U_{Sc,ScEx} + U_{Mi,MiEx} \\ & + U_{Am,AmAb} + \text{VFAExRu} + \text{GasAmMi} + \text{GasPsMi} + \text{GasScVa} \\ & + \text{GasPsAm} + (k_{SoEx} \times Q_{Ash}) \end{aligned} \quad (3.32)$$

Weight of VFA leaving Rumen (g)

$$\text{VFAExRu} = \text{MwVFA} \times (U_{Va,VaEx} + U_{Va,VaAb}) \quad (3.32a)$$

Moles of VFA leaving Rumen (moles)

$$\text{VFAExM} = U_{Va,VaEx} + U_{Va,VaAb} \quad (3.32b)$$

$$\text{GasAmMi} = U_{Am,AmMi} + U_{Sc,AmMi} - P_{Mi,AmMi} - (\text{MwVFA} \times P_{Va,AmMi}) \quad (3.32c)$$

$$\text{GasPsMi} = U_{Ps,PsMi} + U_{Sc,PsMi} + U_{Am,PsMi} - P_{Mi,PsMi} - (\text{MwVFA} \times P_{Va,PsMi}) \quad (3.32d)$$

$$\text{GasScVa} = U_{Sc,ScVa} - (\text{MwVFA} \times P_{Va,ScVa}) \quad (3.32e)$$

$$\text{GasPsAm} = U_{Ps,PsAm} - (\text{MwVFA} \times P_{Va,PsAm}) - P_{Am,PsAm} \quad (3.32f)$$

$$\begin{aligned} RumenDM = {} & Q_{FuUaUe} + Q_{FuAvUe} + Q_{FuAvEs} + Q_{FdUaUe} + Q_{FdAvUe} + Q_{FdAvEs} + Q_{PuUaUe} \\ & + Q_{PuAvUe} + Q_{PuAvEs} + Q_{PdUaUe} + Q_{PdAvUe} + Q_{Pd,AvEs} \\ & + Q_{Fu} + Q_{Fd} + Q_{Si} + Q_{Sc} + Q_{Pu} + Q_{Pd} + Q_{Ps} + Q_{Ld} \\ & + Q_{Mi} + Q_{Ash} + (Q_{Va} \times \text{MwVFA}) \end{aligned} \quad (3.33)$$

Post ruminal zero pools

Absorption of amino acids, A_{Aa} (moles/day)

$$A_{Aa} = \left[U_{Pd,EaEx} + U_{Pd,PdEx} + U_{Pz,PzEx} + Frmic \times U_{Mi,MiEx}\right]/110 \quad (3.34)$$

Absorption of glucose, A_{Gl} (moles/day)

If $D_{Si} = 0$ Then

$$A_{Gl} = \left[U_{Sc,ScEx} + 0.202 \times U_{Mi,MiEx}\right]/181, \text{ Else} \quad (3.35a)$$

If $(U_{Si,SiSc}/D_{Si}) > 1$ then

$$A_{Gl} = \left[U_{Sc,ScEx} + 0.202 \times U_{Mi,MiEx} + 0.75 \times U_{Si,SiEx}\right]/181, \text{ Else} \quad (3.35b)$$

$$A_{Gl} = \left[U_{Sc,ScEx} + 0.202 \times U_{Mi,MiEx} + 0.75 \times (U_{Si,SiSc}/D_{Si}) \times U_{Si,SiEx}\right]/181 \quad (3.35c)$$

Absorption of long chain fatty acids, A_{Ld} (moles/day)

$$A_{Ld} = \left[0.9 \times (U_{Ld,LdEx} + 0.0805 \times U_{Mi,MiEx})\right]/630 \quad (3.36)$$

Absorption of volatile fatty acids, A_{Vf} (moles/day)

If $D_{Si} = 0$ Then

$$A_{Vf} = 0.01064 \times 0.11 \times U_{Fd,FdSc} + U_{Va,VaEx} \quad (3.37a)$$

If $(U_{Si,SiSc}/D_{Si}) > 1$ then

$$A_{Vf} = 0.01064 \times (0.25 \times U_{Si,SiEx} + 0.11 \times U_{Fd,FdSc}) + U_{Va,VaEx} \text{ Else} \quad (3.37b)$$

$$A_{Vf} = 0.25 \times (U_{Si,SiSc}/D_{Si}) \times U_{Si,SiEx} + 0.11 \times U_{Fd,FdSc} \quad (3.38c)$$

$$A_{Vf} = U_{Va,VaEx} + 0.01064 \times A_{Vf} \quad (3.38d)$$

$$A_{VFA} = U_{Va,VaAb} + A_{Vf} \quad (3.39)$$

APPENDIX 3.2

Values of model parameters

$cDMRu = 80.06$ g/L	$k_{FlEx} = 1.921$/day	$M_{Am,AmMi} = 26$ mg/L
$Abite = 0.0000041$	$R_{Sc,AmMi} = 5.28$ gSc/gAm	$J_{Ps,AmMi} = 230$ mg/L
$k_{Pu,UsAv} = 12$/day	$R_{Sc,PsMi} = 3.15$ gSc/gPs	$M_{Sc,AmMi} = 12.08$ g/L
$k_{Pd,UsAv} = 12$/day	$v^{(1)}_{ScVa} = 2.4$ g Sc/ g Mi/day	$M_{Ld,AmMi} = 1.0$ mg/L
$k_{Fd,UsAv} = 12$/day	$v^{(2)}_{ScVa} = 12$ g Sc/g Mi/day	$k_{AmAb} = 12$/day
$k_{Fu,UsAv} = 12$/day	$M_{Sc,ScVa} = 12.08$ g/L	$C_{Am} = 28$ mg/L
$k_{Fd,UsEs} = 6.0$/day	$J_{Ps,ScVa} = 0.230$ g/L	$C_{Ps} = 6.0$ mg/L
$k_{Pu,UsEs} = 6.0$/day	$Y_{Ps,PdPs} = 1.0$ g Ps/g Pd	$C_{Sc} = 600$ mg/L
$k_{Fd,UsEs} = 6.0$/day	$v_{PsMi} = 10.464$ g Ps/g Mi/day	$R_{Ld,AmMi} = 0.545$ g Ld/g Am
$k_{Fu,UsEs} = 6.0$/day	$M_{Ps,PsMi} = 230$ mg/L	$R_{Ld,PsMi} = 0.127$ g Ld/g Ps
frAc = 0.63	$M_{Sc,PsMi} = 12.08$ g/L	$Y_{Mi,AmMi} = 7.89$ g Mi/g Am
frPr = 0.23	$M_{Ld,PsMi} = 1.0$ mg/L	$Y_{Mi,PsMi} = 1.83$ g Mi/g Ps
frBu = 0.14	$v_{PsAm} = 2.28$ g Ps/g Mi/day	$Y_{Va,AmMi} = 6.75$ mmol Va/ g Sc
MwVFA = 67.2040 g	$M_{Ps,PsAm} = 445$ mg/L	$Y_{Va,PsMi} = 8.35$ mmol Va/ g Sc
$k_{SoEx} = 0.72$/day	$J_{Sc,PsAm} = 884$ mg/L	$Y_{Va,ScVa} = 10.64$ mmol Va/ g Sc
$C_{refMi} = 12.5$ g/L	$Y_{Am,UrAm} = 0.971$ g Am/g dietary N	$Y_{Va,PsAm} = 15.67$ mmol Va/ g Ps
$k_{ForEx} = 0.720$/day	$J_{Am,UrAm} = 75$ mg/L	$k_{VaAb} = 9.12$/day
$Y_{Sc,InLd} = 0.19$ kg Sc/kg Ld	$Y_{Am,PsAm} = 0.194$ g Am/g Ps	frMIC = 0.463
$Y_{Sc,FdSc} = 1.0$ kg Sc/kg Fd	$R_{Am,PsMi} = 38$ mg Am/g Ps	
$Y_{Sc,SiSc} = 1.0$ kg Sc/kg Si	$v_{AmMi} = 1.224$ g Am/g Mi/day	

Chapter 4

Competition of three aggregated microbial species for four substrates in the rumen

4.1 The rumen microbial ecosystem

The digestive system of ruminants (for example, cattle and sheep) contains one of the most diverse and abundant natural ecosystems of microorganisms. Over 200 species of bacteria and 20 species of protozoa have been discovered (Hungate, 1966). The consortium of microorganisms play a vital role in the digestion and conversion of food entering the rumen to available energy, in the form of short-chain fatty acids (volatile fatty acids), and nitrogen, in the form of microbial protein, for the host. Microbial numbers and composition are affected by a number of factors of which diet and feeding regime are probably the most important (Dijkstra and France, 1996). The interactions between rumen microorganisms are complex and not well understood (Wolin and Miller, 1988), and are important in the manipulation of rumen fermentation: optimizing fermentation processes to maximize the supply of available nutrients. However, even if all conceivable microbial interrelations have been discovered, the challenge will still be to organize them into a coherent whole. Therefore, it may be more profitable to approach the problem from a different direction and seek general interactions between groups of microorganisms and their competition for the consumption of various substrates (Czerkawski, 1986).

A compartmental model of rumen microorganism interaction was incorporated into a whole-rumen model by Dijkstra *et al.* (1992). Improvements to a previously aggregated microbial pool model (France *et al.*, 1982) included microbial substrate preference, differential outflow of substrate and the recycling of microbial matter. Neal *et al.* (1992) evaluated the whole-rumen model and found that the model did not adequately predict the molar proportions of VFA and suggested that the representation of protozoan activities require further attention. Dijkstra and France (1996) reviewed models of rumen function and outlined a number of challenges in addition to the need to adequately predict VFA molar proportions, these included: simulation of discontinuous feeding regimes and outflow of material from the rumen, the microbial distribution within the rumen, the interactions between microorganisms and the amino acid composition of undegraded feed and microbial matter.

The objective of this chapter is to develop a chemostat-type model of three aggregated functional classes of microbial species in the rumen based on a representation and certain parameter values of a whole rumen model developed by Dijkstra (1994). The aim is to understand the general qualitative behaviour of the model: the effects of changing substrate composition and quantity, the effect of nitrogen (ammonia) supplementation, and the effect of a periodic input of substrate on the abundance of microbial populations in the rumen.

Table 4.1: List of symbols used in the model

Parameters	Definition	Unit
X_i	abundance of microbial population in the rumen	g
S_j	concentration of substrate in the rumen	$\frac{g}{L}$
Flex	rate of fluid flow out of the rumen	$\frac{L}{day}$
$\mu_{i,max}$	maximum growth of X_i	$\frac{1}{day}$
k_{S_j,X_i}	affinity of X_i for S_j	$\frac{g}{L}$
K_{pi}	affinity of X_i due to predation by protozoa	$\frac{g}{L}$
Y_{pi}	yield of protozoa by consumption of X_i	$\frac{g\,X_2}{g\,X_i}$
Y_{s_j,X_j}	yield of X_i by consuming S_j	$\frac{g\,X_i}{g\,S_j}$
S_{jin}	inflow rate of S_j	$\frac{mole}{day}$
C_{S_j}, C_{X_i}	concentration of S_j, concentration of X_i	$\frac{g}{L}$
μ_i	growth rate of X_i	$\frac{1}{day}$
μ_{2p}	rate of consumption of amylolytic microbes by predation by protozoa	$\frac{g\,X_2}{(g\ ).day}$
μ_{3p}	rate of consumption of cellulolytic microbes by predation by protozoa	$\frac{g\,X_2}{(g\ ).day}$

4.2 Model development

A chemostat-type model of three aggregated functional classes of species in the rumen: amylolytic microbes (X_1), protozoa (X_2) and cellulolytic microbes (X_3) competing for four growth-limiting nutrients: ammonia (S_1), soluble protein (S_2), α-hexose (S_3) and β-hexose (S_4) was developed. The microbial species were distinguished according to microbial substrate preference: amylolytic microbes utilize hexose derived from non-structural carbohydrates (soluble sugars, starch and pectin), cellulolytic (fibrolytic) microbes utilize hexose derived from structural carbohydrate (cellulose and hemicellulose), and protozoa, which feeds by predation on rumen bacteria and on soluble starch. The microbial abundance is measured in grams (per rumen) and the substrate concentration in grams per litre. The general model equations are given below and the parameters and variables explained below.

$$\frac{dX_1}{dt} = \underbrace{\mu_1 \times X_1}_{\text{growth of amylolytic microbes}} - \underbrace{Flex \times C_{X_1}}_{\text{washout}} - \underbrace{\mu_{p1} \times X_2}_{\text{predation}} \quad (4.1)$$

$$\frac{dX_2}{dt} = \underbrace{\mu_2 \times X_2}_{\text{growth of protozoa}} - \underbrace{0.45 \times 0.3704 \times Flex \times C_{X_2}}_{\text{washout}} \quad (4.2)$$

$$\frac{dX_3}{dt} = \underbrace{\mu_3 \times X_3}_{\text{growth of cellulolytic microbes}} - \underbrace{0.3704 \times Flex \times C_{X_3}}_{\text{washout}} - \underbrace{\mu_{p2} \times X_3}_{\text{predation}} \quad (4.3)$$

$$\frac{dS_1}{dt} = \underbrace{S_{1in}}_{\text{inflow}} - \underbrace{\frac{\mu_1 \times X_1}{Y_{S_1,X_1}}}_{\text{consumption by } X_1} - \underbrace{\frac{\mu_3 \times X_3}{Y_{S_1,X_3}}}_{\text{consumption by } X_3} - \underbrace{\text{Flex} \times C_{S_1}}_{\text{outflow}} \quad (4.4)$$

$$\frac{dS_2}{dt} = \underbrace{S_{2in}}_{\text{inflow}} - \underbrace{\frac{\mu_1 \times X_1}{Y_{S_2,X_1}}}_{\text{consumption by } X_1} - \underbrace{\frac{\mu_2 \times X_2}{Y_{S_2,X_2}}}_{\text{consumption by } X_2} - \underbrace{\frac{\mu_3 \times X_3}{Y_{S_2,X_3}}}_{\text{consumption by } X_3} - \underbrace{\text{Flex} \times C_{S_2}}_{\text{outflow}} \quad (4.5)$$

$$\frac{dS_3}{dt} = \underbrace{S_{3in}}_{\text{inflow}} - \underbrace{\frac{\mu_1 \times X_1}{Y_{S_3,X_1}}}_{\text{consumption by } X_1} - \underbrace{\frac{\mu_2 \times X_2}{Y_{S_3,X_2}}}_{\text{consumption by } X_2} - \underbrace{\text{Flex} \times C_{S_3}}_{\text{outflow}} \quad (4.6)$$

$$\frac{dS_4}{dt} = \underbrace{S_{4in}}_{\text{inflow}} - \underbrace{\frac{\mu_1 \times X_2}{Y_{S_4,X_2}}}_{\text{consumption by } X_2} - \underbrace{\frac{\mu_3 \times X_3}{Y_{S_4,X_3}}}_{\text{consumption by } X_3} - \underbrace{0.37037 \times \text{Flex} \times C_{S_4}}_{\text{outflow}} \quad (4.7)$$

$$\mu_1 = \frac{\mu_{1,max} C_{S_1} C_{S_2} C_{S_3}}{(K_{S_1,X_1} + C_{S_1})(K_{S_2,X_1} + C_{S_2})(K_{S_3,X_1} + C_{S_3})} \quad (4.8)$$

$$\mu_2 = \frac{\mu_{2,max} C_{S_2} C_{S_3} C_{S_4} C_{X_1} C_{X_2}}{(K_{S_3,X_2} + C_{S_3})(K_{S_2,X_2} + C_{S_2})(K_{S_4,X_2} + C_{S_4})(K_{P_1} + C_{X_1})(K_{P_2} + C_{X_1})} \quad (4.9)$$

$$\mu_3 = \frac{\mu_{3,max} C_{S_1} C_{S_2} C_{S_4}}{(K_{S_1,X_3} + C_{S_1})(K_{S_2,X_3} + C_{S_2})(K_{S_4,X_3} + C_{S_4})} \quad (4.10)$$

$$\mu_{p1} = \frac{\mu_{p1max} \times C_{X_1}}{K_{P1} + C_{X_1}} \qquad \mu_{p1} = \frac{\mu_{p2max} \times C_{X_3}}{K_{P2} + C_{X_3}} \quad (4.11; 4.12)$$

Some general assumptions of the model are as follows:

(i) Since the entrance of the rumen is relatively close to the exit, the system can not function unless the contents are well-stirred (i.e., no spatial variations in concentration of microbes and nutrients).

(ii) The volume of the contents and density throughout the rumen are assumed to be constant.

(iii) Rumen metabolism depends only on the carbohydrate (hexoses) and nitrogen-containing (ammonia or soluble protein) substrates, with other nutrients (such as minerals and vitamins) assumed to be present in non -limiting quantities.

(iv) We assume that the only nitrogen supply is via the food and have not accounted for the nitrogen transferred across the rumen wall or produced by bacteria and protozoa (Firkins, 1996). This assumption is applied in this chapter only and nitrogen across the rumen wall and via the saliva will be accounted for in chapter 7.

We consider a constant and homogeneous environment, and derive an explanation for microbial dynamics based on the dynamics of microbial interactions. Amylolytic (X_1) microbes utilize hexose derived from non-structural carbohydrates (for example, soluble sugars, starch and pectin). We assume that the growth rate of X_1 depends on the concentration of α-hexose (S_3) and nitrogen (ammonia, S_1, and soluble protein, S_2). We will assume that the amylolytic microbes have the fastest growth rate (Hobson, 1988) and estimate its maximum growth rate ($\mu_{1,max}$) from experimental values by van Gylwyk *et al.* (1992) to be 8.61/day. The affinity constant of amylolytic microbes for ammonia ($K_{S_1,X_1} = 0.00135$ g/L), the affinity constant of amylolytic microbes for soluble protein ($K_{S_2,X_1} = 0.0224$ g/L) and the affinity of amylolytic microbes for α-hexose ($K_{S_3,X_1} = 0.0159$ g/L) was estimated by Dijkstra (1993). Since the amylolytic microbes are assumed to reside in the rumen fluid, their washout rate will be the washout rate of the rumen fluid (*Flex*, L/day), which is assumed to be an average of 150 L/day, multiplied by the concentration of amylolytic microbes (C_{X_1}, g/L). The maximum rate of engulfment of amylolytic microbes by protozoa ($\mu_{pl max}$) and the affinity of protozoa for bacteria as a nitrogen source (K_{pl}) was estimated by Dijkstra *et al.* (1992) based on engulfment rate data by Coleman and Sandford (1979), therefore, $\mu_{pl max} = 15.439$ gX_1/(g X_2.day) and $K_{pl} = 34.694$ g/L. It is assumed that the amylolytic bacteria are engulfed in the proportion in which they are present (Dijkstra, 1994), even though experimental evidence (Coleman, 1989) suggests that there are no consistent patterns of engulfment. The yield of protozoa biomass (Y_{pl})

from the predation on amylolytic microbes is assumed to be 149.25 $\frac{g X_2}{g X_1}$. (Dijkstra, 1993).

Carbohydrate and nitrogen sources for protozoal growth are assumed to be complementary following Dijkstra (1993), that is, carbohydrate and nitrogen sources must be taken together by the consumer because each resource fulfils one of the protozoan populations' essential needs (Leon and Thumpson, 1975). The maximum rate of growth of protozoa ($\mu_{2,max}$) is assumed to be less than the maximum rate of growth of amylolytic microbes and assumed equal to the average rate of incorporation of α-hexose and β-hexose, therefore, $\mu_{2,max} = 5.51$/day. It is assumed (Coleman, 1986; Jouany *et al.*, 1988) that most protozoa cannot use urea or ammonia to synthesize amino acids de novo. In the absence of available data, we assume that the affinity for α-hexose by protozoa is much higher than for β-hexose by protozoa and therefore $K_{S_3,X_2} = 0.037$ g/L and $K_{S_4,X_2} = 0.0083$ g/L. Also, we assume that the chance that a protozoan will encounter an amylolytic microbe is approximately equal to the probability of an encounter with a cellulolytic microbe and therefore the affinity constant for cellulolytic microbes by protozoa is $K_{p3} = 34.694$ g/L. The affinity of protozoa for soluble protein (K_{S_2,X_2}) is assumed to be 0.04 g/L (Dijkstra, 1994). Protozoa are selectively retained within the rumen (see review of Jouany *et al.*, 1988) and therefore the outflow rate of protozoa is smaller and is set at 45% of the outflow rate of solid digesta (Faichney, 1989), that is, $0.45 \times 0.37 \times Flex \times C_{X_2}$ where 0.37 is the proportion of solid (1.0/day) and liquid (2.7/day) outflow assumed by Dijkstra (1993).

There is increasing evidence that the microbial degradation of complex polysaccharides in the rumen is accomplished by the cooperative effort of a range of cellulolytic and non-cellulolytic microorganisms (Cheng *et al.*, 1991). We have assumed here that the degradation of complex polysaccharides are accomplished by an aggregated microbe population consisting of cellulolytic fungi and bacteria (Dehority, 1991). The maximum rate of growth ($\mu_{3,max}$) is estimated as the average of the incorporation rate of ammonia and soluble protein, therefore, $\mu_{3,max} = 3.50$/day. The affinity of the cellulolytic microbes for ammonia (K_{S_1,X_3}) is 0.00135 g/L and the affinity of the cellulolytic microbes for soluble protein (K_{S_2,X_3}) is 0.0224 g/L (Dijkstra, 1993). Also, the affinity of the cellulolytic microbes for a carbohydrate source (K_{S_4,X_3}) is assumed to be 0.0159 g/L. The washout rate is assumed to be equal to the outflow rate of the solid digesta, $0.37 \times Flex \times C_{X_3}$. Dijkstra *et al.* (1992)

represented the predation of cellulolytic microbes by protozoa and estimated the maximum engulfment rate (μ_{3pmax}) and affinity constant (K_{p3}) to be the same as the constants for predation of protozoa on amylolytic microbes. We therefore assume an engulfment rate of $\mu_{p3} = 15.439$ $gX_3/(gX_2.day)$ and an affinity constant of $K_{p2} = 34.694$g/L. The yield of protozoa biomass (Y_{p3}) from the predation on cellulolytic microbes was estimated to be 149.25 $\frac{g\,X_2}{g\,X_3}$ (Dijkstra, 1993).

The rate of change of liquid volume in the rumen, $\frac{dRvol}{dt}$, is a function of the average volume of fluid entering the rumen (Drink, L/day) and the average volume of saliva secretion (Saliva, L/day) less the fluid leaving the rumen (Flex, L/day):

$$\frac{dRvol}{dt} = Drink + Saliva - Flex \tag{4.13}$$

The yield of amylolytic biomass from the consumption of ammonia (Y_{S_1,X_1}), soluble protein (Y_{S_2,X_1}) and α-hexose (Y_{S_3,X_1}) is assumed to be 118.91 g/L, 149.48 g/L and 100.00 g/L, respectively. The yield of protozoa biomass from the consumption of α-hexose (Y_{S_3,X_2}) and β-hexose (Y_{S_4,X_2}) is both assumed to be 142.05 g/L. The yield of protozoa biomass from the consumption of soluble protein (Y_{S_2,X_2}) is assumed to be 149.25 g/L. The yield of cellulolytic biomass from the consumption of ammonia (Y_{S_1,X_3}), soluble protein (Y_{S_2,X_3}) and β-hexose (Y_{S_4,X_3}) is assumed to be 118.91 g/L, 149.48 g/L and 100.00 g/L, respectively.

4.3 Model application

The model was run using DRIVER (Hahn & Furniss, 1988) to examine the general qualitative behaviour (medium and long-term), the effect of varying the maximum growth rates, the effects of varying the composition and quantity of the substrate to the rumen, the effect of supplementing with ammonia and the effect of periodic substrate input on microbe population abundance, coexistence and dynamics. DRIVER is an interactive modelling tool coded in Turbo Pascal. The parameter values and initial microbe numbers are given in Appendix 4.1.

4.4 Results and Discussion

4.4.1 General model behaviour and the effect of changing growth rates

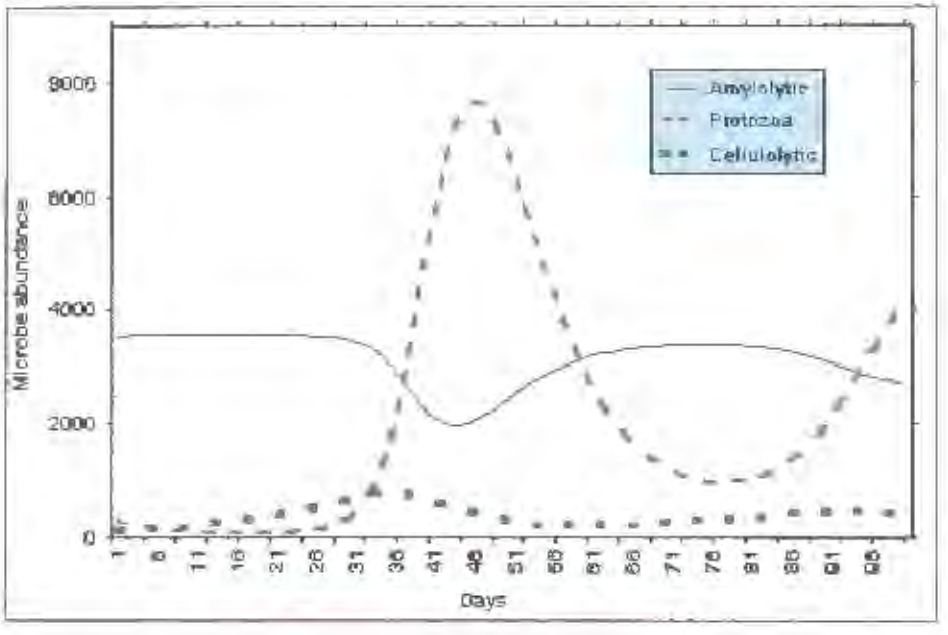
The substrate inputs were estimated based on cattle eating 7 kg dry matter per day of chopped Italian Ryegrass (France *et al.*, 1982) and is given in Table 4.2. The general medium-term (100 days) and long-term (500 days) behaviour of the model was

examined (Figure 4.1). The behaviour observed was expected for a continuous input of substrate: the microbial populations exhibited oscillations initially but the amplitude of the oscillations decreased as the populations tended towards their equilibrium values, with all populations coexisting. The protozoa population was dominant till about 176 days when the amylolytic bacteria became the dominant population. It has been shown experimentally (Hungate, 1966) that the cellulolytic bacteria is the most abundant population in the rumen, in contrast to model results. However, most cellulolytic bacteria are attached to the substrate (Annison and Bryden, 1998) and the model only accounts for the bacteria not attached to substrate. It has been observed that approximately three-quarters of the bacteria are tightly attached to feed particles or are found in biofilms (Hungate 1966). Developing a mathematical model to account for the attached and unattached microbes in the rumen remains a challenge.

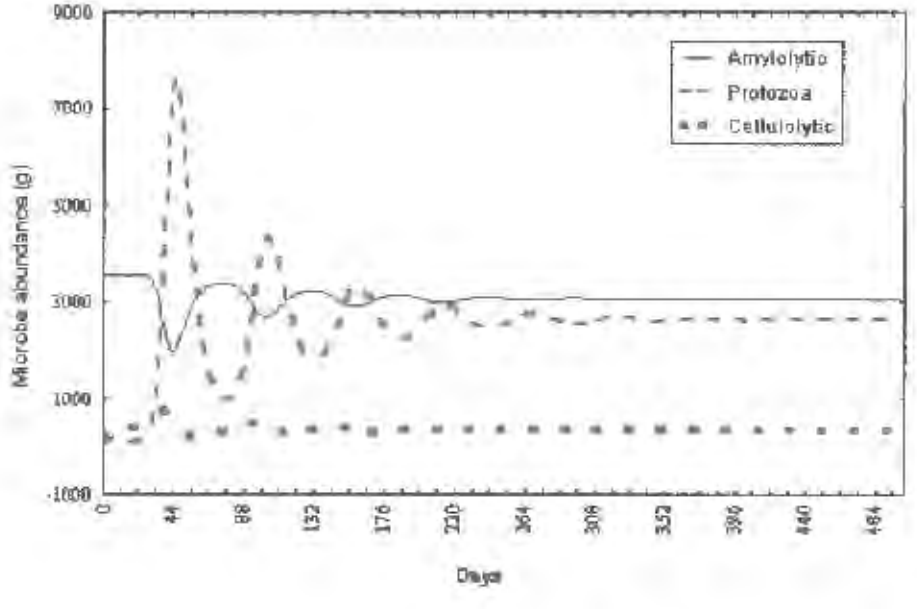
Figure 4.1c shows the oscillations of the three microbial populations more clearly ending in a limit cycle. Significant numbers of ruminal bacteria can be consumed by protozoa, resulting in an inverse relationship between protozoal and bacterial densities (Williams and Coleman, 1992). Ruminal protozoa have been shown to account for half of the microbial mass in the rumen, but defaunation studies indicate that the ruminal protozoa are not essential to their host's nutritional status. Nevertheless, many protozoa take up and store small starch granules, thereby modulating the fermentation rate and protecting the animal from acidosis (Russel and Rychlik, 2001).

There are a number of factors (for example, pH) that affect the maximum growth rates of each microbial population, thereby affecting the outcome of microbial competition (Reichl and Baldwin, 1976). The effect of changing the maximum growth rates on the abundance of each population after 100 days was examined in Figure 4.2. Interestingly, increasing $\mu_{3,max}$ resulted in a large increase in protozoa abundance probably due to the predation on cellulolytic bacteria by protozoa. However, bacterial predation (and protozoal lysis) can deprive the animal of microbial protein and increase excess ruminal ammonia (Bird and Leng, 1978). The model showed an increase from 0.029 to 0.04 g of ammonia in the rumen when cellulolytic growth rate was increased from 3 - 4/day.

(a)


Amylolytic
Protozoa
Cellulolytic
Microbe abundance
Days

(b)


Amylolytic
Protozoa
Cellulolytic
Microbe abundance (g)
Days

(c)

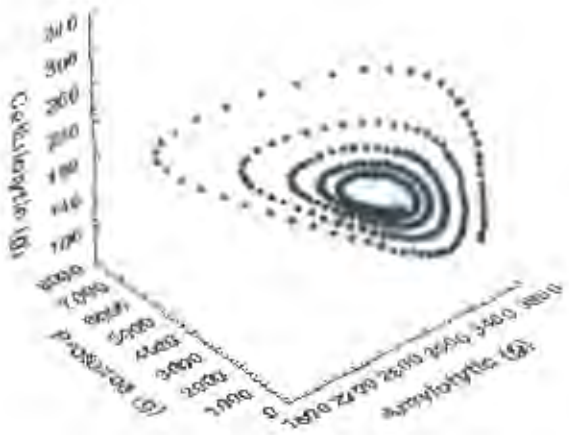
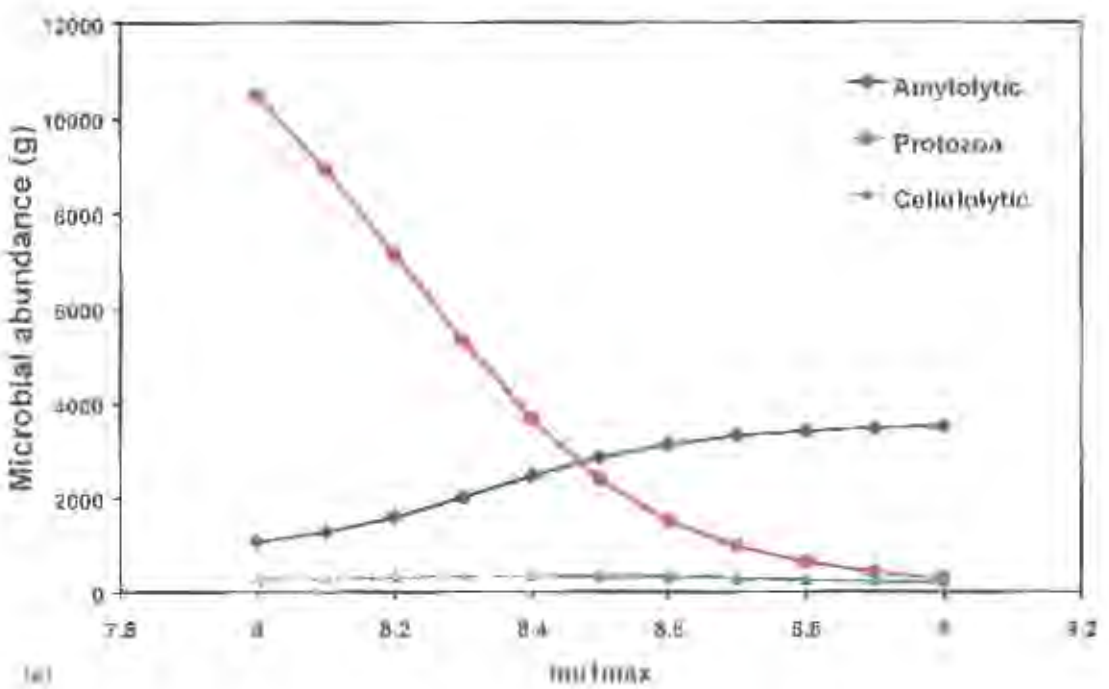
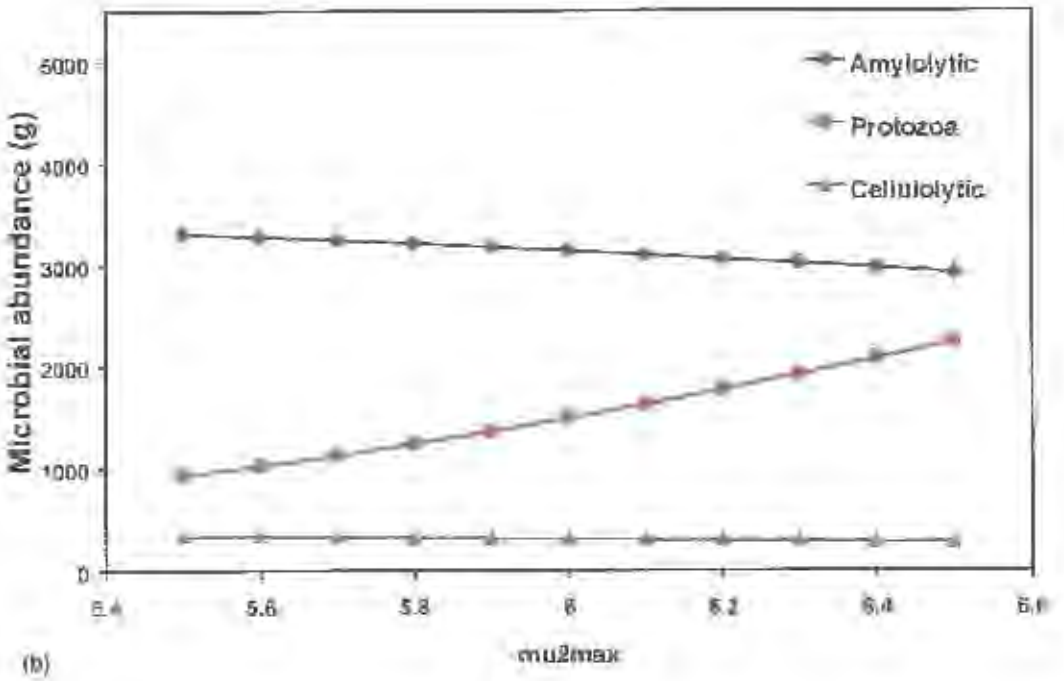


Figure 4.1: (a) Medium-term (100 days) behaviour of three microbial species. (b) Long-term (100 days) behaviour of three microbial species (c) Phase space of the three microbial species.



(a)



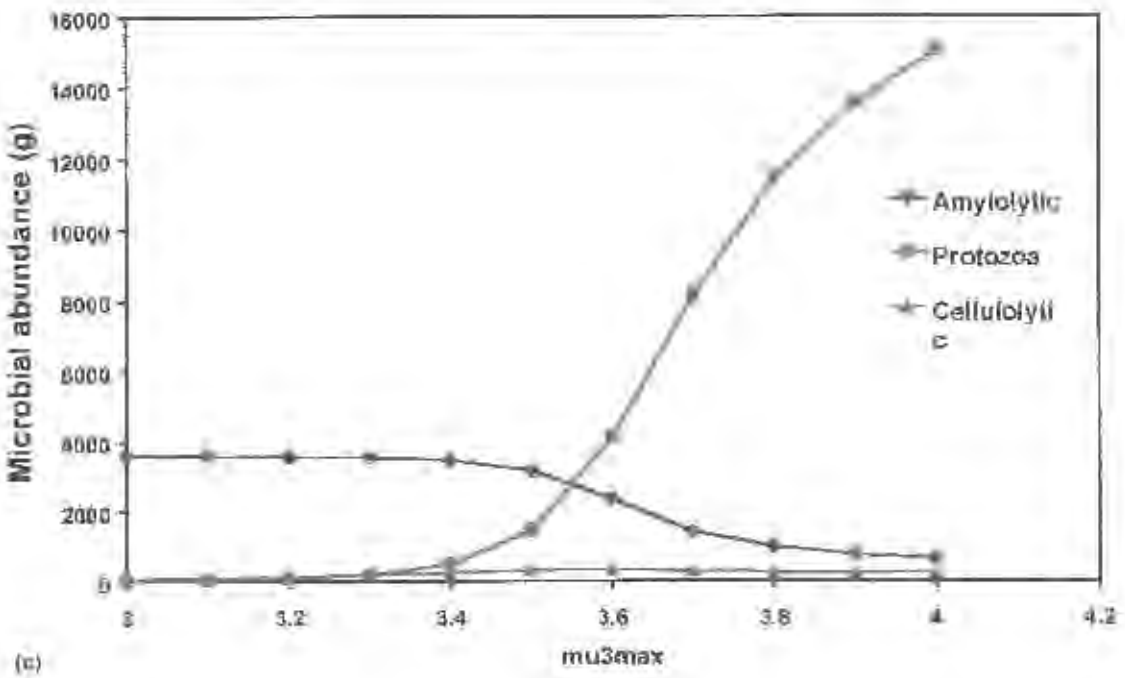


Figure 4.2: (a) Effect of varying μ_{1max} (8.0/day-9.0/day) on microbial abundance. (b) Effect of varying μ_{2max} (5.5/day-6.5/day) on microbial abundance. (c) Effect of varying μ_{3max} (3.0/day-4.0/day) on microbial abundance.

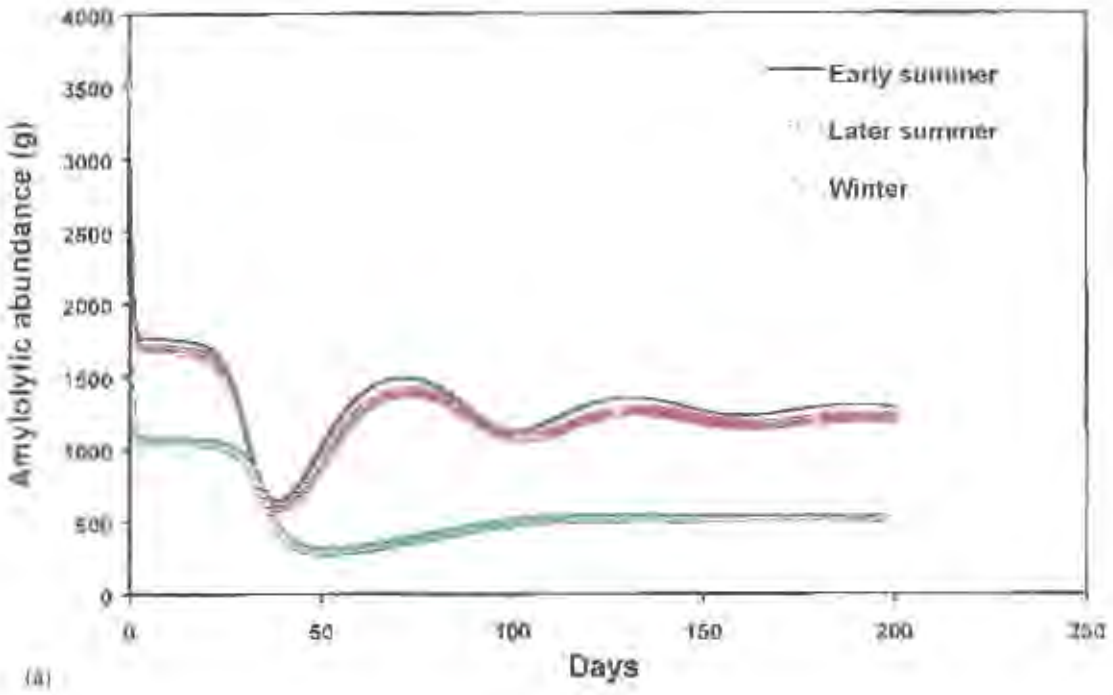
4.4.2 Effect of varying substrate composition and quantity

The input and the composition of substrates entering the rumen vary with the availability of herbage, which varies between seasons for animals on arid and semi-arid rangeland. For example, the natural subtropical herbage of central Africa grows and matures extremely rapidly and has a very low nutritive value for most the year (Elliott *et al.*, 1965). Growth of herbage begins with the onset of rain in October/November and after approximately two months the majority of plant species have flowered and seeded and by March/April, when the rains finish, the herbage is mature. Associated with this rapid growth and early maturity is a sharp seasonal decline in crude protein and a pronounced increase in crude fibre content. During the dry winter season the crude protein content of the dry herbage is 10-30 g/kg while the percentage of crude fibre present ranges from 400-450 g/kg (Elliott *et al.*, 1965). The composition of substrate (of a typical diet) entering the rumen during three periods of a year (early summer, later summer and winter) was estimated and is given in Table 4.2.

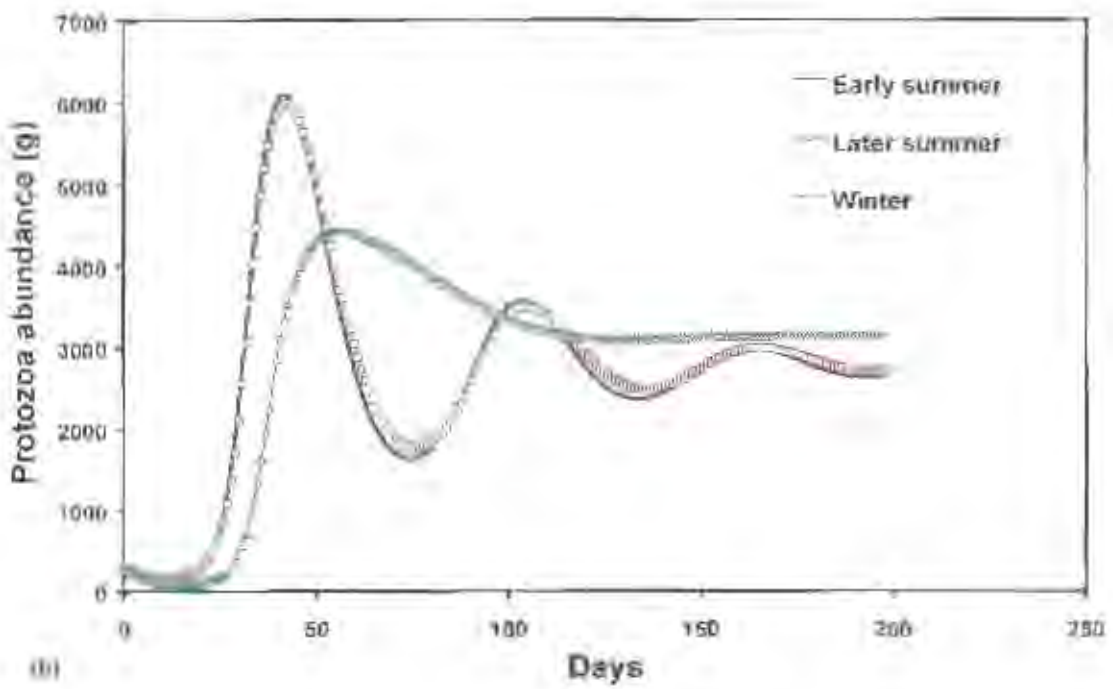
Table 4.2: Substrate input (g/L) for animals eating 7kg of chopped Italian Ryegrass (column 2) and substrate input (g/L) used for three periods (Early summer, Late summer, Winter) of a year.

Input	Italian Ryegrass	Early summer	Late summer	Winter
S_1	50.2	25	24	20
S_2	541.7	271	250	120
S_3	1795.4	898	650	450
S_4	3668.7	1834	1932	1950

The results of the change in substrate composition on microbial abundance is given in Figure 4.3 and the effect of three different quantities of substrate (of diets of 2kg, 4kg and 7kg) on microbial abundance is given in Figure 4.4. All three populations coexisted in each "season", however, microbe populations exhibited less oscillations in winter, this could be because of lower quantities of substrate (Figure 4.4). The early summer and later summer periods exhibited similar trends in microbial abundance, whereas the model predicted a much higher cellulolytic abundance due to the increase in crude fibre content as shown by Elliott *et al.* (1965).


Amylolytic abundance (g)
4000
3500
3000
2500
2000
1500
1000
500
0
0
50
100
150
200
250
Days
Early summer
Later summer
Winter

(a)


Protozoa abundance (g)
7000
6000
5000
4000
3000
2000
1000
0
0
50
100
150
200
250
Days
Early summer
Later summer
Winter

(b)

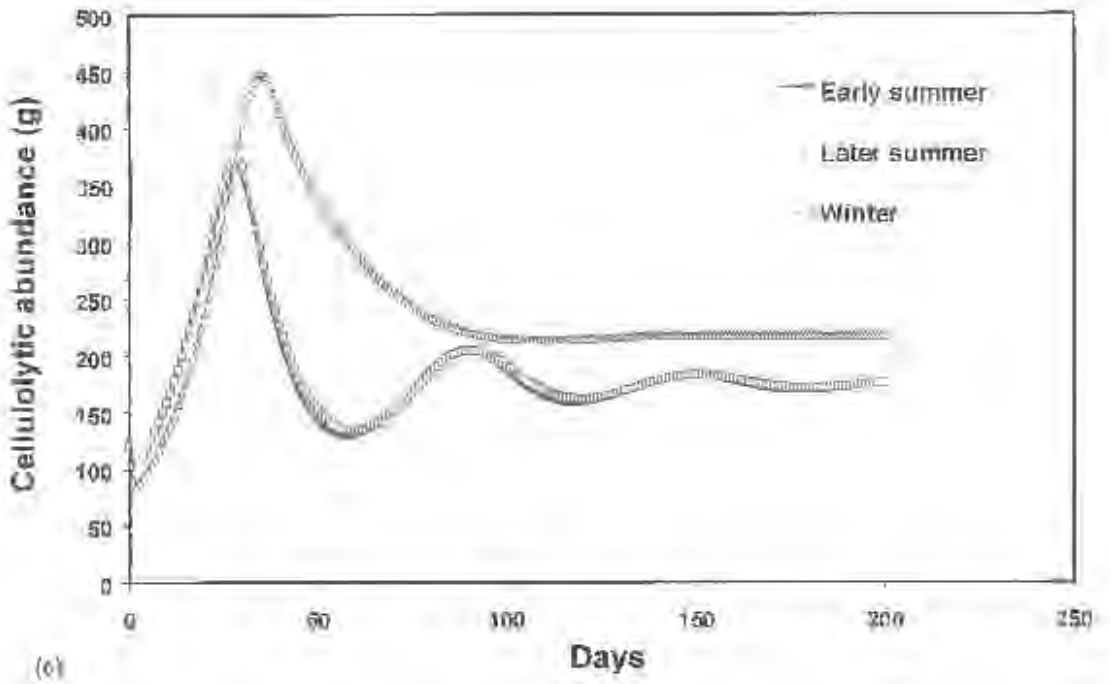
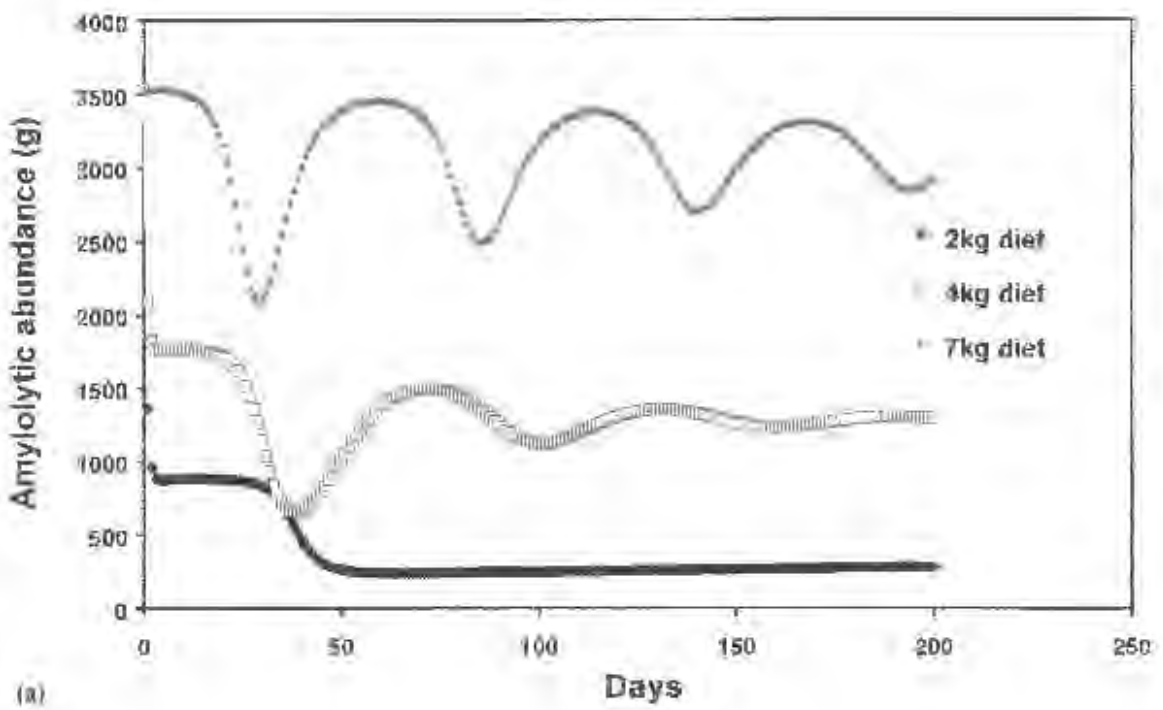
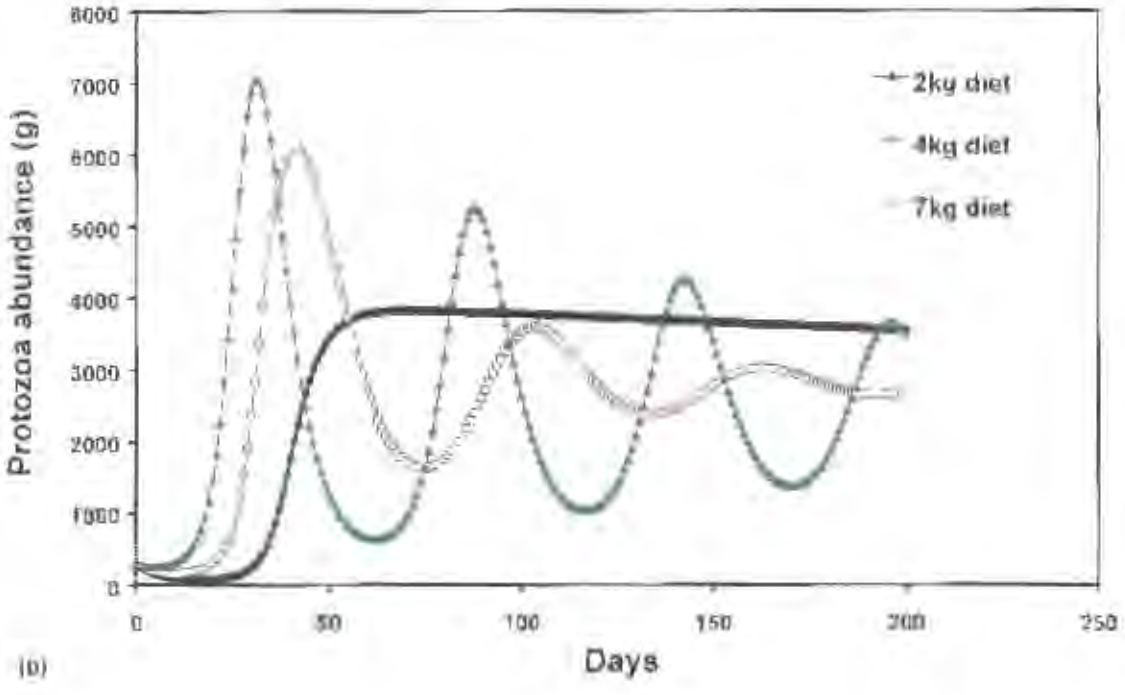


Figure 4.3: (a) The effect of three periods of a year (early summer, later summer, winter) on the abundance of amylolytic bacteria (g), (b) protozoa (g), and (c) cellulolytic bacteria (g).





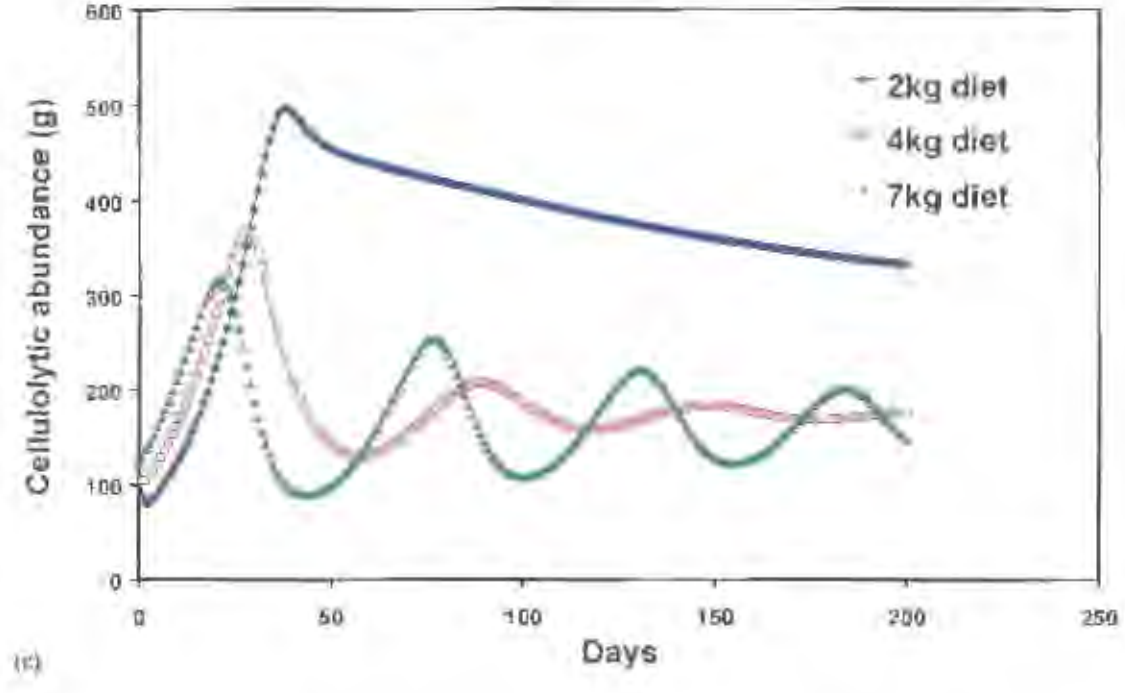
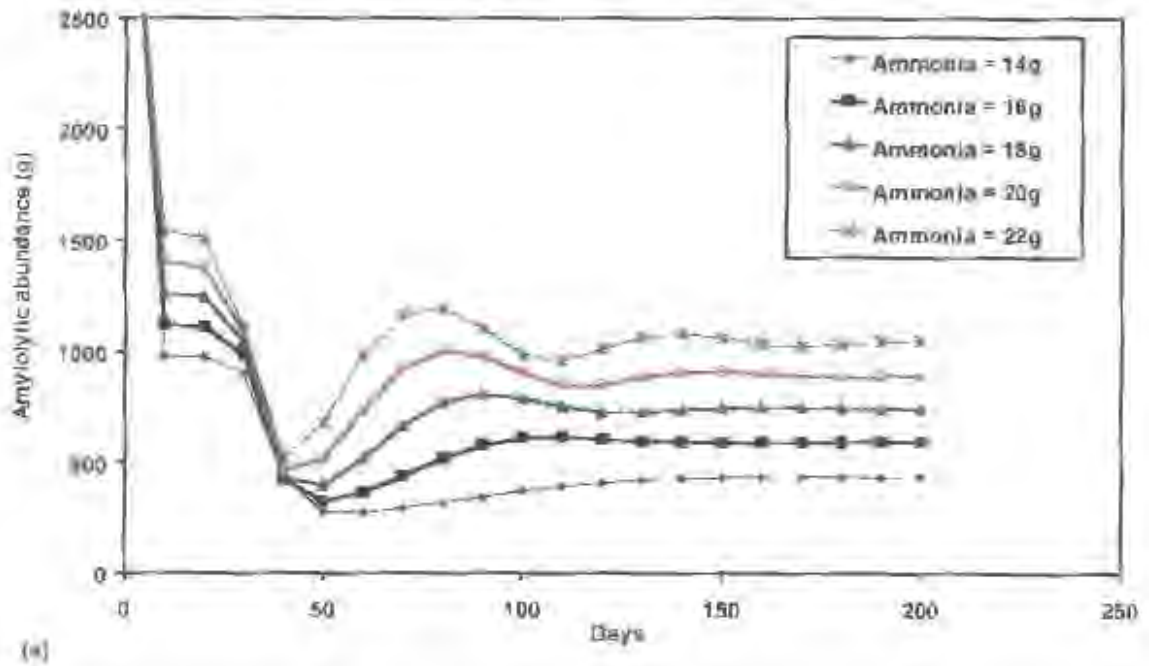


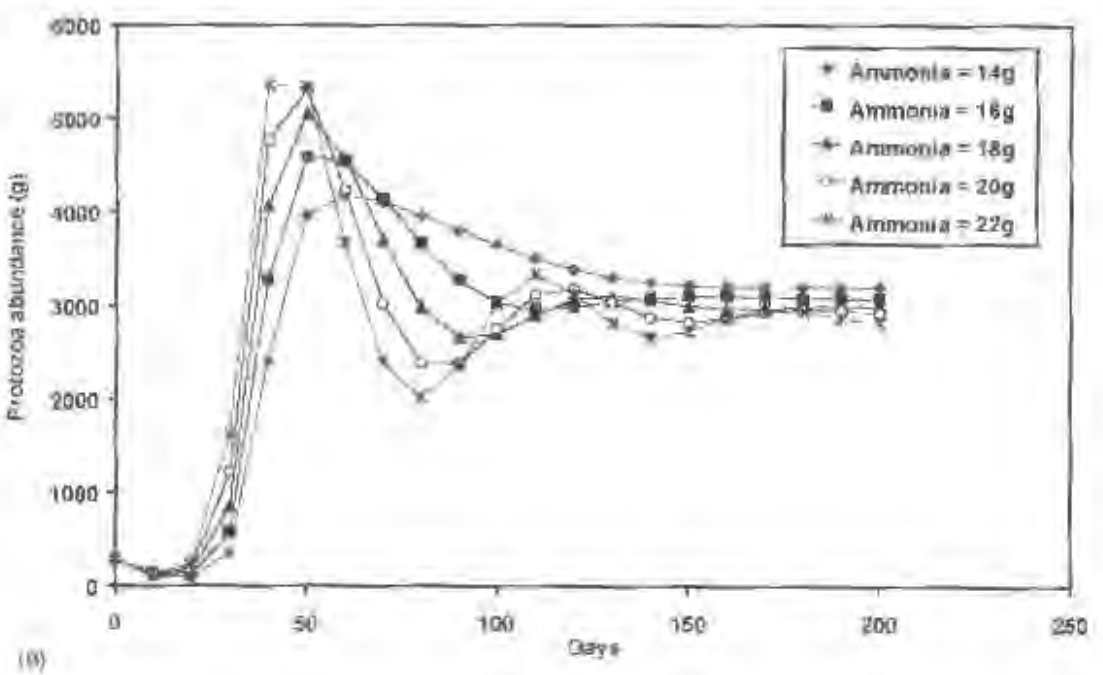
Figure 4.4: The effect of substrate quantity (from a diet of 2kg, 4kg, 7kg) on the abundance of (a) amylolytic bacteria , (b) protozoa, and (c) cellulolytic bacteria.

4.4.3 Effect of nitrogen (ammonia) supplementation and periodic liquid dilution rate

During winter months it is common practice to feed range animals protein-rich concentrates to minimize animal body weight loss. For this reason it will be important to examine the effects of increasing ammonia on the abundance of each microbial species. Figure 4.5 shows the effect of increased ammonia (S_i) on the abundance of each microbial species. As the ammonia entering the rumen was increased (from 14g/L to 22g/L), the oscillations of each population was shown to increase.



(a)



(b)

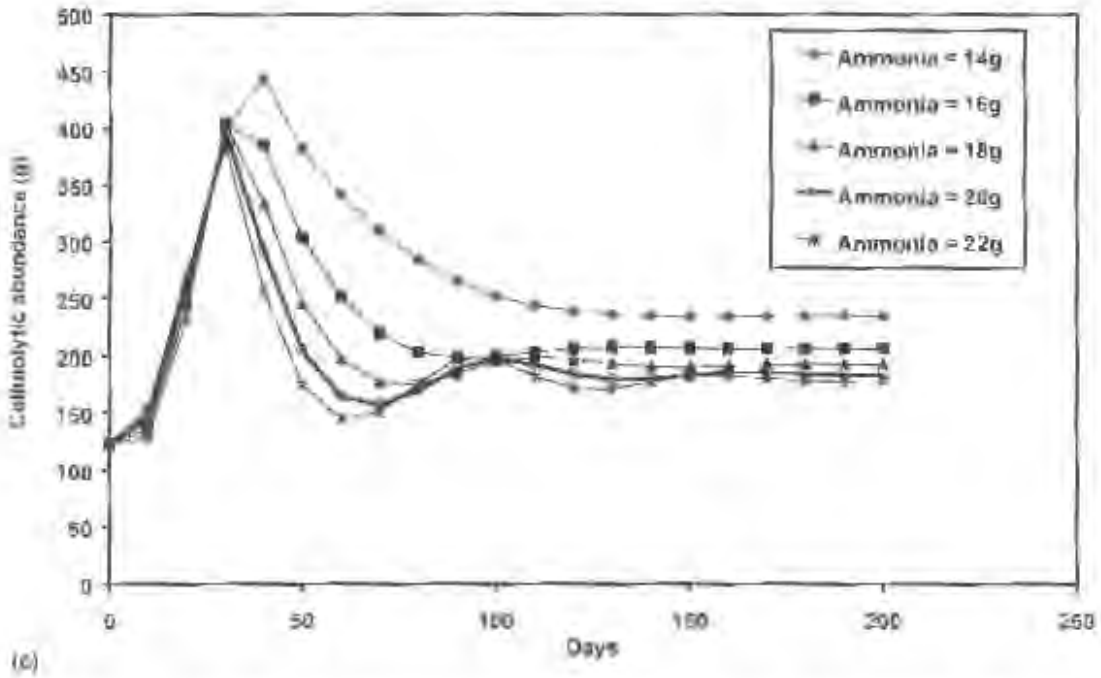
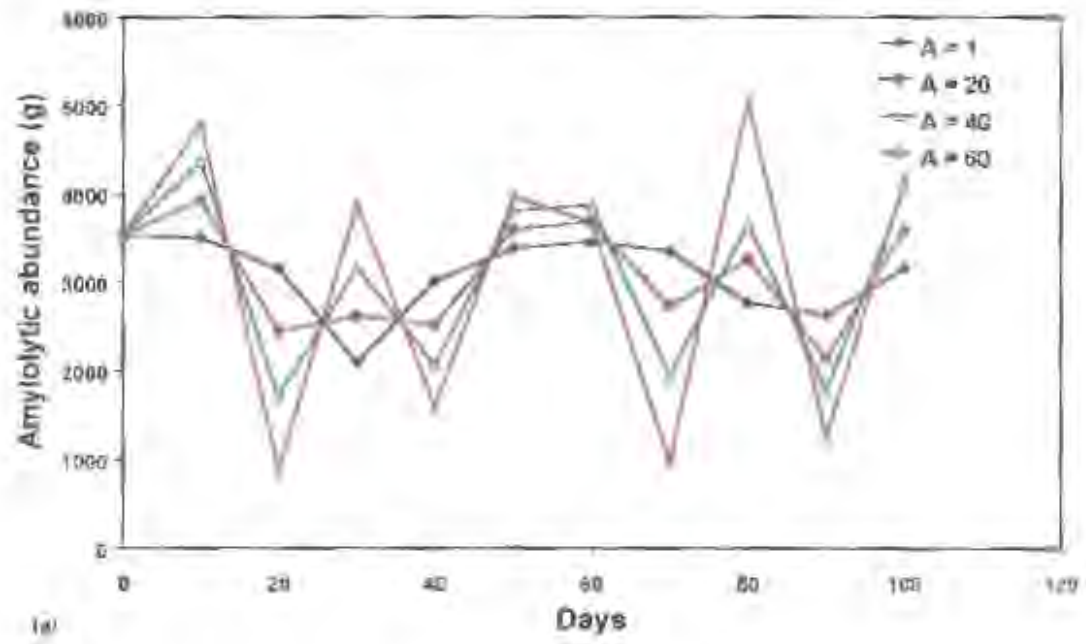
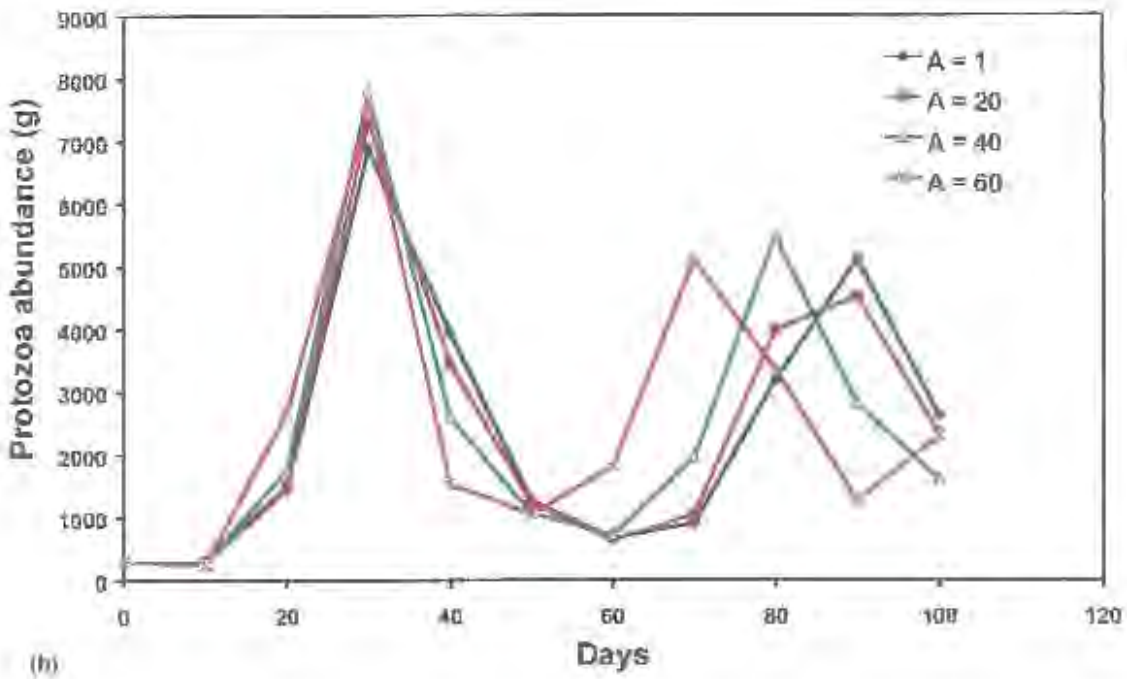


Figure 4.5: The effect of increasing ammonia from 14-22g/l on (a) amylolytic, (b) protozoa, and (c) cellulolytic abundance.

Flex was allowed to vary periodically in order to simulate perturbations within the rumen. $Flex = FLOS + A * cos(days)$ for $A = 0, 20, 40, 60$ and $FLOS = 150$. The results are given in Figure 4.6. As A was increased the microbial populations oscillated with greater amplitude, except for the protozoa numbers which exhibited a phase shift to the left of about 50 days after about 60 days.





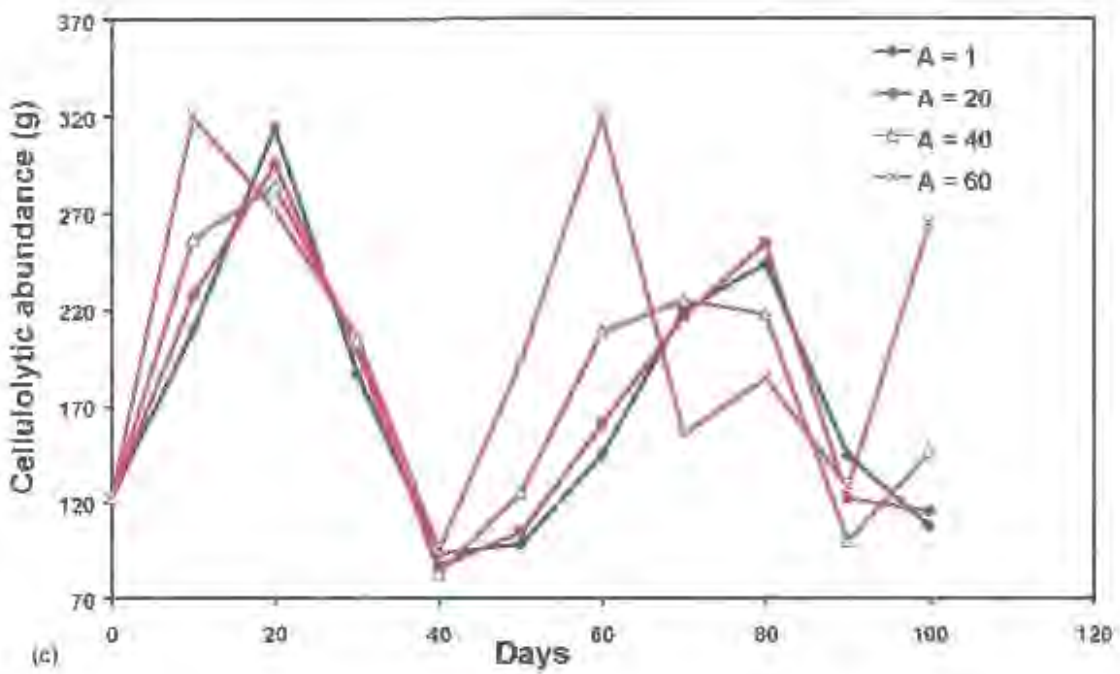


Figure 4.6: Increasing the liquid dilution rate ($Flex$) on the abundance of (a) amylolytic, (b) protozoa, and (c) cellulolytic microbes in the rumen. $Flex$ was allowed to vary periodically in order to simulate perturbations within the rumen: $Flex = FLOS + A \times cos(days)$ for $A = 1, 20, 40, 60$ and $FLOS = 150$.

4.5 Conclusions

An initial analysis of the dynamics of a model of a chemostat with a rumen microbial system consisting of three populations competing for substrate, where one population (the protozoa) also feeds on the other two bacteria populations, was presented. Of particular interest is the dependence of the observed dynamic behaviour on the conditions of the system, that is, on the quantity and composition of substrate and change in chemostat dilution rate (*Flex*). The rumen is inhabited by diverse and interdependent populations of bacteria, protozoa and fungi and a variety of models of rumen function have attempted to include the interactions between rumen microorganisms. However, many questions still remain to be addressed and this paper serves as an initial study as part of a longer term project to understand the dynamics of rumen microorganisms.

The analysis shows that there exists a wide range of conditions for which all three populations coexist. This result is in accordance with earlier observations that the presence of a population preying on two populations competing for a single rate-limiting nutrient stabilizes their coexistence (Jost *et al.*, 1973). Increasing the ammonia supplementation and fluid dilution rate promotes the sustained oscillations of populations. In particular, when increasing the fluid dilution rate, the bacteria populations exhibited chaotic behaviour. This observation is important from an ecological point of view because it is known that a simple food chain with one predator and one prey population exhibits at most periodic behaviour, whereas a three-species food chain can exhibit chaotic behaviour. The bifurcation analysis of Vayenas and Pavlou (1999) showed that an extra trophic level can lead to chaotic behaviour whereas the present analysis shows that a system with an extra trophic level and where all three species compete for substrate can exhibit chaotic behaviour and also identifies the factor (fluid dilution rate) which can lead to the system exhibiting chaotic behaviour. Furthermore, Vayenas and Pavlou (1999) showed that for biologically derived parameters, coexistence is realized in either a steady state or periodic state but not in a chaotic state, however, this analysis shows that increasing the fluid dilution rate within a biologically acceptable range, rumen miccroorganisms can coexist and exhibit chaotic behaviour.

APPENDIX 4.1

Values of model parameters

$\mu^*_{1,max} = 8.61$/day	$K_{p2} = 34.694$g/L	$Y_{S_3,X_1} = 100$g/L
$K_{S_1,X_1} = 0.00135$g/L	$K_{S_2,X_2} = 0.04$g/L	$Y_{S_4,X_2} = 142.05$g/L
$K_{S_2,X_1} = 0.0224$g/L	$\mu^*_{3,max} = 3.50$g/day	$Y_{S_3,X_2} = 142.05$g/L
$K_{S_3,X_1} = 0.0159$g/L	$Y_{S_1,X_3} = 118.91$g/L	$Y_{S_2,X_3} = 149.48$g/L
$\mu^*_{p1} = 15.439 g X_1/g X_1$.day	$K_{S_1,X_3} = 0.00135$g/L	$Y_{S_2,X_2} = 149.25$g/L
$K_{p1} = 34.694$g/L	$K_{S_2,X_3} = 0.0224$g/L	$Y_{S_4,X_3} = 100$g/L
$Y^*_{p1} = 149.25 g X_2/g X_1$	$K_{S_4,X_3} = 0.0159$g/L	$S_{1in} = 50.20$ g/L
$\mu^*_{2,max} = 5.51$/day	$Y^*_{p2} = 149.25 g X_2/g X_3$	$S_{2in} = 541.70$ g/L
$K_{a,X_2} = 0.037$g/L	$\mu_{p2} = 15.439 g X_3/g X_3$.day	$S_{3in} = 1795.40$ g/L
$K_{S_4,X_2} = 0.0083$g/L	$K_{p2} = 34.694$g/L	$S_{4in} = 3668.70$ g/L
$Y_{S_2,X_1} = 149.48$ g/L	$Y_{S_1,X_1} = 118.91$ g/L	

Chapter 5

Modelling the control of energy partitioning during submaintenance feeding in cattle

5.1 Introduction

The utilization of energy by tissues (e.g., muscle, liver and mammary gland) depends partly on the partition of metabolizable energy (ME) among tissue compartments. Before ingested energy can be used for the synthesis of new tissue, certain essential demands to maintain existing tissue must be met (Lawrence and Fowler, 1997). Relative feeding level (RFL: RFL = 1 when ME intake is equal to requirements for ME for maintenance (A.R.C., 1980)) may modify this partition (Ferrell and Jenkins, 1985). However, the effects of feeding level on nutrient partition have mostly been studied with animals fed at levels above maintenance, but the impact of a change of nutrient priorities is obscured when nutrient supply is abundant (p242-245, Lawrence and Fowler, 1997). Animals respond differently to varying food availability and there are many factors (see the review of O' Donovan, 1984, and Table 1.1) that affect an animal's response during and following a period of undernutrition.

Animals adapt to undernutrition because the food required to maintain a constant body weight is not a constant function of weight but decreases with time due to a decrease in the weight of metabolically active tissues (Foot and Tulloh, 1977; Ledger and Sayers, 1977). One of the major factors that contribute to compensatory growth is a reduced maintenance requirement of animals during undernutrition and at the early stages of realimentation. This reduction in maintenance increases the energy available for growth and the extent of this contribution depends on the persistence of the reduced maintenance requirement during realimentation; the longer the reduction in maintenance persists, the greater the contribution to compensatory growth. A mechanism of compensatory growth has been postulated to be the persistent carry over of the reduced metabolic activity by the liver and gut during restriction and into realimentation (Shetty, 1990). When food intake is restricted, the metabolically active tissues, such as the digestive tract and the liver, are likely to be reduced in size and activity (Taylor and Murray, 1991). The liver and gut account for disproportionate amount of whole-body energy consumption compared with its relative mass of $< 10\%$ of body mass (Burrin *et al.*, 1989). Also, the splanchnic tissues contribute a substantial proportion of whole-animal energy expenditure (35-50%: Huntington,

1990) and changes in the plane of feeding are associated with changes in energy expenditure of the splanchnic tissues (Huntington, 1990), especially the liver and small intestine (Koong *et al.*, 1982). Liver mass, and thus energy requirement, has been reported to change dynamically with plane of nutrition (Ferrell and Koong, 1986). It is therefore of particular interest to test whether in the context of undernutrition the drop in energy expenditure of the liver and small intestine is an adaptive mechanism (Ortiques and Durand, 1995).

In this chapter the mechanistic model of intermediary metabolism of France *et al.* (1987) is modified to simulate the effects that changes in the relative absorption of acetate and propionate have on the weights of liver and small intestine and also on heat production. In order to evaluate the modified intermediary metabolism model, model results were compared with the experimental results of Ørskov and MacLeod (1993). In their experiment four steers were maintained wholly by intragastric infusion of VFA and protein. The infusion was given at three energy levels: 450, 675 and 900 kJ/kg liveweight$^{0.75}$, which was calculated to supply 1.0, 1.5 or 2.0 times the maintenance requirement. In addition, the effect of an energy level of 400 kJ/kg liveweight$^{0.75}$ on the heat production was examined.

The model is an aggregation of the catabolic and anabolic processes in metabolism and is based on carbon and nitrogen fluxes (Figure 5.1). The state variables are body ash (Ash, which is a surrogate for bone), glucose equivalents pool (C_6), body lipid pool (Lipid), Acetyl Coenzyme A equivalents pool (C_2), amino acid pool (N) and four protein pools: liver protein pool (Liv), small intestine protein pool (Si), muscle protein pool (Ms) and other protein protein pool (Op). There are three (3) blood metabolite pools (C_6, C_2, N) and six (6) body composition pools (Lipid, Ash, Ms, Liv, Op, Si).

5.2 General Model Structure

The most important end products of rumen digestion and fermentation are energy in the form of volatile fatty acids and nitrogen in the form of microbial protein and undegraded dietary protein. These end products are then absorbed from the digestive tract and enter cellular metabolism. The intermediary metabolism model described in this chapter considers the absorption of metabolites into the blood and deposition into various tissues. The following absorbed nutrients are considered as inputs to the model: the absorption of acetate (CH_3COOH), butyrate (C_3H_7COOH), long chain fatty acids (stearate: $C_{57}H_{110}O_6$), propionate (C_2H_5COOH), glucose ($C_6H_{12}O_6$) and amino acids which are denoted as A_{Ac}, A_{Bu}, A_{St}, A_{Pr}, A_{Gl} and A_{Aa}, respectively. The absorbed

nutrients from the rumen are expressed in moles/day. Since metabolism is considered only in terms of C_2, C_6 and amino acids, it is assumed that acetate, butyrate, stearate and part of propionate are metabolised as C_2, and glucose, the glycerol moiety of tristearin (stearate), and the remainder of propionate as C_6.

The absorbed energy (Absen) of the nutrients is calculated as follows:

$$\text{Absen (MJ/mole)} = \sum_{i=1}^{n} H_i A_i \tag{5.1}$$

where H_i is the heat of combustion per mole of substance and A_i is the quantity of the i^{th} absorbed nutrient. It is assumed that the heat loss during fermentation (Fermen) is 6% of the absorbed energy (Absen), that is, Fermen = 0.06 × Absen. The metabolisable energy (ME) of the diet is the fermentation heat loss plus the absorbed energy less the energy excreted as urea (that is, ME = Absen + Fermen − Urea).

In the description of each pool of the model, input and output equations are constructed and values for the parameters were sought from the literature as far as possible. Because this model is a modification of the model of France *et al.* (1987), parameter values and initial values of the state variables of their model were used. However, only additional and modified parameters and variable values will be explained in the text and a list of all the formulae are given in Appendix 5.1. Also, this model will follow the notation given in France *et al.* (1987). The calculations account for the energy being produced, which is subtracted from the C_2 energy required for maintenance. Glucose is converted to C_6 carbon and amino acids are converted to nitrogen and both these fluxes are assumed not to require or produce energy. Amino acids are assumed to contain 160 grams of nitrogen per kilogram. The relative molecular mass (RMM) of amino acid is assumed to be 110 and the RMM of tristearin is assumed to be 890. Differences between essential and non-essential amino acids are not considered in the model because it is assumed that the amino acid composition of isolated rumen micro-organisms is relatively constant (Storm and Ørskov, 1983). The broad objectives of the mechanistic model are to develop dynamic mechanistic elements representative of the growth process that will enable:

1. The simulation of the change of body weight and composition (including specific organs) of an animal during and following a period of sub -maintenance feeding.
2. The simulation of body composition (and specific organ) changes of animals fed to maintain a constant body weight.

3. The understanding of the mechanisms involved during undernutrition and realimentation.

5.2.1 Assumptions and hypothesis of the model

The model developed by France *et al.* (1987) was developed based on the assumption that the maintenance energy is a function of body weight only. In order to describe the partition of energy during and following a period of undernutrition, three changes were made to the intermediary metabolism model of France *et al.* (1987).

(a) Total basal metabolism (TBM) is a function of the mass of the liver (LIVT), mass of the small intestine (SIT), lipid (Lipid) pool, and lean body (Lbody).

To account for the contribution of the visceral organs to basal metabolism the aggregated protein pool was divided into four: liver protein (Liv) pool, small intestine protein (Si) pool, muscle protein (Ms) pool and other protein protein (Op) pool. This was done to account for the large energy expenditure of the visceral organs, especially the liver and small intestine (Koong *et al.*, 1982). Metabolizable energy expenditure for maintenance (ME_m) was shown to be closely correlated (r = 0.96) with the weights of visceral organs in growing cattle (Ferrell and Jenkins, 1984) and sheep (Ferrell *et al.*, 1986; Burrin *et al.*, 1990). Consequently, total basal metabolism (TBM) was divided as follows:

$$TBM = a\,(LIVT)^{0.67} + b\,(SIT)^{0.67} + c'\,[c(Lbody)^{0.67} + d\,(Lipid)^{0.67}] \qquad (5.2)$$

TBM	Total Basal Metabolism (MJ)
LIVT, SIT	Mass of liver (kg) = 2.38 × Liv Mass of small intestine (kg) = 2.38 × Si (Searle and Griffiths, 1983)
a, b, c, d	Parameters ($a = 2.602$, $b = 1.092$, $c = 0.3804$, $c' = 0.016$, $d = 0.1676$). All units are in MJ/kg.
Lbody, Lipid	Lean body weight (kg)[this excludes the liver and small intestine], fat (kg)

The parameters were estimated by firstly estimating the total basal metabolism as $0.53 \times (\text{Liveweight})^{0.67} = 21.42$ MJ/day for a 250 kg steer (p96, ARC, 1980). It is assumed that 25%, 12% and 63% of the total basal metabolism is accounted for by the liver, small intestine and viscera-free tissue, respectively (Koong *et al.*, 1982). Therefore, the energy expenditure of the liver will be 5.36 MJ/day, 2.57 MJ/day for

the small intestine, and 13.50 MJ/day for the viscera-free tissue for a 250kg steer. The parameter values will vary depending on the liveweight of the animal and must be adjusted accordingly. However, when evaluating the model with the data of Ørskov and MacLeod, 1993), we assumed that these parameters were constant because of the relatively short time period of their experiment. Therefore, assuming a liver mass of approximately 3kg for a 250 kg steer (Taylor and Murray, 1991), $a = \frac{5.36}{(\text{LIVT})^{0.67}} = 2.57$. Similarly, $b = 1.015$ and $c' = 0.016$. The viscera-free tissue consists of the lean body (excluding the liver and small intestine) and the lipid. From Baldwin *et al.* (1987), $d = \frac{c}{2.2713}$ and c and d can be calculated as 0.3314 and 0.1459, respectively.

(b) Taylor *et al.* (1981) indicated that animals attained various equilibrium states at different feeding levels. Taylor and Murray (1991) postulated that the liver is affected by feeding level and that the adaptation of the liver to undernutrition is complete before the adaptation by the body weight. That is, the liver attains its equilibrium weight before body weight changes significantly.

For this reason an equation relating equilibrium body weight (EQBW) and food intake (kg/day) was estimated from the data of Taylor *et al.* (1981). The maximum liver protein weight (Livm) and maximum small intestine protein weight (Sim) were made functions of equilibrium body weight:

$$\text{EQBW} = 66.75 \times (\text{Weight})^{0.96} \quad (5.3a)$$

$$\text{Livm} = \frac{m_1 \times (\text{EQBW})}{2.38} \quad (5.3b)$$

$$\text{Sim} = \frac{m_2 \times (\text{EQBW})}{2.38} \quad (5.3c)$$

EQBW	equilibrium body weight (kg)
Livm	maximum liver protein weight (kg)
Sim	maximum small intestine protein weight (kg)
m_1, m_2	parameters ($m_1 = 0.022189$, $m_2 = 0.025145$)

In addition to the digestive and metabolic effects, total splanchnic tissues also affect nutrient partitioning via the production and the metabolism of a number of hormones. Two general hormones having anabolic (H_A) or catabolic (H_C) functions were defined on the assumption that in many cases hormonal state is influenced by the concentration of circulating glucose (Baldwin *et al.*, 1987):

$$H_A = \left[\frac{C_{gl}}{C_{glref}}\right]^2 \quad (5.4a)$$

$$H_C = \left[\frac{C_{glref}}{C_{gl}}\right]^2 \quad (5.4b)$$

where C_{gl} is the current glucose concentration and C_{glref} is the reference glucose concentration (0.036 kg carbon per m^3 blood). Catabolic hormones affect the rate of lipolysis and gluconeogenesis and the anabolic hormones affect the rate of protein synthesis.

5.3 Model Description

In the description of each compartment that follows, input and output equations are constructed and values for the parameters (if appropriate) were either sought from the original model (France *et al.*, 1987) or from the literature as far as possible.

Glucose equivalent metabolite pool, C_6 (kg carbon per m^3 blood)

The inputs to the C_6 pool are from absorbed propionate (A_{Pr}), absorbed glucose (A_{Gl}), absorbed stearate (A_{St}), the production of C_6 via gluconeogenesis from amino acids (P_{C_6,NC_6}, AA→C_6 transaction) and the release of glycerol in the Lipid→C_2 transaction (lipolysis: P_{C_6,LC_2}). Outputs from the C_6 pool are the breakdown of glucose to the C_2 pool (glycolysis: U_{C_6,C_6C_2}) and the utilization of C_6 as glycerol and NADPH for lipogenesis (U_{C_6,C_2L}).

Acetyl Coenzyme A equivalent metabolite pool, C_2 (kg carbon per m^3 blood)

The C_2 pool serves both as a carbon and ATP source. The inputs to the model are by absorbed acetate (A_{Ac}), absorbed propionate (A_{Pr}), absorbed butyrate (A_{Bu}), absorbed stearate (A_{St}) and from gluconeogenesis (P_{C_2,NC_6}), glycolysis (P_{C_2,C_6C_2}) and lipolysis (P_{C_2,LC_2}). The outputs from the C_2 pool are to provide energy for ash synthesis (U_{C_2,C_2A}), to provide energy and fatty acid for lipogenesis (U_{C_2,C_2L}), energy for muscle protein synthesis ($U_{C_2,NMs}$), liver protein synthesis ($U_{C_2,NLiv}$), small intestine protein synthesis ($U_{C_2,NSi}$), other protein protein synthesis ($U_{C_2,NOp}$), to satisfy the energy needs for maintenance (U_{C_2,C_2M}), and to account for energy utilization via substrate cycling (U_{C_2,C_2X}).

The requirement for energy by the protein pools were calculated as a percentage of the total protein requirement. The requirement of C_2 units for protein synthesis was set at the carbon equivalent of 5 mol ATP (Millward *et al.*, 1976), even though this may be

an under-estimate. Therefore, $R_{C_2,NP} = 0.0909$ kg C_2 carbon per kg protein. The requirement of C_2 for liver and small intestine protein synthesis is assumed to be 40% of $R_{C_2,NP}$, i.e., $R_{C_2,NLiv} = R_{C_2,NSi} = 0.03636$ kg C_2 per kg liver and small intestine protein, respectively.

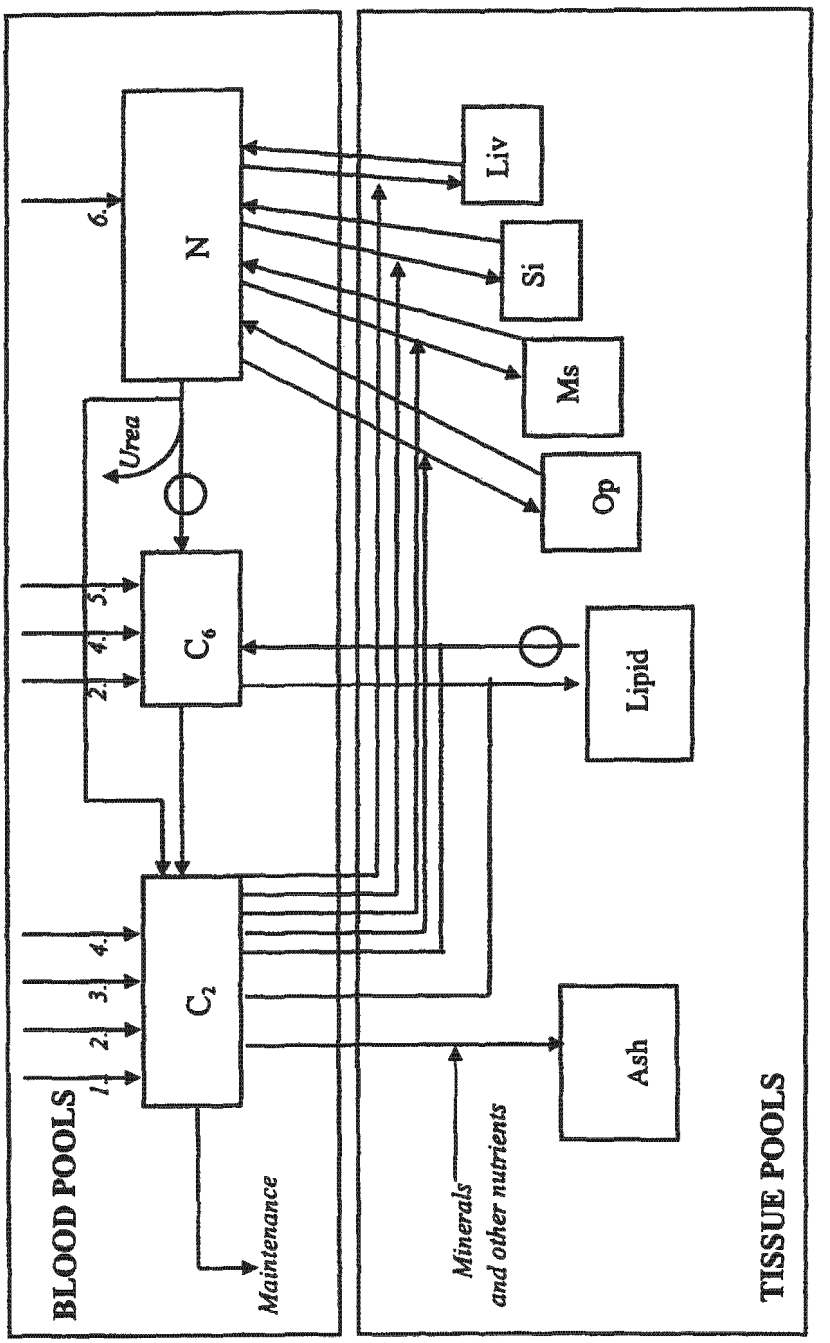


Figure 5.1: An aggregated intermediary metabolism model (modified from France *et al.*, 1987). The absorption of 1. Acetate, 2. Stearate, 3. Butyrate, 4. Propionate, 5. Glucose and 6. Amino acids is shown.

Similarly, the requirements of C_2 for muscle and other protein protein synthesis is assumed to be 10% of $R_{C_2,NProt}$, that is, $R_{C_2,NMs} = R_{C_2,NOp} = 0.00909$ kg C_2 per kg muscle and other protein protein, respectively.

The total energy expenditure of C_2 to meet basal metabolism (P_{M,C_2M}) is the difference between the total basal metabolism (TBM) and the energy produced in converting absorbed nutrients to metabolites:

$$P_{M,C_2M} = TBM - (P_{M,A_{Bu}} + P_{M,A_{St}} + P_{M,A_{Pr}} + P_{M,C_6C_2} + P_{M,NC_6} + P_{M,LC_2}) \quad (5.5)$$

The TBM is calculated to account for the effect of the variable changes in tissue (Burrin *et al.*, 1990)(see equation 5.2). An allometric relation with an exponent of 0.67 is assumed (based on the empirical equation of Kleiber, 1932) between the energy expenditure and the liver weight (LIVT), small intestine weight (SIT) and viscera-free weight, which consists of the lean body (Lbody, excluding the weight of liver and small intestine) and lipid (Lipid) (see equation 5.2). E_{C_2} is the enthalpy value of C_2 metabolite based on acetate and is equal to 38.65 MJ/kg carbon (McDonald *et al.*, 1981).

Amino Acid Pool, N (kg nitrogen per m^3 blood)

The inputs to this pool are absorbed amino acids ($P_{N,Aa}$) and protein turnover from the four protein pools: the turnover of liver protein ($P_{N,LvN}$), the turnover of small intestine protein ($P_{N,SiN}$), the turnover of muscle protein ($P_{N,MsN}$) and the turnover of other protein protein ($P_{N,OpN}$). The yield factors for dietary amino acids and protein breakdown from the four protein pools assume the nitrogen content of amino acid to be 160 g/kg on a weight basis, that is, $Y_{N,Aa} = 0.160g \times 0.110 = 0.0176$ kg nitrogen per mole amino acid (France *et al*, 1987). The yield of nitrogen from the accumulative protein pools ($Y_{N,PN}$) was calculated as 0.16 kg nitrogen per kg protein. The yields of nitrogen from the Liv, Ms, Si and Op pools were assumed to be 40%, 10%, 40% and 10% of the total protein pool (P), respectively, that is, $Y_{N,LvN} = 0.4 \times Y_{N,PN} = 0.064 = Y_{N,SiN}$ and $Y_{N,MsN} = 0.1 \times Y_{N,PN} = 0.016 = Y_{N,OpN}$.

Outputs are via gluconeogenesis to the C_6 pool (with a proportional contribution to acetate and urea), U_{N,NC_6}, and also protein synthesis to the liver protein, small intestine protein, muscle protein and other protein protein pools. Because amino acids are assumed to contain 160g nitrogen per kg, $R_{N,NP} = 0.16$ kg nitrogen per kg amino acid

(France *et al*, 1987). It is assumed that the requirements for the four protein pools are as follows: $R_{N,NLv} = 0.4 \times R_{N,NP} = R_{N,NSi} = 0.064$ and $R_{N,NMs} = 0.1 \times R_{N,NP} = R_{N,NOp} = 0.016$.

Body Ash Pool, Ash (kg)

The body ash pool is a surrogate for the skeleton and refers to the minerals incorporated into the skeleton. Skeletal growth is assumed to be irreversible (Millward, 1995) and therefore there is no output from this pool. The only input to the ASH pool is the production of ASH (kg) from the C_2 pool, P_{A,C_2A}.

Body Lipid Pool, Lipid (kg)

The rate of lipid deposition, and hence hypertrophy, depends on the relative rates of esterification and lipolysis. The only input to this pool is P_{L,C_2L} (kg lipid per day), which is the rate of lipogenesis from C_2 and C_6. The C_2 pool supplies acetyl CoA, ATP and one sixth of the requirements for NADPH (Gill, 1984). The C_6 pool supplies the remaining NADPH and glycerol. The only output from the body lipid pool is U_{L,LC_2}, the rate of triglyceride breakdown via lipolysis to C_2.

The rate of lipolysis, v_L = 0.05 kg/kg body lipid/hour. It is well established that plasma non-esterified fatty acid (NEFA) concentrations are markedly increased during acute energy restrictions in ruminants (Anniston, 1991). This is due to the increased mobilization of adipose tissue in the form of NEFA. From the work of Dunshea and Bell (1988), the influence of prolonged underfeeding on lipid catabolism is due more to a decrease in lipogenesis and intracellular reesterification than to increased lipolysis. More recently, Dawson *et al.* (1998) examined the effects of growth hormone on the flux of [C^{14}] palmitate and found that the flux increased (with growth hormone) when the animal was fed sub-maintenance and concluded that growth hormone had little or no effect on the flux in cattle fed at or above maintenance. A general catabolic hormone (H_C) was added to increase the rate of lipolysis when the animal is fed sub-maintenance. The syntax of the hormone term in the programme allows for the hormone to have no effect on lipolysis when the animal was fed at or above maintenance.

Protein pools

The splanchnic bed, comprising the gastrointestinal tract and liver, plays a pivotal role in moderating the pattern of nutrients available for peripheral tissues. The intestinal

tissues form an interface between the diet and the animal and have a direct influence on the flux of nutrients from the rumen into the bloodstream. The liver forms the central metabolic junction, further moderating and distributing nutrients to peripheral tissues for maintenance and productive functions such as muscle protein synthesis. Because of the importance of these tissues we have divided the protein pool of France *et al.* (1987) into four protein pools to accommodate for the variable energy expenditure of these tissues. The synthesis and breakdown rates of the protein pools were estimated from Lobley (1978).

a. Liver Protein Pool, Liv (kg)

There is only one input to the liver protein pool, that is, from the amino acid pool ($P_{N,NLv}$). The total body protein production was 1.55 kg/day (Lonsdale, 1976), which is the sum of the net synthesis and breakdown at a body protein value of 16.62 kg (France *et al.*, 1987). These values were used to calculate the rate constant for the synthesis of protein of 0.19 kg protein per kg protein per day (France *et al.*, 1987). The maximum rate of liver protein synthesis (k_{Liv}) is 0.076 liver protein per kg protein per day and is taken to be 40% of the total body protein synthesis (k_p), which was estimated from the carcass protein and energy retention data of Lonsdale (1976). There is much evidence of the marked effect of feeding level on liver proportion, for example, McMeekan (1940) found that the liver proportion on a low plane of nutrition was 0.77 times that on a high plane of nutrition. Baldwin *et al.* (1980) expressed liver proportion as a logistic function of energy intake and Koong *et al.* (1982) showed that liver weight was strongly correlated with daily food intake. However, Taylor and Murray (1991) showed that liver proportion was largely unaffected by feeding level when an equilibrium body weight was attained. To account for the apparent contradiction, they argued that the liver weight is approximately directly proportional to the new level of food intake, that is, the liver will have a rapid response to a change in feeding level until its equilibrium weight is attained. From the result of the experiment of Taylor *et al.* (1981), the relation between the equilibrium body weight and food intake was estimated to be

$$ln(\text{Weight}) = 4.219 + 0.96 \times ln(\text{Food/day}). \qquad (5.6a)$$

Because the cattle were fed a diet of 9.3 MJ/kg, the equilibrium body weight equation becomes

$$ln(\text{Weight}) = 4.219 + 0.96 \times ln(\frac{\text{ME}}{9.3}). \qquad (5.6b)$$

Taking antilogs of equation 5.6b gives us a power function for the equilibrium body weight, which is a function of the ME of the diet. The proportion of the liver per kg equilibrium body weight (0.0097g liver per kg equilibrium body weight) was calculated from Gibb *et al.* (1992). θ_{Liv}, the steepness parameter, was set to 1 assuming no immediate switch off characteristic of the reaction. Because of the rapid response of liver to feed intake, the affinity constant, $K_{N,NLv}$, was assumed to be 10% of the affinity constant for the protein pool ($K_{N,NP}$: 0.013 kg protein per m^3 blood), that is, $K_{N,NLv} = 0.1 \times 0.013 = 0.0013$. Burrin *et al.* (1990) also showed that the liver oxygen consumption rates increased rapidly with increasing feed intake, indicating the high priority of the liver for energy. Therefore, $K_{C_2,NLv} = 0.1 \times K_{C_2,NP} = 0.1 \times 0.008 = 0.0008$.

The only output from the liver protein pool is the liver protein breakdown ($U_{N,LvN}$) to produce amino acid. v_{Liv} was assumed to be 40% of the breakdown of protein, that is, $v_{Liv} = \frac{0.4 \times 0.19}{24} = 0.003167$ per hour.

b. Small Intestine Protein Pool, Si (kg)

The rate of small intestine protein synthesis was assumed to be 40% of the total body protein synthesized: $k_{Si} = \frac{0.4 \times 0.19}{24} = 0.0033167$ per hour. The proportion of the small intestine protein (g per kg equilibrium body weight) was calculated from Gibb *et al.* (1992) as 0.00904g small intestine protein per kg equilibrium body weight. θ_{Si} was assumed to be 1 and the rate of breakdown of small intestine protein was assumed to be 40% of the breakdown of protein, that is, $v_{Si} = \frac{0.4 \times 0.09}{24} = 0.0015$ per hour. Based on the same argument of priority of the liver protein pool, the affinity constant of small intestine protein for amino acid ($K_{N,NSi}$) was assumed to be 10% of the affinity constant for the protein pool ($K_{C_2,NP}$): $K_{N,NSi} = 0.1 \times 0.013 = 0.0013$ kg protein per m^3 blood. Also, $K_{C_2,NSi} = 0.1 \times K_{C_2,NP} = 0.1 \times 0.008 = 0.0008$ kg protein per m^3 blood.

c. Muscle Protein pool, Ms (kg)

There is only one input to the muscle protein pool, that is, the production of muscle protein from the N pool ($P_{N,NMs}$). The rate of muscle protein synthesis, k_{Ms}, is taken as 10% of the total protein synthesis (0.00079 per hour) (Lobley 1978). $K_{N,NMs}$ was estimated at 40% of $K_{N,NP}$, which was set equal to half the normal amino acid concentration, that is, 0.4 × 0.031 kg nitrogen per m^3 blood = 0.0124 kg nitrogen per m^3 blood (France *et al.*, 1987). $K_{C_2,NP}$ was calculated relative to the affinity constants

for lipid and ash and, therefore, $K_{C_2,NMs}$ was estimated as $0.4 \times K_{C_2,NP} = 0.4 \times 0.008 = 0.0032$. The steepness parameter (θ_{ms}) was set at 1.

The only output from the muscle protein pool is to the N pool ($U_{N,MsN}$). The rate of utilization of muscle protein was taken to be 10% of the rate of utilization of total protein ie, that is, $v_{Ms} = \frac{0.1 \times 0.09}{24} = 0.000375$ per hour (Lobley, 1978).

d. Other Protein Pool, OP (kg)

The only input to this pool is the production of protein from the amino acid pool, ($P_{N,NOp}$) and k_{Op} was calculated as $0.1 \times k_p = \frac{0.1 \times 0.19}{24} = 0.00079$ per hour (Lobley, 1978). $K_{N,NOp}$ was estimated as 40% of $K_{N,NP}$, which was set equal to half the normal amino acid concentration, that is, 0.4 × 0.031 kg nitrogen per m^3 blood = 0.0124 kg nitrogen per m^3 blood. $K_{C_2,NP}$ was calculated relative to the affinity constants for lipid and ash (as described in France *et al.*, 1987), therefore, $K_{C_2,NOp}$ was calculated as $0.4 \times K_{C_2,NP} = 0.4 \times 0.008 = 0.0032$. θ_{Op} is the steepness parameter and was set at 1. The maximum weight of the other protein protein pool (Opm) is set at 45 kg.

The output from this pool is the utilization of protein to the amino acid ($U_{N,OpN}$). The rate constant of this utilization (v_{Op}) is calculated as $\frac{0.1 \times 0.09}{24} = 0.000375$ per hour (Lobley, 1978).

5.4 Application of the model

Much controversy exists over the differences of the utilization of ME observed between roughage and concentrate diets. McClymont (1952) suggested that the inefficient utilization of acetic acid and the fact that the proportion of acetic acid in the rumen is greater with roughage diets are the reasons for the differences in ME utilization. However, Ørskov *et al.* (1979) showed that there were no differences in the utilization of different VFA by intragastric infusions. Ørskov *et al.* (1991) found no differences in animal metabolism and energy utilization when the proportions of VFA (acetic and propionate) were varied within their normal rumen range. The model described in this chapter was evaluated with experimental data of Ørskov and MacLeod (1993), where steers were wholly fed with constant energy levels by intragastric infusion. The effect of four energy levels on the partition of energy in intermediary metabolism and on energy expenditure for maintenance was examined. In addition, the effect of the relative proportion of acetic and propionic acid on the

partition of energy in intermediary metabolism and on heat production was examined. The model was run using DRIVER (Hahn & Furniss, 1988).

5.4.1 The effect of four constant energy levels on blood metabolite pools and the partition of energy

The model was used to simulate the effect of four constant volatile fatty acid input levels on the partition of energy in intermediary metabolism. The proportion of volatile fatty acid used as input is given in Table 5.1.

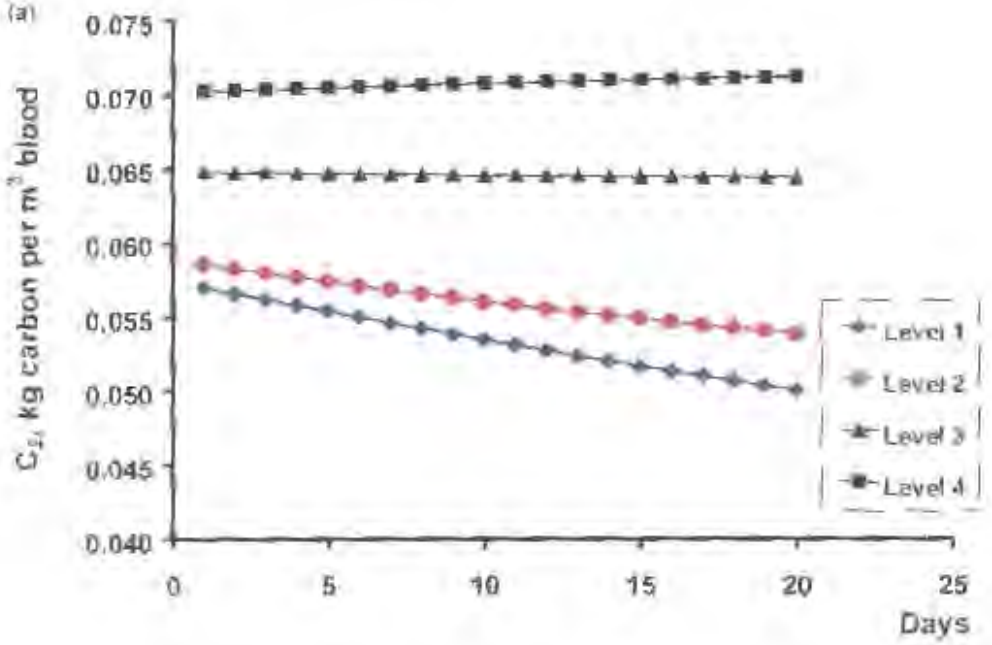
Table 5.1: The input of volatile fatty acid at four different energy levels: Energy Level 1 (25.15 MJ), Energy Level 2 (28.30 MJ), Energy Level 3 (42.44 MJ), Energy Level 4 (56.58 MJ)

	Energy level 1	**Energy level 2**	**Energy level 3**	**Energy level 4**
A_{Ac}	14.01	15.76	23.64	31.52
A_{Pr}	3.96	4.46	6.69	8.92
A_{Bu}	1.54	1.73	2.59	3.46
A_{Aa}	1.55	1.714	2.62	3.49

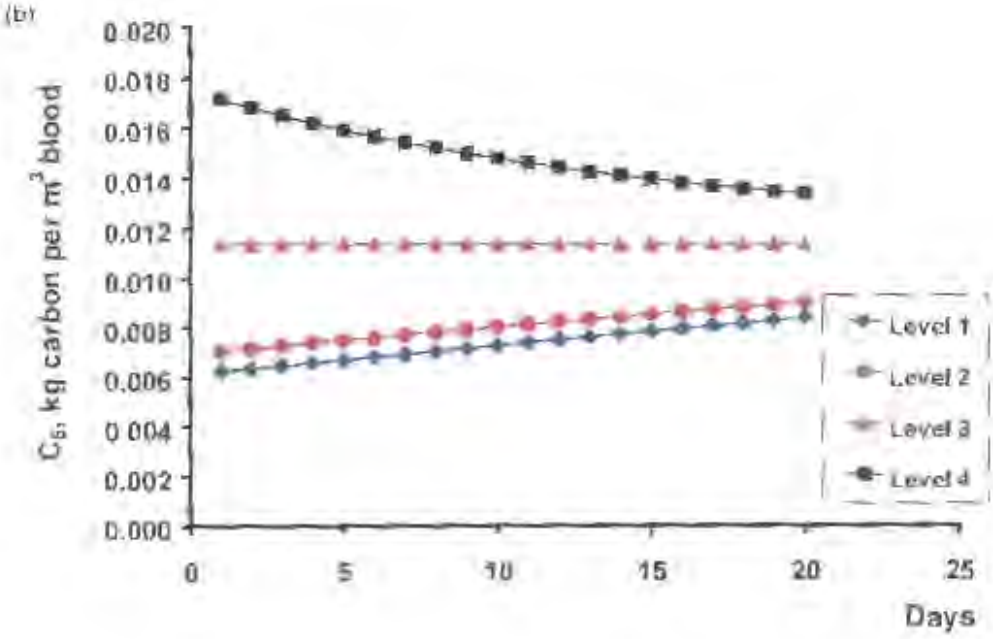
The model was run for twenty days with $\Delta t = 1$ day and the concentration of the blood metabolite pools (C_6, C_2) and the size of body composition pools (Lipid, Liver) were recorded. In addition, the partition of energy between particular tissues, the utilization of C_2 from C_2 to Lipid ($U_{C_2,C_{2L}}$), and the energy expenditure for maintenance (TBM), was examined.

At energy level 1, the C_2 pool decreased (Figure 5.2a) due to the low input of absorbed nutrients and due to the energy demand of the peripheral tissues. The combined effect of the decreased size of the splanchnic tissues and the increased utilization of lipid (Figure 5.2c) resulted in the decrease in the energy expenditure for maintenance (Figure 5.3c) and the increase in the size of the C_6 concentration at energy level 1 and 2 (Figure 5.2b). At higher energy input (Energy level 3 and 4) the concentration of the blood metabolites remained relatively constant or increased, respectively. Figure 5.4 shows the reactions (in bold) that increased the most at maintenance (Energy level 2) and below maintenance (Energy level 1).

(a)


0.075
0.070
0.065
0.060
0.055
0.050
0.045
0.040
C_{L1}, kg carbon per m^3 blood
0
5
10
15
20
25
Days
Level 1
Level 2
Level 3
Level 4

(b)


0.020
0.018
0.016
0.014
0.012
0.010
0.008
0.006
0.004
0.002
0.000
C_{L}, kg carbon per m^3 blood
0
5
10
15
20
25
Days
Level 1
Level 2
Level 3
Level 4

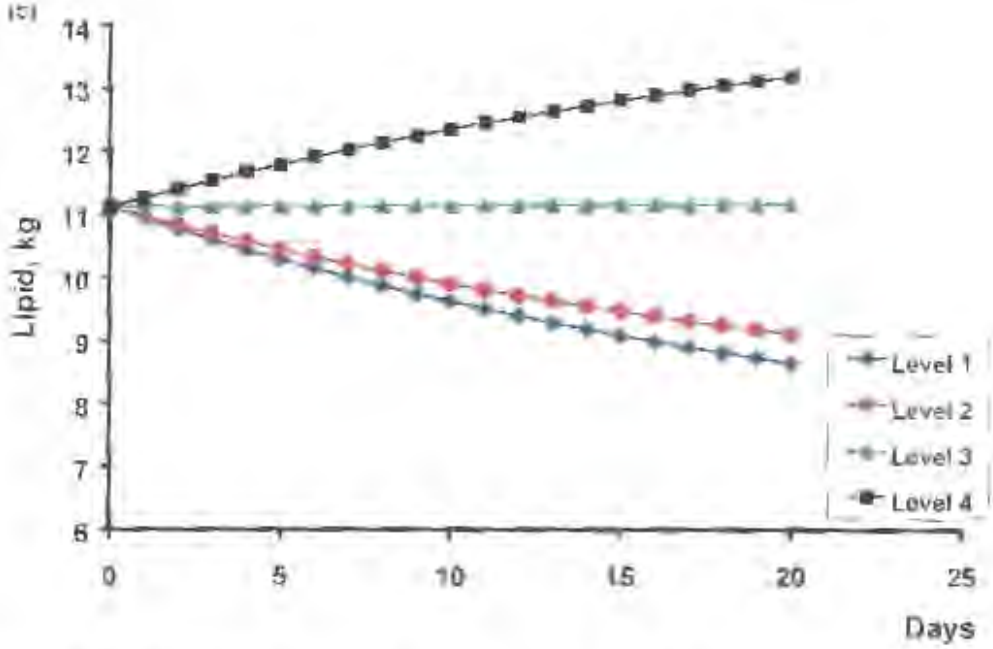
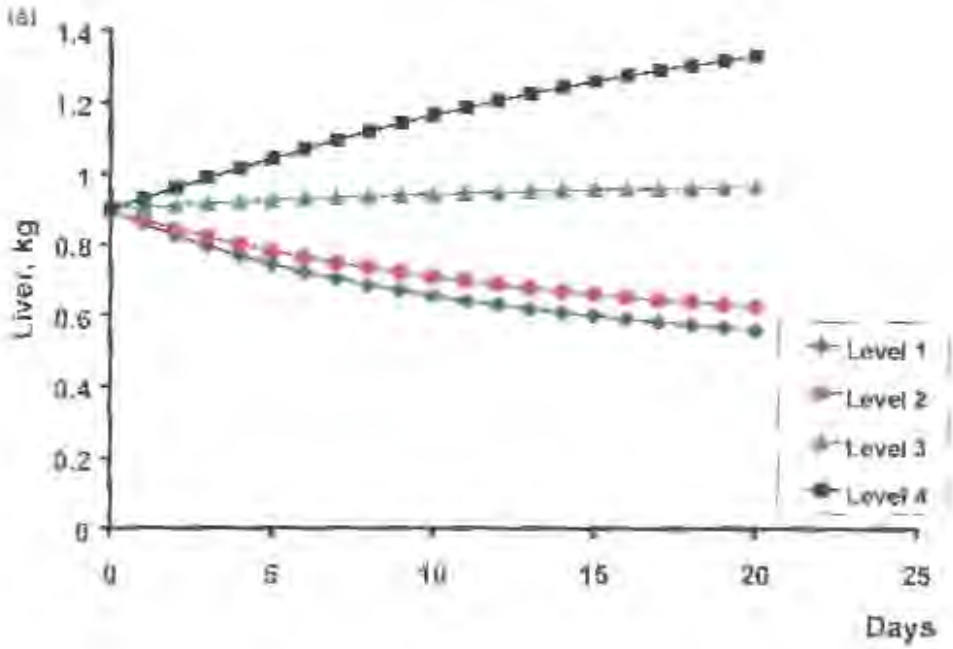


Figure 5.2: Model results at four constant energy levels: (a) C_1 (kg carbon per m^3 blood, (b) C_2 (kg carbon per m^3 blood, and (c) Lipid (kg).



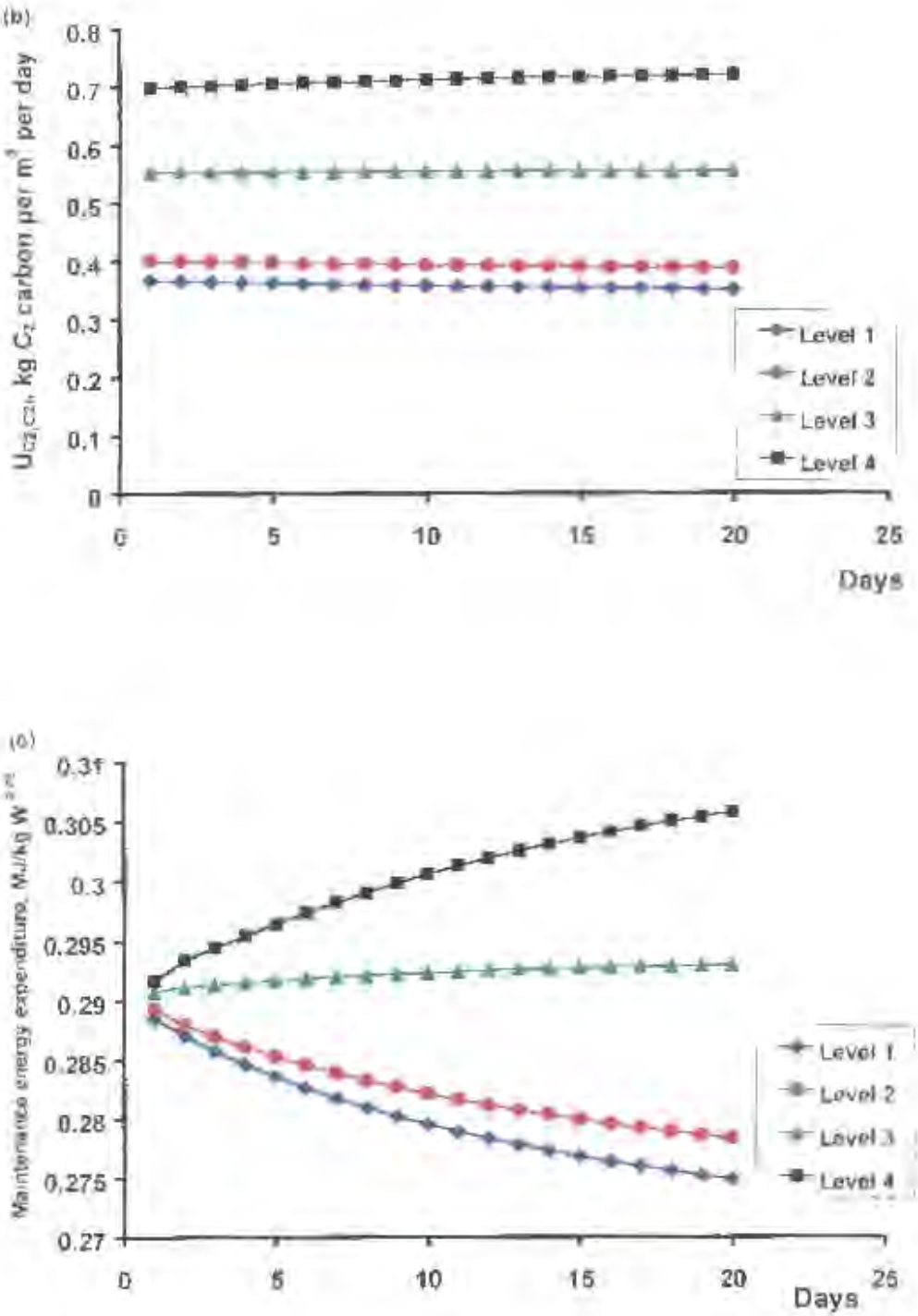


Figure 5.3: Model results: (a) Liver protein (Liv, kg), (b) $U_{C_2|C_2H}$, the utilization of C_2 lipid, and (c) TBM, the energy expenditure for maintenance.

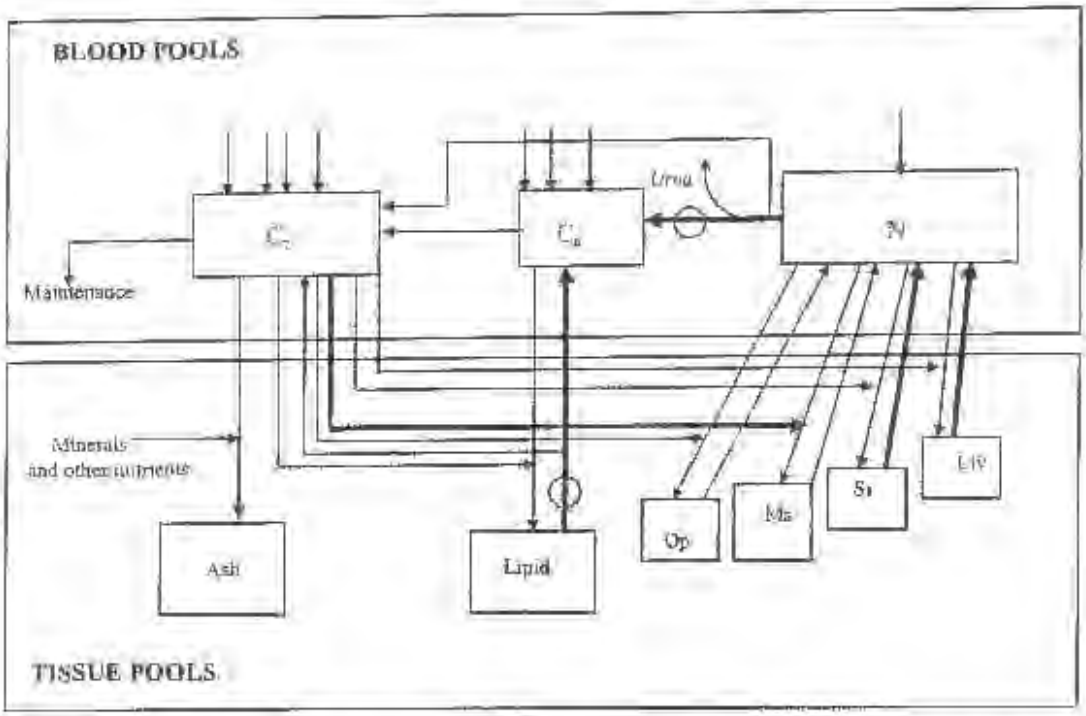


Figure 5.4: Block diagram showing the reactions (in bold) that increased the most for energy input at and below maintenance.

5.4.2 The effect of varying the relative proportion of acetate and propionic acid on heat production

Ørskov and MacLeod (1993) showed that the constant infusion of energy did not affect the point at which increasing the proportion of acetic acid in the infusion mixture results in glucose deficiency. They showed that the plasma glucose concentration (mmol/L) remained relatively constant until the proportion of acetate was about 75% and then the plasma glucose concentration decreased sharply. The model simulated a sharp decrease in plasma glucose concentration (Figure 5.5) but when the steepness parameter (θ_{C_6}) of the glycolysis reaction (U_{C_3,C_6C_2}) was increased, the model simulated the experimental data closely until the proportion of acetate was 45%, thereafter, increasing the steepness parameter had no effect on the sharp decline of plasma glucose concentration. France *et al.* (1987) set θ_{C_6} so that the rate of glycolysis is minimal until the concentration of C_6 is 1.5 times a standard concentration ($\theta_{C_6} = 5$). Increasing θ_{C_6} has the effect of a more rapid increase in the rate of the glycolysis reaction followed by a relatively constant rate of glycolysis.

Figure 5.6 shows a relatively constant heat production at the four energy levels, besides the increase in heat production from approximately 65% of acetate, which may be an artifact of the model since energy lost by a different route would be

included in the heat production. Orskov and Ryle (1990) found that high proportions of infused acetate would be excreted in the urine. Simulation results support the experimental data of Orskov and MacLeod (1993) that the proportion of propionic acid is more important than the levels at which it was infused.

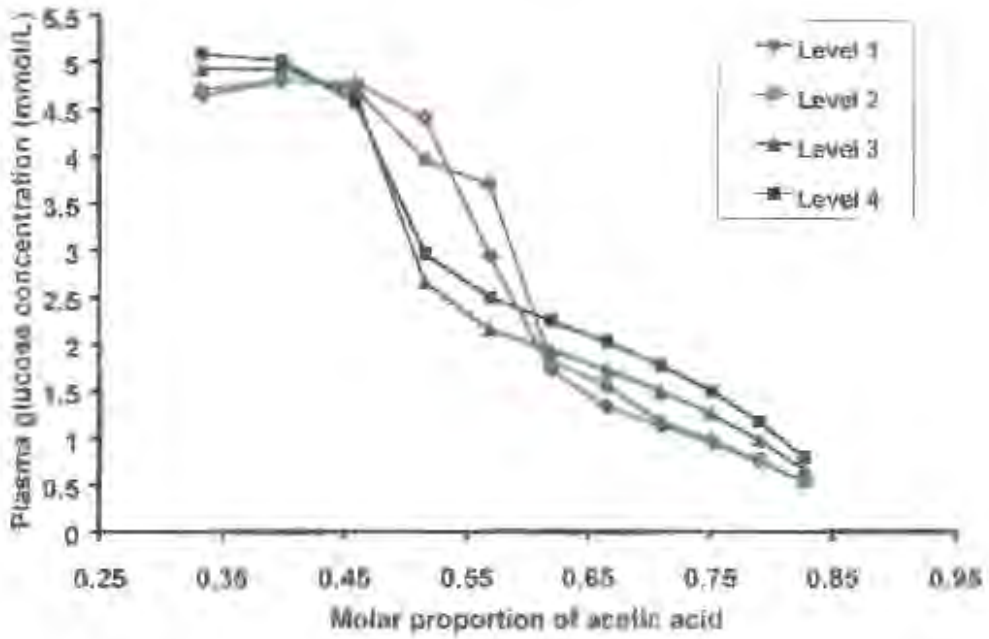


Figure 5.5: Model simulated plasma glucose concentration (mmol/l.)

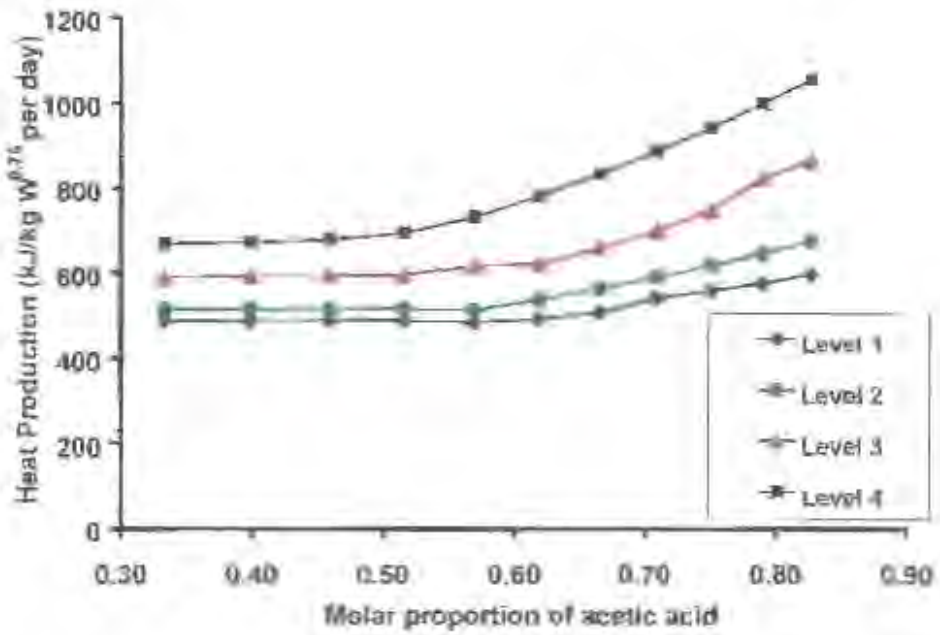


Figure 5.6: Model simulated heat production at four different energy inputs.

5.5 Conclusions

A simulation model was presented to analyze the effect that a constant input of energy per day over several days at four different energy levels had on the concentration of blood metabolites and the partition of energy. Of particular interest is the partition of energy to the liver and lipid pools. In addition, the effect of changing the proportion of acetic acid and propionic acid on the energy expenditure for maintenance and heat production was examined.

The analysis shows that the utilization of absorbed energy is unlikely to be affected by the proportion of absorbed VFA. This was also shown in experiments by Ørskov and MacLeod (1993). It seems as though the utilization of VFA is not dependent on the relative amounts and proportions of absorbed acetic and propionic acid but due to additional energy expenditure, such as rumination, or other differences in physical activity (Ørskov and Ryle, 1990). However, the model does demonstrate a decrease in glucose when absorbed acetic acid was increased but this seems to be entirely due to the decreased absorption of propionate. It could be possible that the yield of absorbed propionate is dependent on the relative proportion of acetic and propionic acid absorbed to the C_x pool, which will require further investigation.

APPENDIX 5.1

MODEL EQUATIONS

$$\text{Absen (MJ/mole)} = \sum_{i=1}^{n} H_i A_i \qquad (5.1)$$

$$\text{TBM} = a\,(\text{LIVT})^{0.67} + b\,(\text{SIT})^{0.67} + c\,[c(\text{Lbody})^{0.67} + d\,(\text{Lipid})^{0.67}] \qquad (5.2)$$

$$\text{EQBW} = 66.75 \times (\text{Weight})^{0.96} \qquad (5.3a)$$

$$\text{Livm} = \frac{m_1 \times (\text{EQBW})}{2.38} \qquad (5.3b)$$

$$\text{Sim} = \frac{m_2 \times (\text{EQBW})}{2.38} \qquad (5.3c)$$

$$H_s = \left[\frac{C_{gl}}{C_{gref}}\right]^2 \qquad (5.4a)$$

$$H_r = \left[\frac{C_{gref}}{C_g}\right]^2 \qquad (5.4b)$$

$$P_{MC_7M} = \text{TBM} - (P_{M,Apr} + P_{M,Ast} + P_{M,Ap} + P_{M,C_6} + P_{M,C_2} + P_{M,C_7}) \qquad (5.5)$$

$$ln(\text{Weight}) = 4.219 + 0.96 \times ln(\text{Food/day}) \qquad (5.6a)$$

$$ln(\text{Weight}) = 4.219 + 0.96 \times ln\left(\frac{\text{ME}}{9.3}\right) \qquad (5.6b)$$

Glucose equivalent metabolite pool, C_6 (kg carbon per m^3 blood)

INPUTS

$$P_{C_6,Apr} = Y_{C_6,Apr} \times A_{pr} \qquad (5.7a)$$

$$P_{C_6,Ad} = Y_{C_6,Agl} \times A_{gl} \qquad (5.7b)$$

$$P_{C_6,Ast} = Y_{C_6,Ast} \times A_{st} \qquad (5.7c)$$

$$P_{C_6,MC_6} = Y_{C_6,MC_6} \times U_{MC_6} \qquad (5.7d)$$

$$P_{C_6,LC_6} = Y_{C_6,LC_6} \times U_{LC_6} \qquad (5.7e)$$

OUTPUTS:

$$U_{C_6,C_6(i)} = \frac{v_{C_6} \times \text{Weight}}{1 + \left[\frac{K_{C_6,C_6(i)}}{C_6}\right]^{\theta_{C_6}}} \qquad (5.7f)$$

$$U_{C_6,C_2L} = R_{C_6,C_2L} \times P_{LC_2} \qquad (5.7g)$$

$$\frac{dC_6}{dt} = P_{C_6,Apr} + P_{C_6,Ad} + P_{C_6,Ast} + P_{C_6,MC_6} + P_{C_6,LC_6} - U_{C_6,C_6(i)} - U_{C_6,C_2L} \qquad (5.7)$$

Acetyl Coenzyme A equivalent metabolite pool, C_2 (kg carbon per m^3 blood)

INPUT:

$$P_{C_2,Aa} = Y_{C_2,Aa} \times A_{Ac} \quad (5.8a)$$

$$P_{C_2,Ap} = Y_{C_2,Ap} \times A_{Pr} \quad (5.8b)$$

$$P_{C_2,Ab} = Y_{C_2,Ab} \times A_{Bu} \quad (5.8c)$$

$$P_{C_2,as} = Y_{C_2,as} \times A_{S} \quad (5.8d)$$

$$P_{C_2,NC_2} = Y_{C_2,NC_2} \times U_{N,NC_2} \quad (5.8e)$$

$$P_{C_2,C_6C_2} = Y_{C_2,C_6C_2} \times U_{C_6,C_6C_2} \quad (5.8f)$$

$$P_{C_2,LC_2} = Y_{C_2,LC_2} \times U_{L,LC_2} \quad (5.8g)$$

OUTPUT:

$$U_{C_2,C_2L} = R_{C_2,C_2L} \times P_{L,C_2L} \quad (5.8h)$$

$$U_{C_2,C_2L} = R_{C_2,C_2L} \times P_{L,C_2L} \quad (5.8i)$$

$$U_{C_2,Mc} = R_{C_2,Mc} \times P_{Mc,Mc} \quad (5.8j)$$

$$U_{C_2,Mb} = R_{C_2,Mb} \times P_{Mb,Mb} \quad (5.8k)$$

$$U_{C_2,NMa} = R_{C_2,NMa} \times P_{Ma,NMa} \quad (5.8l)$$

$$U_{C_2,NOp} = R_{C_2,NOp} \times P_{Op,NOp} \quad (5.8m)$$

$$U_{C_2,C_2M} = \frac{P_{M,C_2M}}{E_{C_2}} \quad (5.8n)$$

$$U_{C_2,C_2X} = \frac{v_{C_2} \times W}{1 + \left[\frac{K_{C_2,C_2X}}{C_2}\right]^{\theta_{C_2}}} \quad (5.8o)$$

DIFFERENTIAL EQUATION:

$$\frac{dC_2}{dT} = P_{C_2,Aac} + P_{C_2,Apr} + P_{C_2,Abu} + P_{C_2,As} + P_{C_2,NC_2} + P_{C_2,C_6C_2} + P_{C_2,LC_2} - U_{C_2,C_2L} - U_{C_2,C_2X} - U_{C_2,C_2L} - U_{C_2,Mc} - U_{C_2,Mb} - U_{C_2,NMa} - U_{C_2,NOp} - U_{C_2,C_2M} \quad (5.8)$$

Amino Acid Pool, N (kg nitrogen per m^3 blood)

INPUT:

$$P_{N,Aaa} = Y_{N,Aaa} \times A_{Aa} \quad (5.9a)$$

$$P_{N,GN} = Y_{N,GN} \times U_{G,GN} \quad (5.9b)$$

$$P_{N,LN} = Y_{N,LN} \times U_{L,LN} \quad (5.9c)$$

$$P_{N,MaN} = Y_{N,MaN} \times U_{Ma,MaN} \quad (5.9d)$$

$$P_{N,OpN} = Y_{N,OpN} \times U_{Op,OpN} \quad (5.9e)$$

OUTPUT:

$$U_{N,NC_6} = \frac{v_N \times Weight \; \Delta W_t}{1 + \left[\frac{K_{N,NC_6}}{N}\right]} \quad (5.9f)$$

$$U_{N,NL} = R_{N,NL} \times P_{L,C_2L} \quad (5.9g)$$

$$U_{N,NSr} = R_{N,NSr} \times P_{Sr,SrN} \quad (5.9h)$$

$$U_{\text{[illegible]}} = R_{\text{[illegible]}} + P_{\text{[illegible]}} \quad (5.9i)$$

$$U_{\text{[illegible]}} = R_{\text{[illegible]}} + P_{\text{[illegible]}} \quad (5.9j)$$

DIFFERENTIAL EQUATION:

$$\frac{dAAN}{dt} = P_{\text{[illegible]}} + P_{N,SIN} + P_{\text{[illegible]}} + P_{\text{[illegible]}} + P_{\text{[illegible]}} - U_{\text{[illegible]}} - U_{\text{[illegible]}} - U_{N,NS} - U_{N,NAS} - U_{\text{[illegible]}} \quad (5.9)$$

Body Composition Pools

Body Ash Pool, Ash (kg)

INPUT: $P_{\text{[illegible]}} = \frac{\text{[illegible]}}{\text{[illegible]}}$ (5.10a)

where Amax $= \lambda_{\text{[illegible]}} \times$ Prot (5.10b)

OUTPUT: None

DIFFERENTIAL EQUATION:

$$\frac{dAsh}{dt} = P_{\text{[illegible]}} \quad (5.10)$$

Body Lipid Pool, Lipid (kg)

INPUT: $P_{\text{[illegible]}} = \frac{\text{[illegible]}}{\text{[illegible]}}$ (5.11a)

where Lmax $= \lambda_{\text{[illegible]}} \times$ Ash (5.11b)

OUTPUT: $U_{\text{[illegible]}} = v_{\text{[illegible]}} \times$ Lipid $\times H_c$ (5.11c)

DIFFERENTIAL EQUATION:

$$\frac{dLipid}{dt} = P_{\text{[illegible]}} - U_{\text{[illegible]}} \quad (5.11)$$

Liver Protein Pool, Liv (kg)

INPUT: $P_{\text{[illegible]}} = \frac{\text{[illegible]}}{\text{[illegible]}}$ (5.12a)

where Livm $= 0.0097 \times \left[87.966 \times e^{(0.96 \times \ln(\frac{MS}{87.1}))}\right]$ (5.12b)

OUTPUT: $U_{N,LvN} = v_{Lv} \times Liv$ (5.12c)

DIFFERENTIAL EQUATION:

$$\frac{d\,Liver}{dt} = P_{N,NLv} - U_{N,LvN} \quad (5.12)$$

Small Intestine Protein Pool, Si (kg)

INPUT: $$P_{N,NSi} = \frac{k_{Si} \times Si \times \left[(1-\left(\frac{Si}{Sim}\right)^{\theta Si}\right]}{1+\frac{K_{N,NSi}}{N}+\frac{K_{C_2,NSi}}{C_2}} \quad (5.13a)$$

$$\text{where } Sim = 0.00904 \times \left[67.966 \times e^{(0.96 \times ln(\frac{ME}{9.3}))}\right] \quad (5.13b)$$

OUTPUT: $U_{N,SiN} = v_{Si} \times Si$ (5.13c)

DIFFERENTIAL EQUATION:

$$\frac{d\,Si}{dt} = P_{N,NSi} - U_{N,SiN} \quad (5.13)$$

Muscle Protein pool, Ms(kg)

INPUT: $$P_{N,NMs} = \frac{k_{Ms} \times Ms \times \left[(1-\left(\frac{Ms}{Msm}\right)^{\theta Ms}\right]}{1+\frac{K_{N,NMs}}{N}+\frac{K_{C_2,NMs}}{C_2}} \quad (5.14a)$$

OUTPUT: $U_{N,MsN} = v_{Ms} \times Ms$ (5.14b)

DIFFERENTIAL EQUATION:

$$\frac{dMs}{dt} = P_{N,NMs} - U_{N,MsN} \quad (5.14)$$

Other Protein Pool, Op (kg)

INPUT: $$P_{N,NOp} = \frac{k_{Op} \times Op \times \left[(1-\left(\frac{Op}{Opm}\right)^{\theta Op}\right]}{1+\frac{K_{N,NOp}}{N}+\frac{K_{C_2,NOp}}{C_2}} \quad (5.15a)$$

OUTPUT: $U_{N,OpN} = v_{Op} \times Op$ (5.15b)

DIFFERENTIAL EQUATION:

$$\frac{dOp}{dt} = P_{N,Nop} - U_{N,OpN} \quad (5.15)$$

PART III

EVALUATION AND APPLICATIONS

become abundant, growth rates accelerate and exceed the growth rates of continuously well fed animals. However, the response of an animal to a period of undernutrition is variable, due partly to the complex processes of digestion and metabolism. The response depends (among other factors, see Table 1.1) on the severity and length of restriction, the age of the animal when the restriction was imposed and on the quality of the food during undernutrition. Partial compensation is possible when restricted animals increase their growth rate but do not attain the same weight for age as those animals not restricted. In some cases there is no increase in growth rate of a restricted animal once the restriction is removed, and hence there is no compensation.

Animals are able to adapt to undernutrition because the food needed to maintain a constant body weight is not a function of weight but decreases with time in response to low feed intake (Ledger and Sayers, 1977). One of the factors that contribute to compensatory growth is a reduced maintenance requirement of animals during undernutrition at the early stages of realimentation. This reduction in maintenance increases the energy available for growth and the extent of this contribution depends on the persistence of the reduced maintenance requirement during realimentation; the longer the reduction in maintenance persists, the greater the contribution to compensatory growth. When food intake is restricted, the metabolically active tissues, such as the digestive tract and the liver, are likely to be reduced in size and activity (Taylor and Murray, 1991; Burrin *et al.*, 1990).

Over the past fifteen years there has been considerable progress in modelling of both rumen and intermediary metabolism processes due to the increased availability of data and computer power, and more importantly, to the new concepts that have been formulated due to the increase in knowledge. However, two problems still occur (Witten and Richardson, 2000):

1. Most mathematical models are developed to simulate continuous growth and do not adequately predict animal responses to limited (sub-maintenance) feeding.
2. Although suggested by France *et al.* (1987) that the modelling focus should also be on the integration of components to increase adequate predictions, the integration of models of whole digestive processes and metabolism have been slow.

For these reasons, two mechanistic models, the rumen model described in chapter 3, and the intermediary metabolism model described in chapter 5, were linked: output from the rumen is stochiometrically converted to their C_6 and C_2 equivalents and used as input to the intermediary metabolism sub-model. The rumen model comprises 23 state variables and the intermediary metabolism model comprises 9 state variables. Four zero pools are defined for the post ruminal gastrointestinal tract, which represents nutrients available for absorption. Because the time scale of the rumen model is in hours and the time scale of the intermediary metabolism model in days, all the rate constants of the rumen model were multiplied by twenty four so that both models could be integrated simultaneously. This could be done because the output from the rumen model on the hourly and daily time scales gave similar output.

The aim of this chapter is to evaluate the linked rumen and metabolism model (RUMET = RUmen + METabolism) by simulating the effects of undernutrition and accounting for some of the factors that influence maintenance energy expenditure over time. The framework of the model is shown in Figure 6.1 and the scheme of the model is given in Figure 6.2. In this section the qualitative behaviour of the model will be assessed by behavioural analysis, sensitivity analysis and testing the behaviour of the model with input of different proportions of roughage and concentrate diets. In section 6.3 a quantitative assessment of the model will be carried out by challenging the model with the experimental data of Ryan *et al.* (1993), Foot and Tulloh (1977) and Ledger and Sayers (1977). The model was run using DRIVER (Hahn & Furniss, 1988) to simulate the pattern of weight and body composition changes of growing cattle.

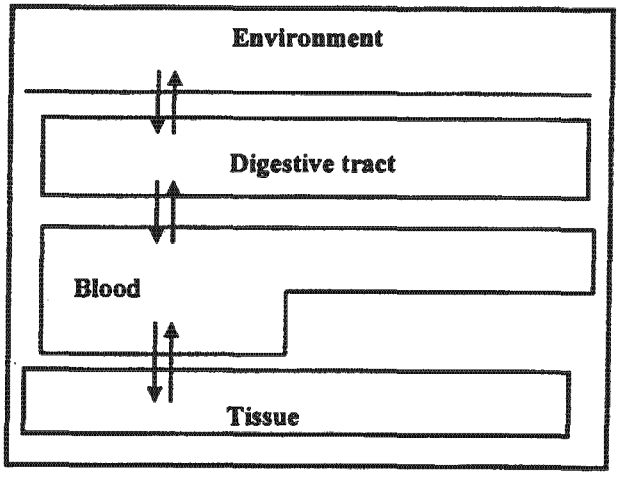


Figure 6.1: General framework for the RUMET Model

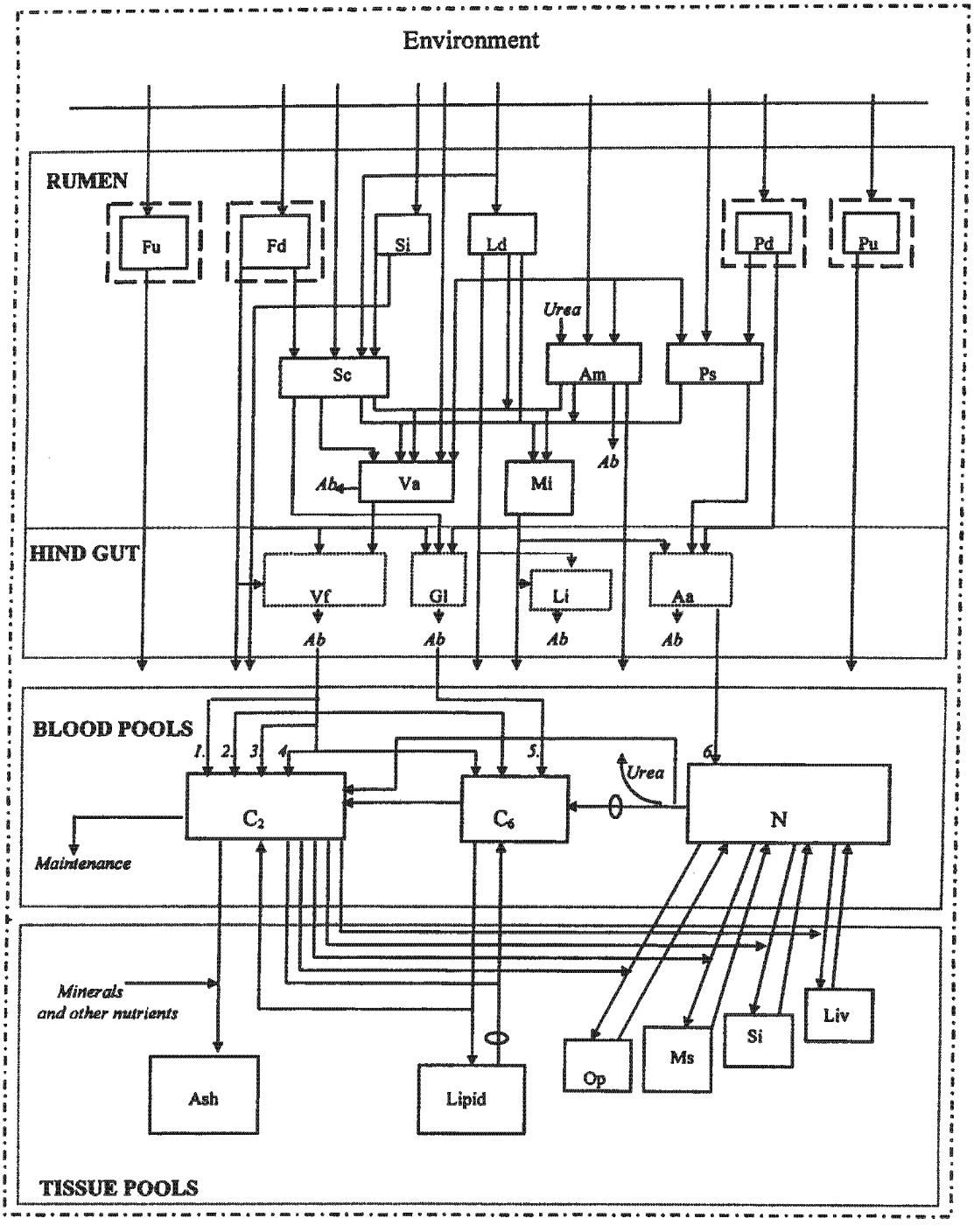


Figure 6.2: The RUMET model, an integrated rumen and intermediary metabolism model. The absorption of 1. Acetate, 2. Stearate, 3. Butyrate, 4. Propionate, 5.Glucose and 6. Amino acids are shown.

6.2.1 Simulation methodology

In order to test the qualitative behaviour of the model, the model was run to follow the scheme (Figure 6.3) below.

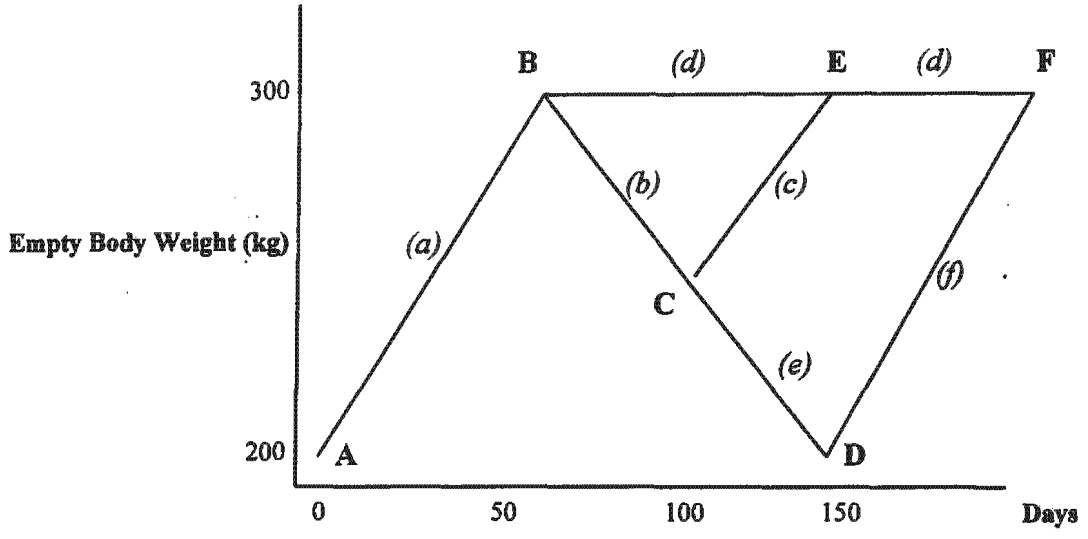


Figure 6.3: Simulation Plan.

The initial body weight for the model animal was set at 100 kg. During the preliminary stage, the model simulated the growth of an animal to 200 kg (point A) on a mixed diet of 50 % autumn grass (mature grass harvested at the end of the rainy season) and 50% concentrate (refer to estimated diet composition in Table 6.1). This diet was used as input to the model for the full duration of the simulation exercise. Point **A** was taken as the start of the simulation exercise. Growth path *(a)* shows the growth of the model animal fed ad libitum till point **B**, that is, till the model animal reached 300 kg. The model was then run to follow growth path *(b)* so that the model animal lost body weight at a rate of about 0.6 kg/day to point **C**, and was then refed ad libitum (growth path *(c)*) to reattain 300 kg empty body weight. The model was also run to simulate the prolonged deprivation of food (growth path *(e)*). During this pattern of weight change, the model animal continued to lose about 0.6 kg/day of its liveweight till point **D**. At this point the model was run to simulate the ad libitum feeding of the model animal until it reached 300 kg. The first restricted phase (from **B** to **C**) and the second restricted phase (from **C** to **D**) lasted for 50 days each. The model also simulated an animal fed to maintain a constant body weight (growth path *(d)*).

6.2.2 Behavioural analysis

The simulated patterns of growth by the model are shown in Figure 6.4. The food intake, body weight and body composition at each of the six points (**A, B, C, D, E, F**; labelled in Figure 6.4) are shown in Table 6.2. Following growth path *(b)* to point **C**, the model predicts a 10.27% weight loss due to a 39.2% decrease in food intake, that is, the dry matter intake was reduced from 5.74 kg/day to 3.49 kg/day (Figure 6.5) which corresponds to 79.0 g/kg $W^{0.75}$/day and 52.5 g/kg $W^{0.75}$/day, respectively.

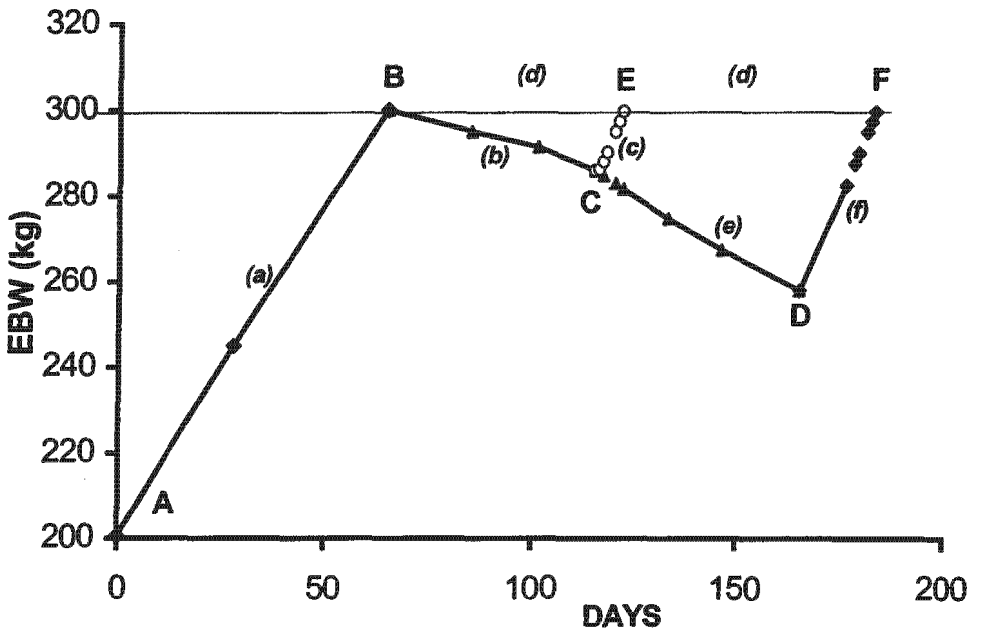


Figure 6.4: Simulated growth paths by the model.

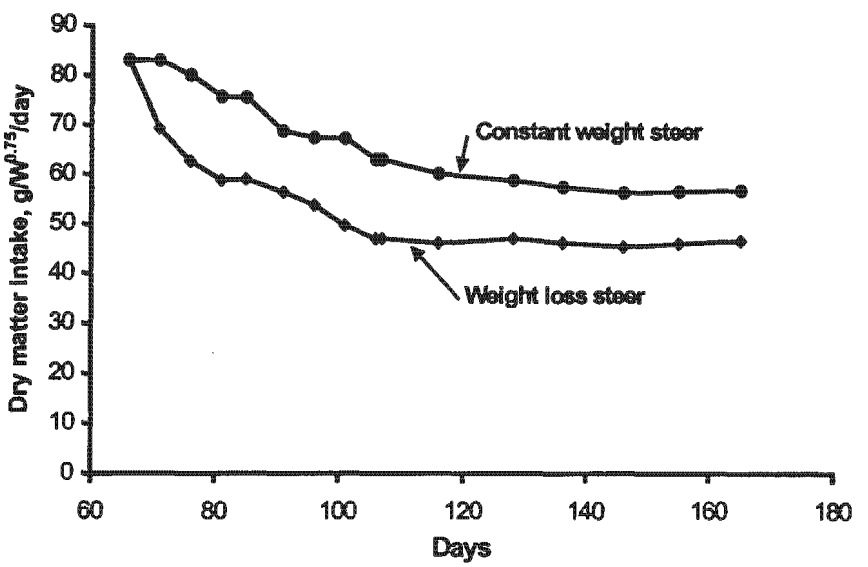


Figure 6.5: Simulation of dry matter intake (g/$W^{0.75}$/day) from **B** to **D.**

Following growth path *(b)*, ash continued to increase and muscle increased till day 10 of the 50 day weight loss path and then began to decrease. Therefore, the model

predicts a lag in muscle protein response to the decrease in dry matter intake. The visceral organs (liver and small intestine) responded immediately to the decrease in food intake and lost 73.0% and 67.0% of its protein weight during the first 50 days of weight loss, respectively. In the model equations, the basal metabolism is partly dependent on the weights of the visceral organs and we would expect a decrease in the maintenance energy expenditure during the weight loss period (Figure 6.6).

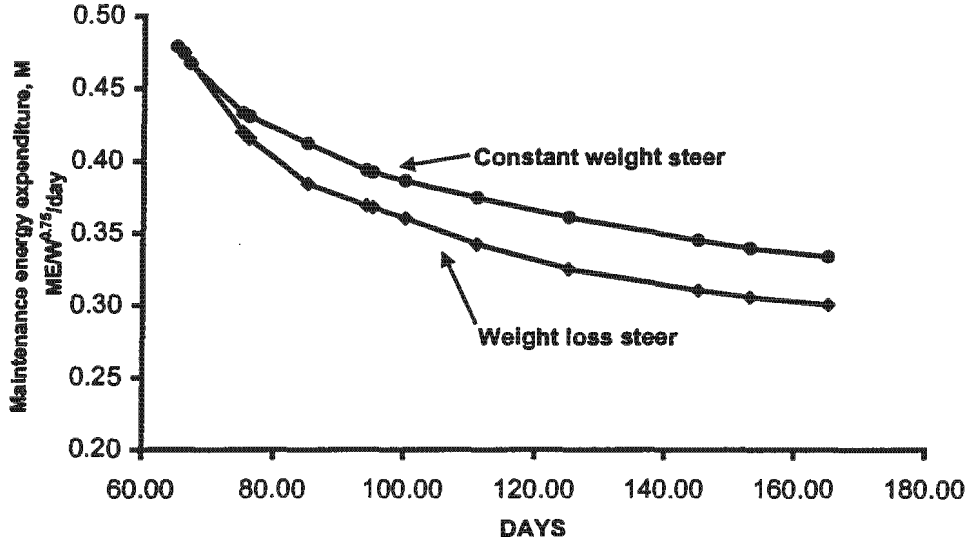


Figure 6.6: Simulation of maintenance energy expenditure ($MJ/W^{0.75}$/day) from **B** to **D.**

Weight loss path *(e)* shows the effect of prolonged nutritional deprivation, which resulted in a further 28.04 kg loss of body weight. During the first 50 days of weight loss (path *(b)*), the model simulates the adaptation to a prolonged decrease in food intake by predicting that an animal eats less per day during the second 50 days of restriction (3.0kg/day vs 3.3kg/day). The ash continued to grow but at a slower rate (0.048kg/day) than during the initial 50 days of restriction (0.076kg/day). The visceral organs also continued to decrease in weight but at a slower rate than during *(b)*. The model predicts a loss of muscle protein during the second 50 days of nutritional deprivation (Table 6.2).

The model also predicts a decrease in the maintenance energy expenditure and dry matter intake of an animal fed to maintain a constant body weight (growth path *(d)*). During this phase, the amount of food needed to maintain a constant body weight decreased from 8.5kg/day to 4.1kg/day and the maintenance energy expenditure decreased from 0.48 MJ $ME/W^{0.75}$/day to 0.33 MJ $ME/W^{0.75}$/day.

Table 6.1: Estimated feed composition and hydrolysis rates of feed components of **(a)** a diet mixture of concentrate and roughage (autumn grass) **(b)** the diet of Foot and Tulloh (1977) and **(c)** the diet of Ledger and Sayers (1977).

(a)	Feed Composition (g/kg)										Hydrolysis rate (/day)		
Feed	Fd	Fu	Sc	Si	Ps	Pd	Pu	Am	Ld	Ash	Fd	Pd	Si
Autumn grass	536	204	160	0	7	25	10	0	8	5	0.95	0.0	0.84
Concentrate	99	15	188	462	18	111	4	10	30	6.3	1.224	1.272	0.96

This diet composition was estimated relative to the diet in their experiment.

(b)	Feed Composition (g/kg)										Hydrolysis rate (/day)		
Feed	Fd	Fu	Sc	Si	Ps	Pd	Pu	Am	Ld	Ash	Fd	Pd	Si
Roughage	338	222	160	0	43	112	17	0	23	84	1.68	2.40	1.92

This diet composition was estimated relative to the diet in their experiment.

(c)	Feed Composition (g/kg)										Hydrolysis rate (/day)		
Feed	Fd	Fu	Sc	Si	Ps	Pd	Pu	Am	Ld	Ash	Fd	Pd	Si
Cottonseed Meal	263	135	47	13	57	359	8	0	50	0	1.56	3.0	1.824
Maize Grain	99	11	203	533	16	62	4	0	34	38	1.224	0.96	0.84

This diet composition was estimated relative to the diet in their experiment.

Fd = Degradable fiber, Fu = Undegradable fiber, Sc = Soluble sugars and starch, Si = Insoluble starch and sugars, Ps = Soluble protein, Pd = Degradable protein, Pu = Undegradable protein, Am = Ammonia, Ash, Ld = Fat.

Table 6.2: Simulated data from the model at points **A, B, C, D, E** and **F.**

	A	B	C	D	E	F
General						
Days	5	65	115	165	122	185
Weight (kg)	209	300	286	258	300	304
ME expenditure (MJ/day)	85.44	108.43	36.90	32.71	108.88	109.12
Total DM intake (kg)	6.70	8.49	3.30	3.00	8.50	8.55
Body composition:						
Ash (kg)	6.5	9.8	13.7	16.0	14.2	17.0
Total protein weight (kg)	48.9	69.2	65.8	56.9	68.2	66.4
Liver protein weight (kg)	2.8	4.1	1.1	0.6	1.9	2.6
Small intestine protein weight (kg)	3.7	5.3	1.7	1.1	2.4	2.9
Muscle protein weight (kg)	25.9	39.5	46.8	45.2	47.7	47.6
Lipid (kg)	25.1	36.6	21.1	15.2	24.6	25.9
Energy:						
Energy Retention (MJ)	18.18	12.89	-11.53	-6.15	31.91	30.99
Maintenance energy expenditure (MJ ME/W^0.75/day)	0.481	0.479	0.337	0.300	0.372	0.399

6.2.3 Diet composition

The response of the model animal fed ad libitum while varying the proportions of roughage and concentrate in the diet was examined and is shown in Figure 6.7. This was done because changing the proportion of roughage and concentrate in the diet affects the proportion of acetic and propionic acid absorbed from the rumen. The simulated body weight and composition at 300kg empty body weight for each diet is given in Table 6.3. For an animal fed a high roughage diet, intake is limited by the rate of digestion and the capacity of the rumen and a greater weight gain is expected on a diet with more concentrate (Figure 6.7).

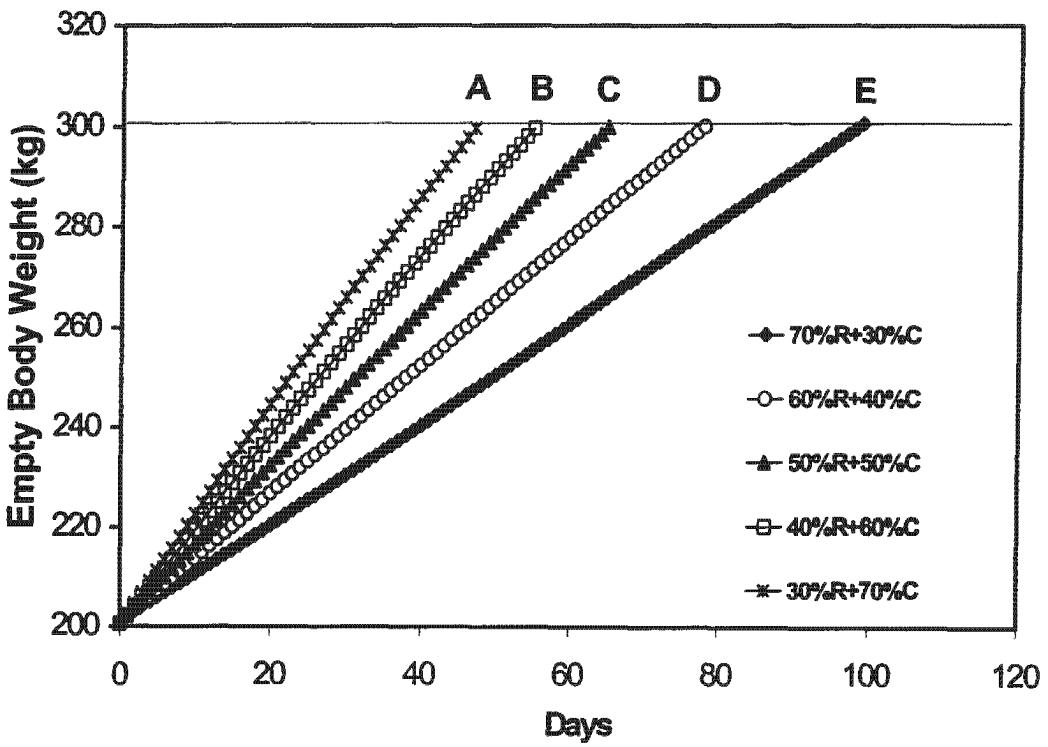


Figure 6.7: Model predicted empty body weight while varying the proportion of roughage and concentrate in the diet.

Table 6.3: Model predicted body composition at 300kg empty body weight for an animal eating five different proportions of a mixed roughage and concentrate diet.

	A	B	C	D	E
Days	48	55	65	78	99
Weight (kg)	300	300	300	300	301
Maintenance energy expenditure (MJ/day)	129.89	118.18	108.43	99.09	89.63
Total DM intake (kg/day)	9.39	8.87	8.49	8.13	7.80
Body composition:					
Ash (kg)	8.87	9.18	9.84	10.76	12.35
Total protein (kg)	70.25	69.12	69.2	69.05	68.33
Liver protein weight (kg)	5.11	4.54	4.06	3.58	3.04
Small intestine protein weight (kg)	6.47	5.81	5.26	4.71	4.09
Muscle protein weight (kg)	37.26	37.85	39.50	41.16	42.94
Lipid (kg)	41.66	38.84	36.56	34.10	31.61
Maintenance energy expenditure (MJ ME/$W^{0.75}$/day)	0.517	0.499	0.479	0.458	0.432

6.2.4 Sensitivity Analysis

It is impractical to report tests of the sensitivity of the model to changes in all kinetic parameters on all the variables in the model. Also, model parameters and variables are interrelated and changing one parameter affects several variables which are influenced by other parameters. Ten model parameters were chosen for the sensitivity analysis: k_{Lv} (the maximum rate of liver protein synthesis), v_{Lv} (the rate of breakdown of liver protein) and v_N (the rate of gluconeogenesis from the blood amino acid pool to C_6 and C_2 pools), k_{Ms} (the maximum rate of muscle protein synthesis), v_{Ms} (the rate of breakdown of muscle protein), v_L (the rate of lipolysis), $R_{C_2,NMs}$ (the requirement of C_2 for muscle protein synthesis), $R_{C_2,NLv}$ (the requirement of C_2 for liver protein synthesis), $frLHB$ (fraction of equilibrium liver protein weight of the equilibrium body weight), $frSHB$ (fraction of equilibrium small intestine protein weight of the equilibrium body weight). The parameters were increased by 50% of their estimated values given in the text. The sensitivity analysis was done on two cases: for an animal fed ad libitum on a 50% roughage and 50% concentrate diet (Above), and for an animal fed a constant dry matter intake of 3.0 kg/day (Below). The metric used in each case is the absolute difference between the initial values of the variables of interest (at EBW = 200kg) and the values of these variables after 20 days of simulation time. Table 6.4 gives the effects of changing the ten parameters on the

maintenance energy expenditure (HBW, MJ ME/$W^{0.75}$/day), the lipid pool (Lipid, kg), and weight (Weight, kg).

Table 6.4: Effect of sensitivity analysis on Weight, HBW and Lipid.

	Weight		HBW		Lipid	
Parameters	Above	Below	Above	Below	Above	Below
No parameters changed	31.8	2.13	0.479	0.105	4.817	5.014
k_{Lv}	29.71	3.85	0.048	0.081	4.583	4.971
v_{Lv}	30.51	2.19	0.059	0.143	4.820	5.018
v_N	23.45	8.18	0.0067	0.106	4.785	4.869
$R_{C_2,NLv}$	30.89	2.59	0.0039	0.106	4.762	5.004
$R_{C_2,NMs}$	31.16	3.07	0.0042	0.119	4.779	4.992
k_{Ms}	37.05	1.91	0.0183	0.097	4.821	5.118
v_{Ms}	27.81	5.61	0.0174	0.081	4.797	4.925
$frLHB$	30.74	4.24	0.0471	0.094	4.687	4.959
$frSHB$	33.06	2.31	0.0174	0.101	4.803	5.005
v_L	26.43	5.49	0.0110	0.109	2.318	10.06

Table 6.4 shows that increasing k_{Lv} and v_{Lv} had a greater effect on the maintenance energy expenditure for animals fed the constant intake of 3kg/day than for an animal fed ad libitum. In addition, changing v_{Lv} had the greatest effect on HBW on both diets. Increasing the rate of lipolysis (v_L) had the greatest effect on the Lipid pool on the constant diet but v_{Lv} had the greatest effect on the lipid pool on the ad lib diet. Increasing k_{Ms} had the greatest effect on the weight on the ad lib diet but v_{Ms} had the greatest effect on the constant diet. Figure 6.8 shows the effect of increasing the ten parameters on the liver protein mass (kg) and blood amino acid concentration (N, kg/m^3). Changing most of the parameters had a greater effect on the liver protein mass fed below maintenance (Figure 6.8a) than above maintenance. Also, Figure 6.8b shows that the blood amino acid concentration is most sensitive to the rate of gluconeogenesis.

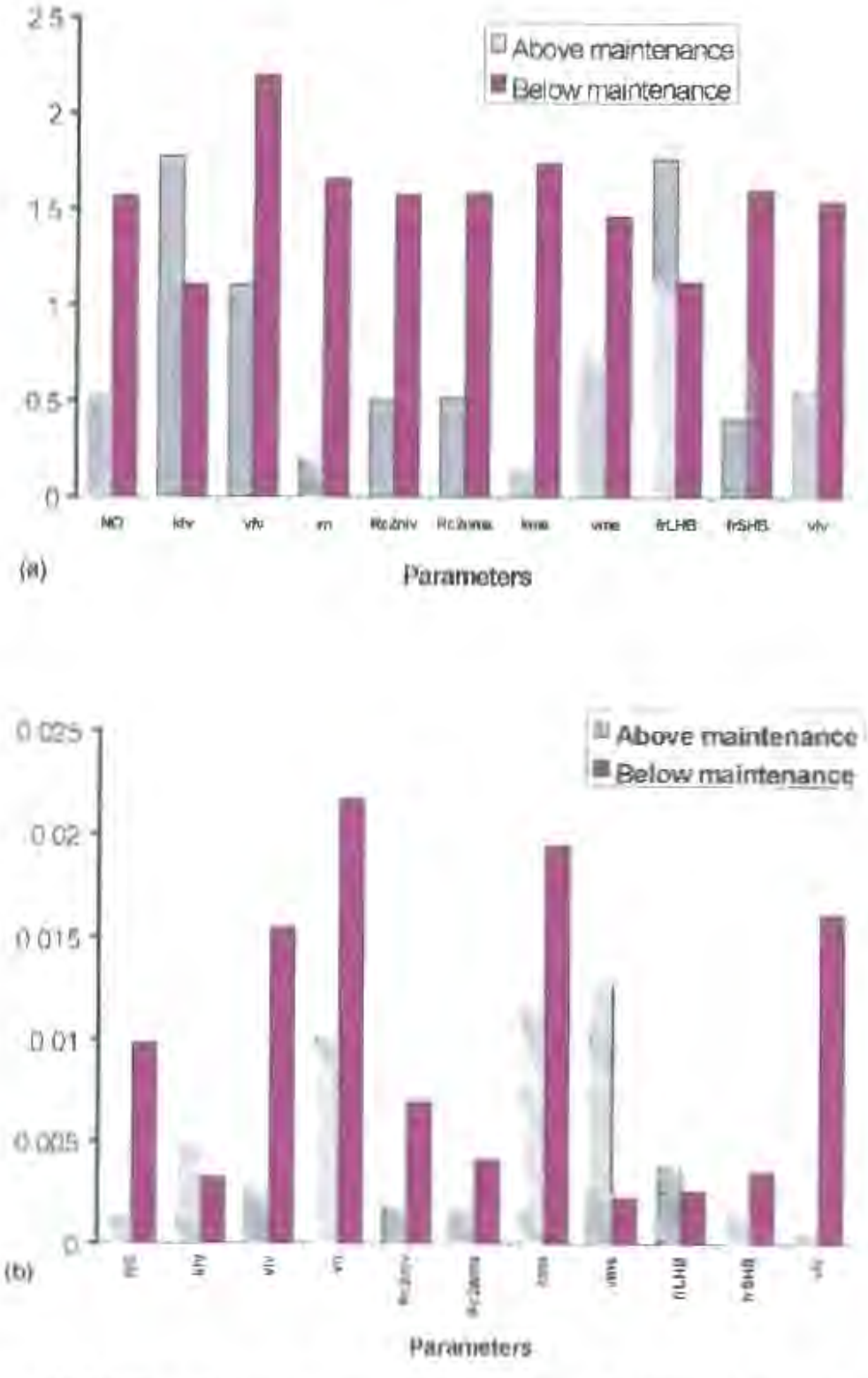


Figure 6.8: Effect of increasing ten parameters on (a) liver protein mass (kg) and (b) amino acid concentration (N, kg/m^3).

6.3 Quantitative evaluation of the RUMET model

The model was challenged with the pattern of dry matter intake estimated from the experiment of Ryan *et al.* (1993), Fool and Tulloh (1977) and Ledger and Sayers (1977). The composition of the diets were estimated from the experiments and is given in Table 6.4 and Table 6.5. In the experiment of Ryan *et al.* (1993a,b), one group of Hereford steers were offered feed ad libitum and grown continuously from 250 kg to 600 kg (control group). A similar[1] group had their diet restricted for 89 days so that they lost body weight at a rate of about 0.48 kg/day and were then fed ad libitum. In the experiment reported by Fool & Tulloh (1977) there were two groups of steers: a group whose diet was such as to maintain a constant body weight (CW group) and another group (WL/WG group) that lost 15% of its initial body weight (body weight at the beginning of the experiment) and was then fed to regain its initial weight. At the beginning of the weight-loss phase (phase 1) the ME intake was reduced so that the WL/WG group lost on average of 0.5 kg of body weight per day (for 100 days). The animals were then allowed to eat ad libitum and regained their initial body weight after 42 days. In part of the experiment of Ledger and Sayers (1977), *Bos indicus* steers were fed to maintain a constant liveweight of 275 kg. They showed that the feed intake and metabolizable energy expenditure of the steers decreased with time, therefore, the efficiency of nutrient use increased with time.

6.3.1 Challenging the model with the diet of Ryan *et al.* (1993a,b)

The dry matter intake (kg/day) of the control group and weight loss/weight gained (WL/WG) group was estimated from Ryan *et al.* (1993a) using STATISTICA (STATISTICA, 1989):

(Constant weight)

$$\text{DMIfot (kg/day)} = 9.103010 - 3.69446 \times e^{(-0.02797 \times Days)} + 0.31861 \times \sin(0.029765 \times Days - 1.09424) \quad (6.1)$$

(Weight loss)

$$\text{DMIfot (kg/day)} = 7.970215 - 2.182338 \times e^{(-0.39503 \times Days)} + 0.260885 \times \sin(0.023470 \times Days - 0.220672) \quad (6.2)$$

The composition of the diet is given in Table 6.5. Figure 6.9 below shows the model predicted weight change paths of their experiment. Table 6.6 shows the body weight and composition predicted by the model at points **A**, **B**, **B***, **C** and **C*** shown in

[1] group of cattle of the same breed, age and weight

Figure 6.9. Table 6.6 also shows the body composition and weight at the end of the experiment of the control (**Control***) and restricted (**R***) animals estimated from the experimental data of Ryan *et al.* (1993a).

The model was run to simulate an animal losing 0.44kg/day for 90 days. The model predicted a 21.71% weight loss (which was also demonstrated by Ryan *et al.*, 1993a), and the compensatory growth of an animal: increase in weight gain (Figure 6.9) and dry matter intake during the first few days of realimentation. During the 90 days of restriction, the model predicted the weight loss and reduction in feed intake (2.4 kg/day to 1.3 kg/day) reported by Ryan *et al.* (1993a).

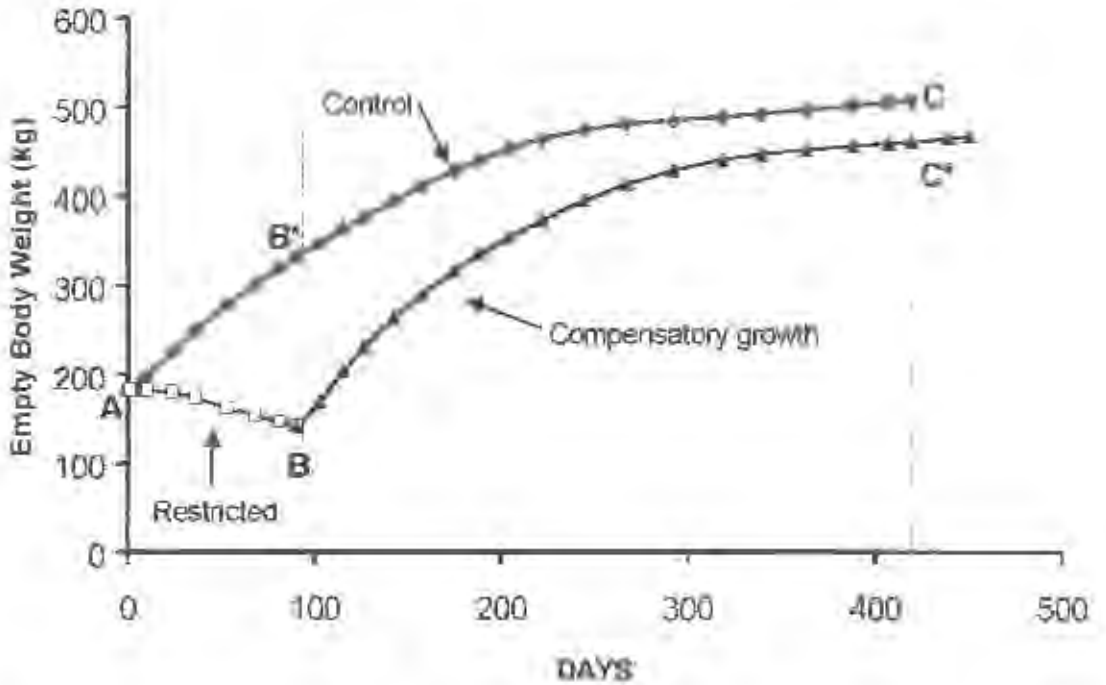


Figure 6.9: Model simulated growth paths with the diet of Ryan *et al.* (1993a).

During the simulation the rate of lipolysis (V_L) was decreased from 0.05/day to 0.01/day for a continuous growing animal and during realimentation. This was done in order to simulate the increased deposition of fat towards maturity. The model predicted the available experimental data well except for the overestimation of lipid and ash. This was probably due to the low rate of lipolysis value that was chosen. The body composition for the control and restricted animals were found to be similar, as reported by Ryan *et al.* (1993b). Figure 6.10 shows the predicted liver protein (kg) and maintenance energy expenditure during the restricted period. The model also

predicts the continuous increase of ash during the restricted period and that the muscle only begins to decrease after about 60 days of nutritional restriction (Figure 6.11).

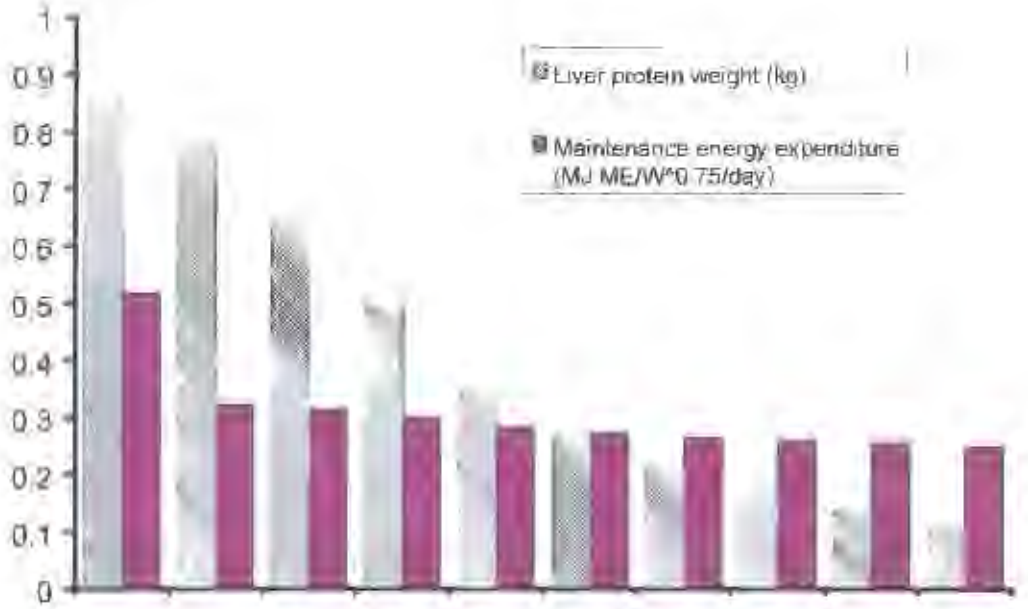


Figure 6.10: Model predicted liver protein weight (kg) and maintenance energy expenditure (MJ ME/$W^{0.75}$/day) during the 100 day restricted period for an animal fed the diet of Ryan *et al.* (1993a)

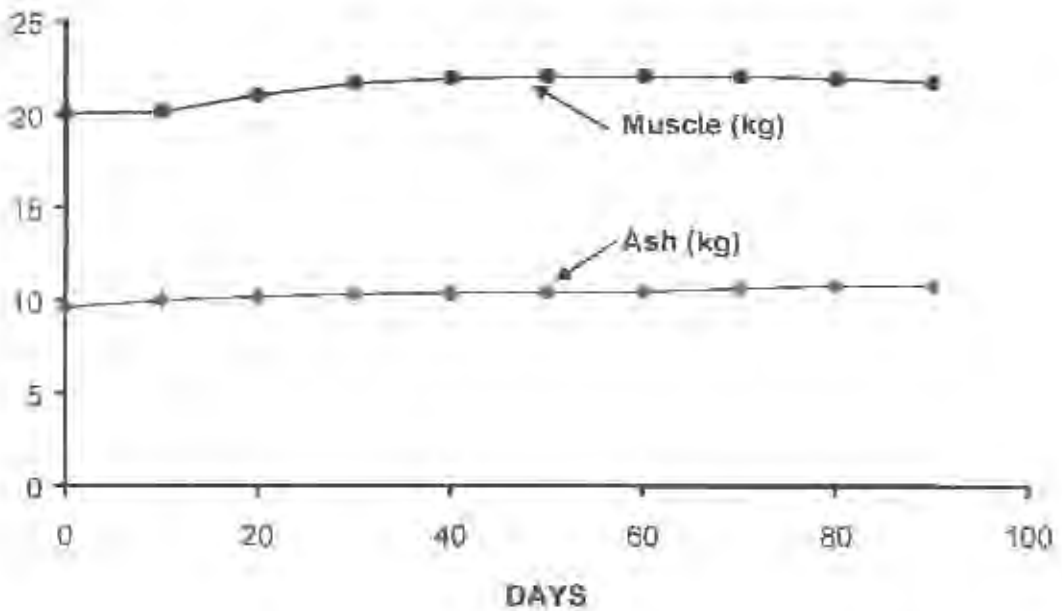


Figure 6.11: Model predicted muscle and ash weight (kg) during the 100 day restricted period for an animal fed the diet of Ryan *et al.* (1993a)

Table 6.5: Estimated food composition and hydrolysis rates for the diet of Ryan *et al.* (1993).

Feed	Feed Composition (g/kg)										Hydrolysis rate (/day)		
	Fd	Fu	Sc	Si	Ps	Pd	Pu	Am	Ld	Ash	Fd	Pd	Si
Roughage (0.49)	462	176	160	0.0	2	81	32	0	8	50	0.950	0.0	0.84
Concentrate (0.5)	99	15	188	462	18	111	4	10	30	63	1.224	1.272	0.96
Urea (0.01)	0	0	0	0	0	0	0	549	0	0	0.0	0.0	0.0

Fd = Degradable fiber, Fu = Undegradable fiber, Sc = Soluble sugars and starch, Si = Insoluble starch and sugars, Ps = Soluble protein, Pd = Degradable protein, Pu = Undegradable protein, Am = Ammonia, Ash, Ld = Fat.

Table 6.6: Predicted body weight and composition at points **A, B, B*, C and C*** of the model and estimated body composition and weight at the end of the experiment of Ryan *et al.* (1993) of the control (**Control***) and restricted (**R***) animals.

	A	**B**	**B***	**C**	**C***	**Control***	**R***
Days	1	90	90	420	420	420	420
Weight (kg)	181	142	332	573	537	584	602
Roughage DM intake (g/day)	5668	1300	8490	7879	9374	8700	8300
Body composition:							
Ash (kg)	9.8	9.8	15.5	21.7	22.1	17.1	17.2
Total protein weight (kg)	34.9	27.8	68.9	75.9	73.8	75.4	77.2
Liver protein weight (kg)	0.7	0.1	4.1	0.9	0.9	------	------
Small intestine protein weight (kg)	0.9	0.3	5.4	0.9	1.0	------	------
Muscle protein weight (kg)	72.5	21.7	38.5	46.9	52.3	------	------
Lipid (kg)	20.6	5.7	37.5	159.9	203.2	165.5	176.4
Energy:							
Energy Retention (MJ)	22.29	-5.73	9.73	13.39	6.60	-------	------
Maintenance energy expenditure (MJ ME/W^0.75/day)	0.317	0.249	0.449	0.325	0.230	-------	------

6.3.2 Challenging the model with the data of Foot and Tulloh (1977)

The model was also challenged with the dietary regimes imposed by Foot and Tulloh (1977). The estimated diet composition and hydrolysis rates are shown in Table 6.1. The scheme of the experiment is shown in Figure 6.12 and the simulated empty body weight is shown in Figure 6.13. The body weight and composition of the steers were recorded for the weight-loss and rehabilitation phase, and for the constant weight (CW) phase (Table 6.7). The model was run for a 82 kg animal to point A, where the body composition values were set equal to the body composition values of the animals in their experiment. The dry matter intake (kg/day) of the control group and weight loss group was given in their paper as:

Control group:

$$\text{DMItot (kg/day)} = [82.4 - 0.22 \times Days] \times Weight^{0.75} \quad (6.3)$$

Restricted group:

$$\text{DMItot(kg/day)} = [81.6 - 0.86 \times Days + 0.03 \times Days^2] \times Weight^{0.75} \quad (6.4)$$

The model predicted a weight loss of 15% in 100 days and the 42 days of realimentation as demonstrated by Foot and Tulloh (1977). Table 6.7 also shows the body weight and composition predicted by the model and given in the experiment.

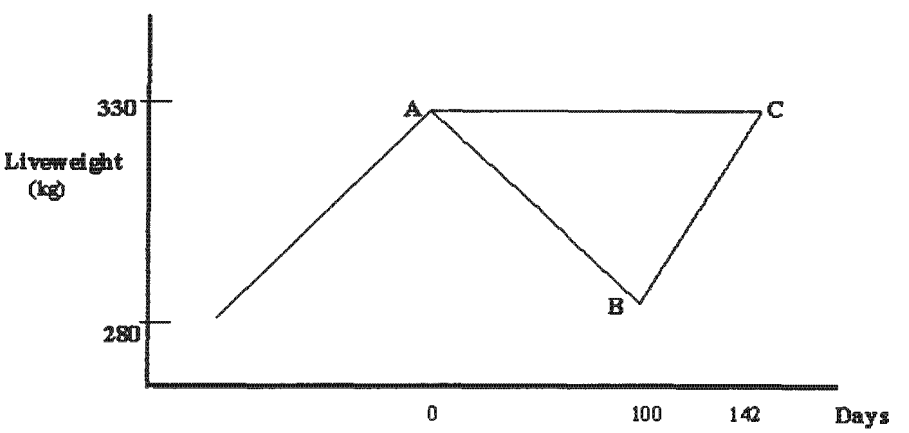


Figure 6.12: Plan of the experiment of Foot and Tulloh (1977).

Table 6.7: Model predicted body composition and weight of a steer during undernutrition and at a constant weight at points A, B*, B, C and C* of Figure 6.13 for the diet of Foot and Tulloh (1977).

	A	B*	B	C	C*
Days	10	100	100	144	142
Weight (kg)	279	281	228	278	272
Roughage DM intake (g/day)	7920	4147	1503	6745	3426
Ash (kg)	10.9	17.2	16.3	16.8	17.9
Total protein weight (kg)	66.6	62.4	50.1	61.2	59.8
Liver protein weight (kg)	2.4	1.3	0.2	1.9	0.8
Small intestine protein weight (kg)	3.4	1.9	0.5	2.5	1.4
Muscle protein weight (kg)	41.7	43.6	41.7	44.6	45.4
Lipid (kg)	22.9	15.8	7.5	18.5	13.3
Energy Retention (MJ)	- 5.93	- 3.44	- 12.20	13.08	- 4.18
Maintenance energy expenditure (MJ ME/W^0.75/day)	0.417	0.344	0.267	0.378	0.316

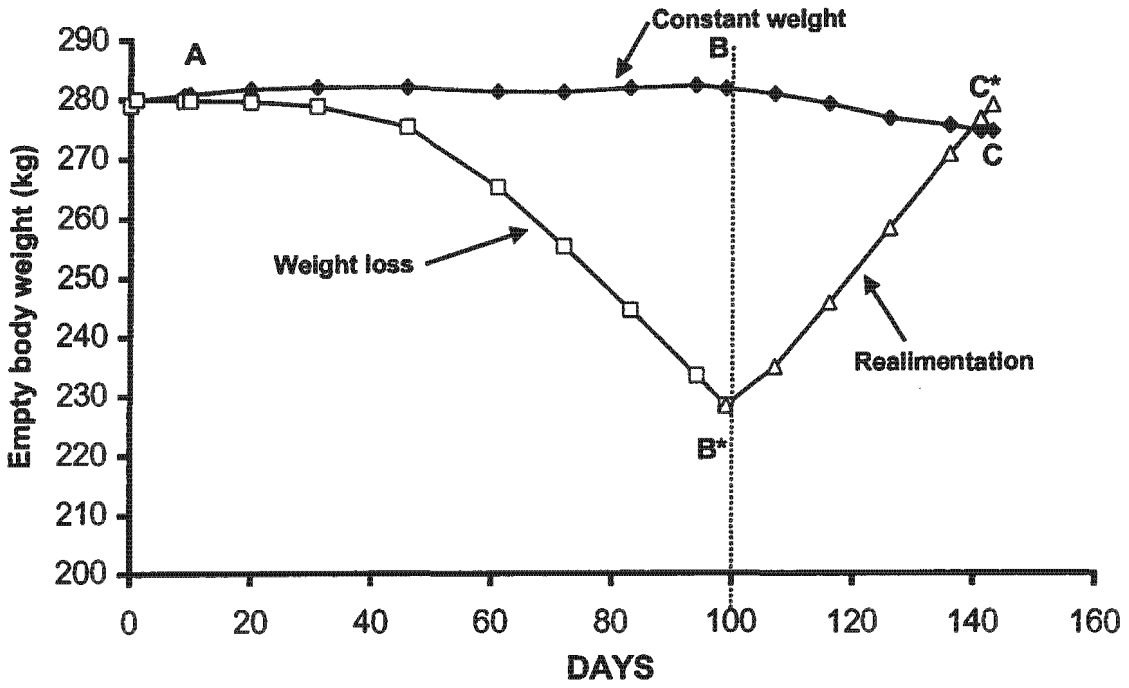


Figure 6.13: Simulated empty body weight (kg) of the model during the weight-loss phase and during realimentation using the dietary regime and diet of Foot and Tulloh (1977) as model input.

6.3.3 Challenging the model with the data of Ledger and Sayers (1977)

Figure 6.14 shows the simulated empty body weight predicted by the models. The dry matter intake (g/week) of the constant weight animal is given in their paper as:

$$\text{DMItot (g/week)} = [0.8444 + 1.70830 \times Weeks^{0.796}] \times Weight \times 10. \quad (6.5)$$

The model was run for 12 weeks and the maintenance energy expenditure decreased by 22.3%, which was close to the value (29.3) estimated by Ledger and Sayers (1977). Figure 6.15 shows that the muscle protein weight (kg) and ash weight (kg) increased and the maintenance energy expenditure decreased during the 12 week simulated period.

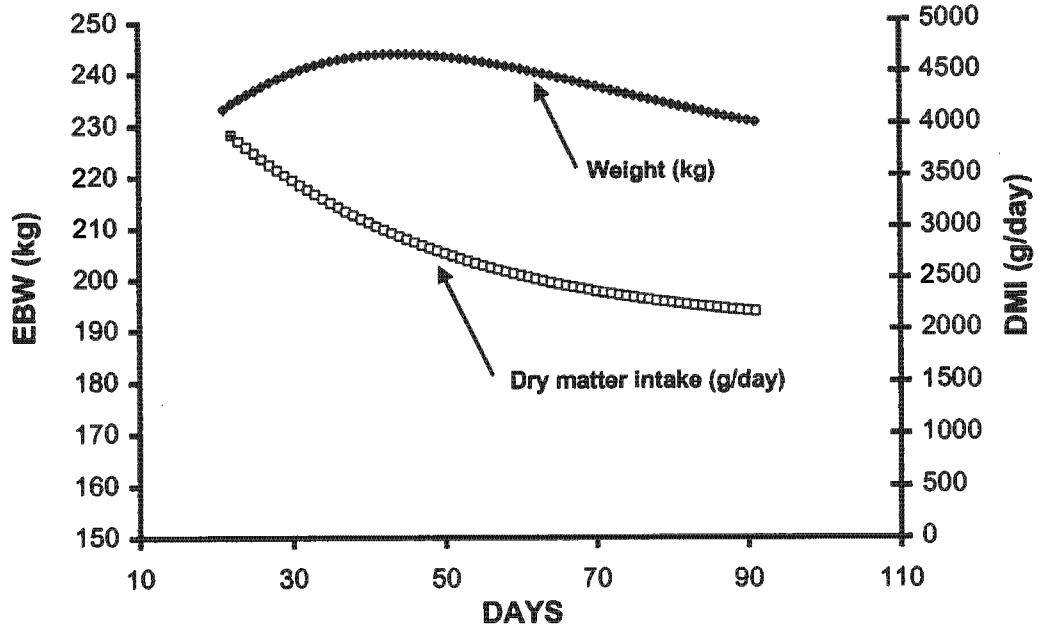


Figure 6.14: Simulated empty body weight (kg) and dry matter intake for animals subjected to the experimental diet of Ledger and Sayers (1977).

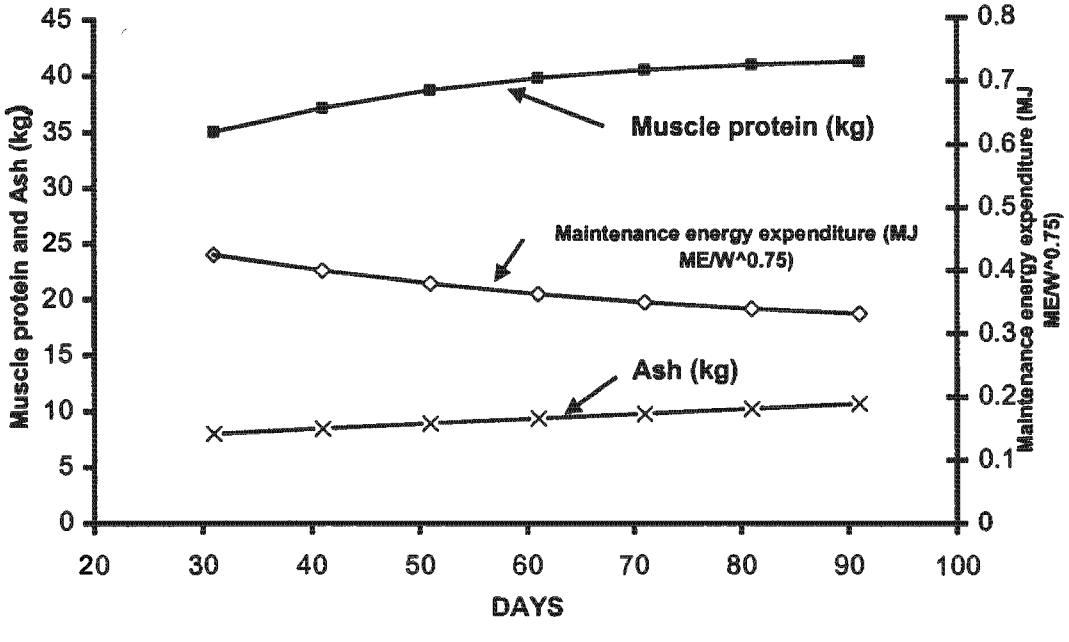


Figure 6.15: Simulated muscle protein weight (kg), ash weight (kg) and maintenance energy expenditure for animals subjected to the experimental diet of Ledger and Sayers (1977).

6.4 Discussion and Conclusions

The only conclusions we can derive from the qualitative assessment is whether model behaviour corresponds to the behaviour reported in the literature. Once the correct

model behaviour is confirmed is then possible to continue the evaluation process by comparing the model behaviour with experimental data and with the behaviour of other models (see section 6.5). Decreasing the plane of nutrition had several effects on the body composition and basal energy expenditure of the model animal. Firstly, the visceral organs (liver and small intestine) decreased immediately (within one day) to the decrease in feed intake and lost a substantial amount of its weight. A substantial decrease in liver and small intestine weight with a decrease in the plane of nutrition was demonstrated in cattle (Foot and Tulloh, 1977) and in sheep (Graham and Searle, 1976). The immediate response in liver weight was shown by Burrin *et al.* (1988). They showed that the liver was most affected by the decrease in feed intake and lost a quarter of its weight within 72 hours and that most of this loss occurred during the first 24 hours.

Predicted basal metabolism also decreased during the restricted phase. High correlations between visceral organ weights, food intake and fasting metabolism were demonstrated by Koong *et al.* (1982), Ferrell (1984) and Pekas (1991). There is therefore no question at this point about the correlations among feed intake, organ weights and fasting heat production, and apparent maintenance requirements. The question has now become what portion of the changes in fasting heat production and apparent maintenance requirements are attributable to relative changes in organ weight *per se*, and what portions are attributable to changes in tissue functions that vary in a collinear fashion with feed intake, physiological and hormonal state, and relative organ size (Baldwin, 1995). One of the factors considered to have a possible influence on compensatory liveweight gains is an increased appetite resulting in an increased food intake. The enhanced food intake following undernutrition is only possible if the size of the digestive system relative to liveweight is larger in the restricted animals than those fed ad libitum continuously. The intake of herbage by grazing animals is also a function of bite size and the time the animal spends grazing. Ferrer *et al.* (1995) suggested that the increased intake of herbage in relation to body size in animals previously retarded in growth in winter periods are related not to bite size but to the willingness to graze for longer periods of time and to increase the rate of grazing. Another factor contributing to the large increase in body weight of restricted animals at the beginning of the grazing period is the fact that the restricted animal will be smaller in body size and will have smaller viscera. As a consequence of the enhanced food intake after the restricted phase, there will be more food available for growth purposes.

The ash pool continued to increase during the restricted period. This was demonstrated by numerous investigators (Fowler, 1960; Foot and Tulloh, 1977) which have shown that the head, tail and feet continue to gain during nutritional deprivation. Lipid stores were mobilized during nutritional deprivation. Dawson *et al.* (1998) showed that growth hormone increases the plasma palmitate flux during an experiment when steers were fed at 0.8 × maintenance. The model, however, does not predict the quantity of fat correctly unless the rate of lipolysis is adjusted. The model does predict the breakdown of fat during the feed restriction phase. It was thought that this effect was due to the catabolic hormone effect.

6.5 Comparing four simulation models

As mentioned in chapter 2, there has been a shift from empirical models to more dynamic mechanistic models, such as the models of Gill *et al.* (1984) and France *et al.* (1987), to predict the body composition and growth of animals. Arnold and Bennet (1991) compared three empirical models (Notter, 1977; Loewer *et al.*, 1986 and Sanders and Cartwright, 1979) and a mechanistic model (Oltjen *et al.*, 1986) and showed that empirical models can result in a 25% error in estimates of body composition and suggested that empirical models not be used to predict body growth and composition outside the range of diet and animal genotype data on which they are based. They also suggested that more detailed mechanistic models be developed but that the level of aggregation be carefully considered. Most mathematical models of growing cattle are designed to simulate the effects of food intake and diet composition on the rate of growth and body composition in continuously growing animals. They do not adequately predict the changes in the weights of the viscera in the chemical composition of the growing bovine during and following a period of weight loss as a result of undernutrition (Witten and Richardson, 1998).

The aim of this section is to compare four mechanistic mathematical models, three of which are based on different assumptions of growth, when simulating different feeding patterns and comparing their responses. The models based on different growth assumptions are a modified model of Oltjen *et al.* (1986), the intermediary metabolism model of France *et al.* (1987), and the model of Keele *et al.* (1991). The fourth model is the linked rumen model and intermediary metabolism model, called the RUMET model, described in section 6.2. For simplicity, the original model of Oltjen *et al.* (1986) will be called the OLTJEN model and the modified model of Oltjen *et al.* (1986) will be called the OLTBLAX model, the model of France *et al.* (1987) the FRANCE model, and the model of Keele *et al.* (1991) the KEELE model.

The FRANCE and KEELE models were linked with the modified rumen model described in chapter 3. The models were compared using the same logic and parameter values as presented by the authors in their published papers. The models were coded in Turbo Pascal and run using DRIVER (Hahn and Furniss, 1989). The predictions of the four models will be compared and challenged with the data presented by Foot and Tulloh (1977) and Ledger and Sayers (1977).

6.5.1 Description of model equations

OLTJEN model

Oltjen *et al.* (1986) based their mechanistic model on the premises of Baldwin and Black (1979), which showed that the concepts accommodating DNA accumulation (hyperplastic growth) and protein per unit of DNA (hypertrophic growth) is required to simulate tissue growth. They integrated their model, which predicts growth and net protein synthesis, into the Lofgreen and Garrett (1968) system to estimate gain of fat and lean tissue. The model equations are shown in Table 6.8 with the coefficients for a beef steer of mature body weight of 650kg. The terms $NUT1$ and $NUT2$ are nutritional constants set to 1 for normal *ad libitum* intake. $DNAmx$ is the content of DNA at maturity and was calculated indirectly as 385g by Baldwin and Black (1979) for a 750kg mature weight animal. Assuming a linear relation between mature DNA content and mature weight, the mature DNA content of a 650 kg animal was estimated as 334g. Parameters were estimated using a non-linear least squares fit of observed body gains in 53 groups of feedlot steers (Oltjen *et al.*, 1986). The rate constants $K1 = K2 = 0.0462$ and $K3 = 0.143$. Taylor's (1980) size-scaling rules and the assumption of equal composition at equal degrees of maturity were used to adjust for cattle of different mature weights. For animals of different mature size (A'), the rate constants are adjusted by a scaling factor (equation 6.10). Hence, $K1 = 0.00429(\frac{A'}{650})^{-0.27}$ but $K2$ and $K3$ was not changed. The effects of energy intake on growth were added using the $NUT1$ and $NUT2$ terms. A ratio P was defined as in equation 6.11 where $MEINORM$ is the pattern of ME intake that supports normal growth of a reference steer (equation 6.12) and MEI is the metabolizable energy intake per day (MJ/day). Rates of DNA accretion and $PROT$ synthesis are adjusted by the NUT terms (equations 6.13 and 6.14). Daily empty body fat gain ($DFAT$) is calculated as the net energy available after daily feed intake (FI, kg/day) is used for maintenance ($MAINT$) and protein gain ($DPROT$, kg/day) as in equations 6.15 and 6.16, respectively, where $a_m = 0.359$ and the energy content (MJ/day) of the protein and fat gains are 23.18 and 39.27, respectively

(Garrett and Hinman, 1969). Empty body weight is the sum of fat and fat-free body masses, where fat-free body mass is $\frac{PROT}{0.2201}$ (Garrett and Hinman, 1969).

Table 6.8: Model equations of Oltjen *et al.* (1986) for a 650 kg steer.

$\frac{dDNA}{dt} = K1 \times (DNAMX - DNA) \times NUT1$	6.6
$\frac{dPROT}{dt} = SYNTHESIS - DEGRADATION$	6.7
$SYNTHESIS = K2 \times DNA^{0.73} \times NUT2$	6.8
$DEGRADATION = K3 \times PROT^{0.73}$	6.9
$r' = r(\frac{A'}{A})^{0.73}$	6.10
$P = \frac{MEI}{MEINORM}$	6.11
$MEINORM = (1.83 - 1.094 \times \frac{EBW}{A'}) EBW^{0.75}$	6.12
$NUT1 = -0.7 + 1.7P$	6.13
$NUT2 = 0.83 + \frac{0.2P}{0.15+P}$	6.14
$MAINT = a_m \times EBW^{0.75}(\frac{A'}{650}) - 0.02$	6.15
$DFAT = (FI - \frac{MAINT}{NE_m}) \times NE_g - 23.18 \times \frac{DPROT}{39.27}$	6.16

FRANCE model

Their model is based on the enzyme kinetic principles developed by Gill *et al.* (1984). There are three basic principles: firstly, the kinetics apply to the entire system, transactions are independent, secondly, the rate of a transaction is a function of the state of the animal and the rates are determined by substrate concentrations, thirdly, model accuracy is a function of the representation of the biochemical processes and the estimation/assumption of parameters. The prediction accuracy of the model of Oltjen *et al.* (1986) is a function of the modification of the equations based on the premises of Baldwin and Black (1979). The objective of the model was to simulate over a period of many weeks, the effects of varying nutrient inputs on carcass composition and they proposed a dynamic model of cattle growth based on carbon and nitrogen metabolism. Inputs of absorbed rumen volatile fatty acids, stearate and amino acid are stoichiometrically converted to their equivalent two or six carbon metabolites in three blood metabolite pools: C_2 (acetyl-CoA equivalents), C_6 (glucose equivalents) and N (amino acids concentration). All fluxes between body composition and metabolite pools are based on nine principal transactions, six of them catabolic and three biosynthetic.

Stoichiometry is used to define the yield and requirement factors for the transfer of carbon, nitrogen and other elements between substrates and products, accounting for the energy requirements or yields within the reaction. An additional modification in

the FRANCE model is that the V_{max} of synthesis reactions is a variable. The model consists of first order ordinary differential equations which describe how the state variables change with time. The model equations will not be given here because they have been used in the development of the model described in chapter 5.

KEELE model

Keele *et al.* (1991) described how the average empty body composition of cattle belonging to a breed or cross-breed group will respond to changes in nutrition. They base their model on a blend of the assumptions of France *et al.* (1987) and Oltjen *et al.* (1986): firstly, there is a greater proportion of fat in the empty body gain than in the empty body for cattle growing at intermediate or higher growth rates, secondly, differences in empty body composition caused by plane of nutrition (that are not associated with empty body weight (EBW)) may be predicted from the rate of EBW gain, thirdly, the full effects of a change in nutrition on empty body composition are not exerted immediately nor are they permanent and, fourthly, if the EBW of an animal is not changing, its empty body composition approaches an equilibrium value. The objective of their study was to develop a dynamic model that uses $\frac{dEBW}{dt}$ to predict differences in empty body fat caused by plane of nutrition. The equations of the model are given below and parameter values given in Table 6.9.

$$\frac{dEBW}{dt} = \frac{\text{Hb}\times\left[\text{Bke}\times\frac{1}{(1-e^{-p\times g})}\right]}{\text{Evgbar}} \qquad \text{(Blaxter and Boyne, 1978)} \qquad 6.17$$

$$\frac{dFFM}{dt} = k(t)*(\frac{FFM}{EBW})*\frac{dEBW}{dt} + \epsilon(t) \qquad 6.18$$

$$\text{where } k(t) = kmax*(1 - (\frac{FFM}{FFMmat})^{\beta(t)}) \qquad 6.19$$

$$\text{and } \beta(t) = \theta*e^{-LAG} \qquad 6.20$$

$$\frac{dLAG}{dt} = \alpha*(\frac{dEBW}{dt} - LAG) \qquad 6.21$$

$k(t)$ is the relative priority for nutrient use between lean and adipose tissue and depends on the maturity of the animal which is modelled by $\frac{FFM}{FFMmat}$. $k(t)$ increases with the increasing priority for nutrient use by lean tissue. It is assumed that $k(t)$ is at its maximum value at conception ($kmax$) and that it decreases to its minimum at some point near maturity ($kmin$). $\beta(t)$ affects the degree of concavity of the $k(t)$ graph but the effects of empty body composition cannot simply be accounted for by making $k(t)$ dependent on maturity and hence the chosen function for β, which accounts for the plane of nutrition on FFM (free-fat mass). A function for $\epsilon(t)$ was chosen based on a corollary of assumption 4 and described in Keele *et al.* (1991):

If $\frac{dEBW}{dt} = 0$ then the rate of FFM $= 0$ iff $FFM = FFMeq$, thus $\epsilon(t) = \left[FFMeq - FFM\right]$. Also, $FFMeq = EBW \times (1 - \frac{PFATeq}{100})$. Keele *et al.* (1991) noted that their model was inappropriate when growth rate is limited by protein intake because this would violate their second assumption. Modifications to this model has been made with F. D. Richardson (personal communications) and presented in Witten and Richardson (2001).

Table 6.9: Estimated parameter values for the KEELE model (Keele *et al.*, 1991)

Parameter	Value
α	0.001
k_{max}	0.95
k_{min}	0.2
FFM_{mat}	480
θ	8.0
λ	0.03

6.5.2 Comparing the models

The models were run to simulate the body weight and composition of a steer fed ad libitum on a 50% roughage and 50% concentrate diet (Table 6.1a). The mature weight in the KEELE, OLTJEN and OLTBLAX model was set at 650kg. The mature weight of the FRANCE model is a function of the maximum weight of protein (200kg for a 1000kg animal), and was not modified. Because the protein pool in the FRANCE model was divided into four in the RUMET model, the maximum weights of the individual pools were estimated from Gibb *et al.* (1992) for an animal of 640kg. When the model of Oltjen *et al.* (1986) was simulated on a diet of 3.0 kg/day, the animal model gained weight for the first 90 days and then lost about 14% of its maximum weight during the next 510 days. The model did not yield acceptable estimates of fat gain (see Figure 6.16). The reason for this is because fat accretion is computed after the energy requirements for maintenance and protein gain are satisfied (Baldwin, 1995). Therefore, any errors in the estimates of maintenance or protein gain results in biased fat gain predictions. To overcome this problem, the Blaxter and Boyne (1978) equation was used to calculate the daily energy retention and used to calculate the rate of change of fat as $\frac{dFAT}{dt} = \frac{\frac{dER}{dt} - 23.6 \times \frac{dPROTEIN}{dt}}{39.3}$ (see Appendix 6.1 for model equations of the OLTBLAX model). The model using the Blaxter and Boyne (1978) equation predicted body lipid better than the original Oltjen *et al.* (1986) model (Table 6.10).

Table 6.10: Simulated results of the original Oltjen *et al.* (1986) (OLTJEN) model and the modified Oltjen *et al.* (1986) (OLTBLAX) model.

OLTJEN MODEL

	FEED = 1 kg/day			FEED = 2 kg/day			FEED = 3 kg/day			FEED = 4 kg/day			FEED = 5 kg/day		
Days	EBW (kg)	FAT (kg)	Protein (kg)	EBW (kg)	FAT (kg)	Protein (kg)	Protein (kg)	EBW (kg)	FAT (kg)	EBW (kg)	FAT (kg)	Protein (kg)	EBW (kg)	FAT (kg)	Protein (kg)
0	196	27.5	34.2	196	27.5	34.2	34.2	196	27.5	196	27.5	34.2	196	27.5	34.2
150	111	-30.3	35.0	208	10.8	41.0	27.8	162	-10.3	243	24.7	46.1	274	36.0	36.0
300	39	-36.6	26.8	198	11.7	38.4	12.4	120	-16.3	258	30.8	48.1	310	48.0	56.2
450	32	-34.5	21.9	190	13.7	36.2	10.4	101	-14.8	269	35.9	49.4	336	57.7	60.2
600	32	-32.7	19.5	186	16.4	34.5	9.9	94	-11.7	277	40.1	50.3	357	65.3	63.2

OLTBLAX MODEL

	FEED = 1 kg/day			FEED = 2 kg/day			FEED = 3 kg/day			FEED = 4 kg/day			FEED = 5 kg/day		
Days	EBW (kg)	FAT (kg)	Protein (kg)	EBW (kg)	FAT (kg)	Protein (kg)	EBW (kg)	FAT (kg)	Protein (kg)	EBW (kg)	FAT (kg)	Protein (kg)	EBW (kg)	FAT (kg)	Protein (kg)
0	196	27.5	34.2	196	27.5	34.2	196	27.5	34.2	196	27.5	34.2	196	27.5	34.2
150	133	-5.6	27.4	178	8.6	34.6	217	21.2	40.0	250	32.6	45.8	280	42.8	50.4
300	50	-8.0	8.2	133	5.2	24.8	204	22.1	37.5	265	40.1	47.5	318	58.2	55.8
450	33	-4.1	3.3	107	7.6	18.2	195	24.5	34.9	275	46.1	48.6	346	70.2	62.5
600	33	-1.6	2.7	97	12.0	14.6	190	27.9	32.7	283	51.0	49.3	366	79.5	79.5

However, both models were unable to simulate body composition correctly when the model was used to simulate lower feed intakes (1-5 kg/day) (Table 6.10). For this reason, the original Oltjen *et al.* (1986) model and the modified Oltjen *et al.* (1986) model are of little use for our modelling objective and, therefore, these models were not used in further analysis.

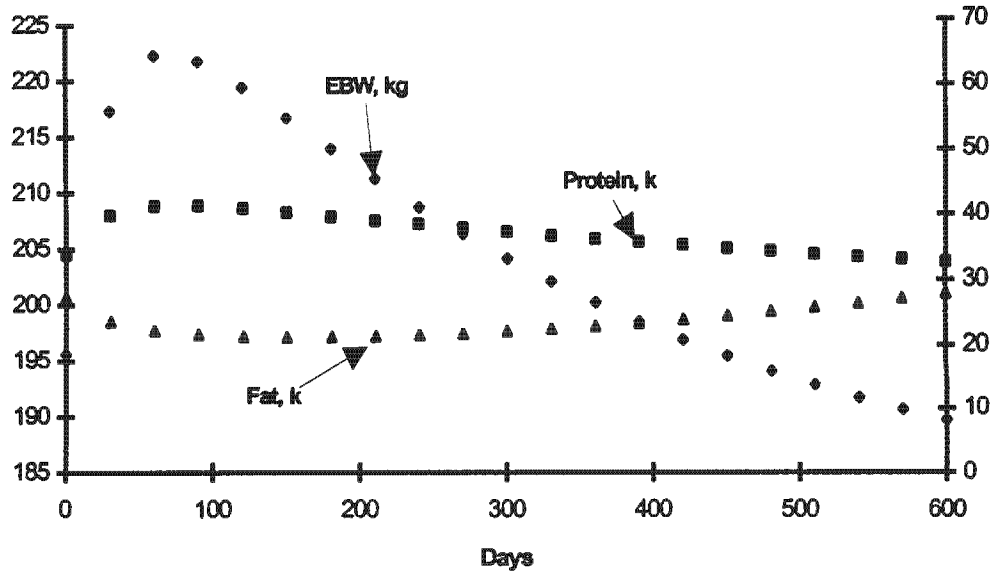
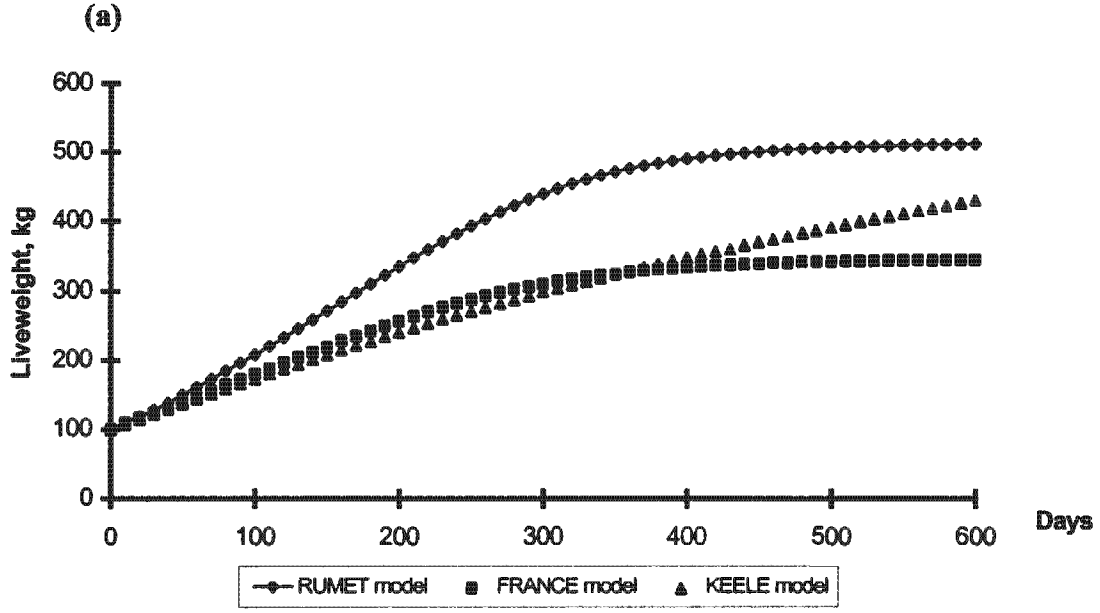
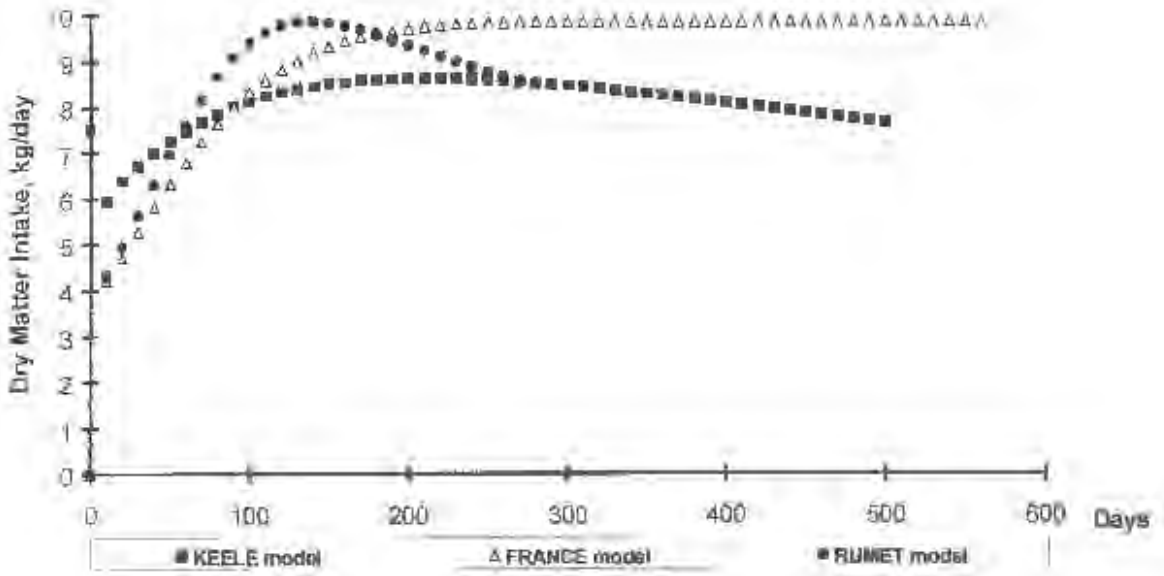


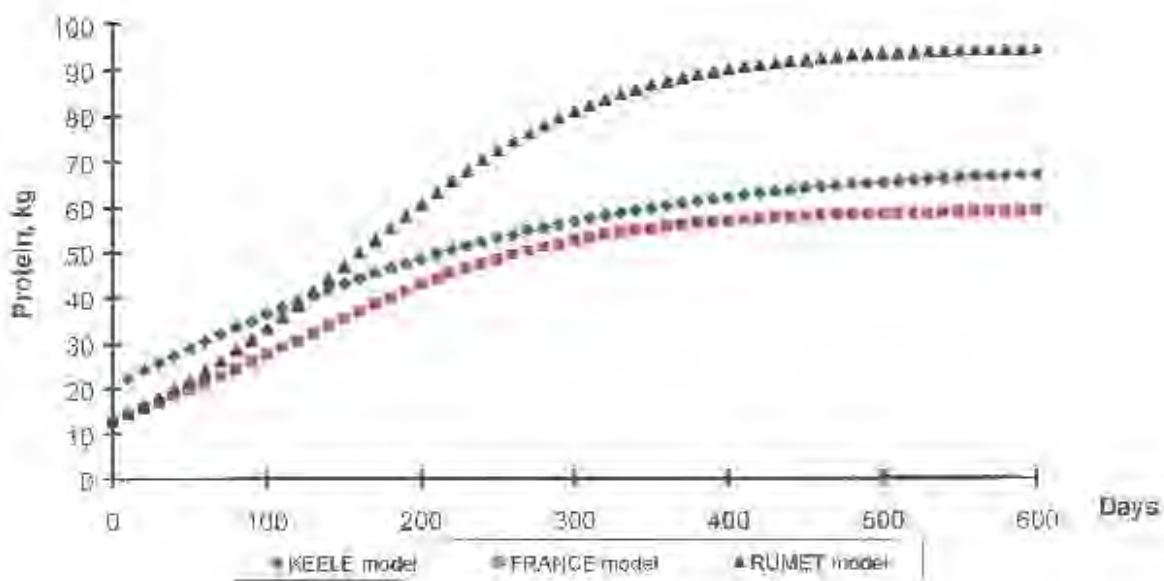
Figure 6.16: Simulated EBW (kg) and fat (kg) gain at a feed intake of 3.0 kg/day for the original model of Oltjen *et al.* (1986).



(b)


Dry Matter Intake, kg/day
0
1
2
3
4
5
6
7
8
9
10
0
100
200
300
400
500
600
Days
KEELE model
FRANCE model
RUMET model

(c)


Protein, kg
0
10
20
30
40
50
60
70
80
90
100
0
100
200
300
400
500
600
Days
KEELE model
FRANCE model
RUMET model

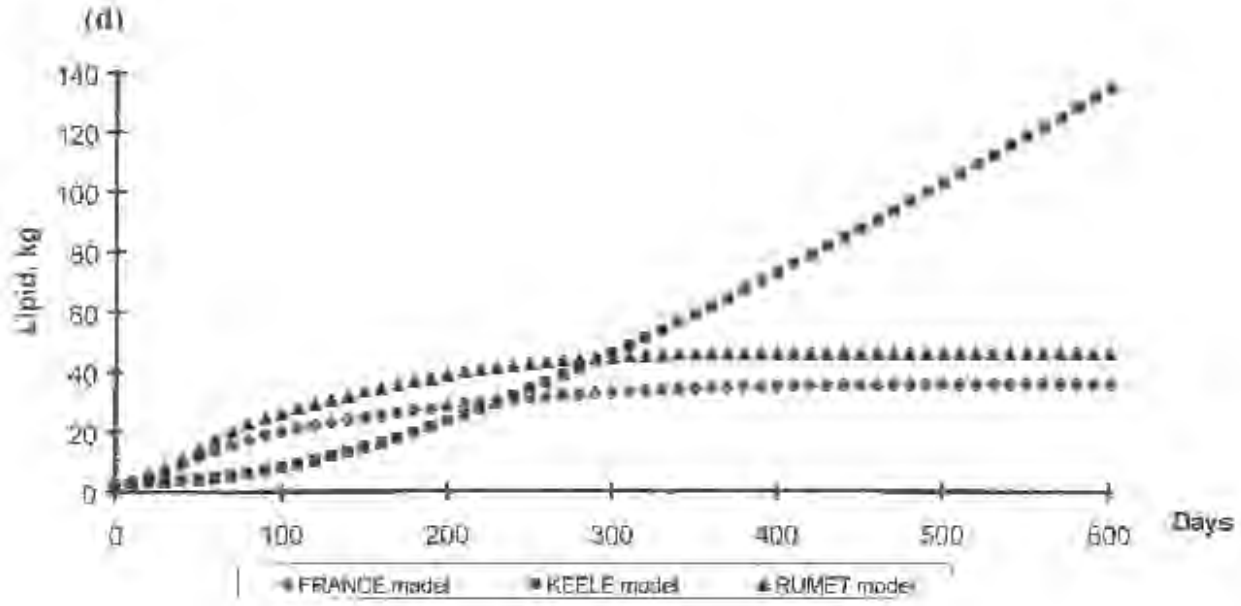
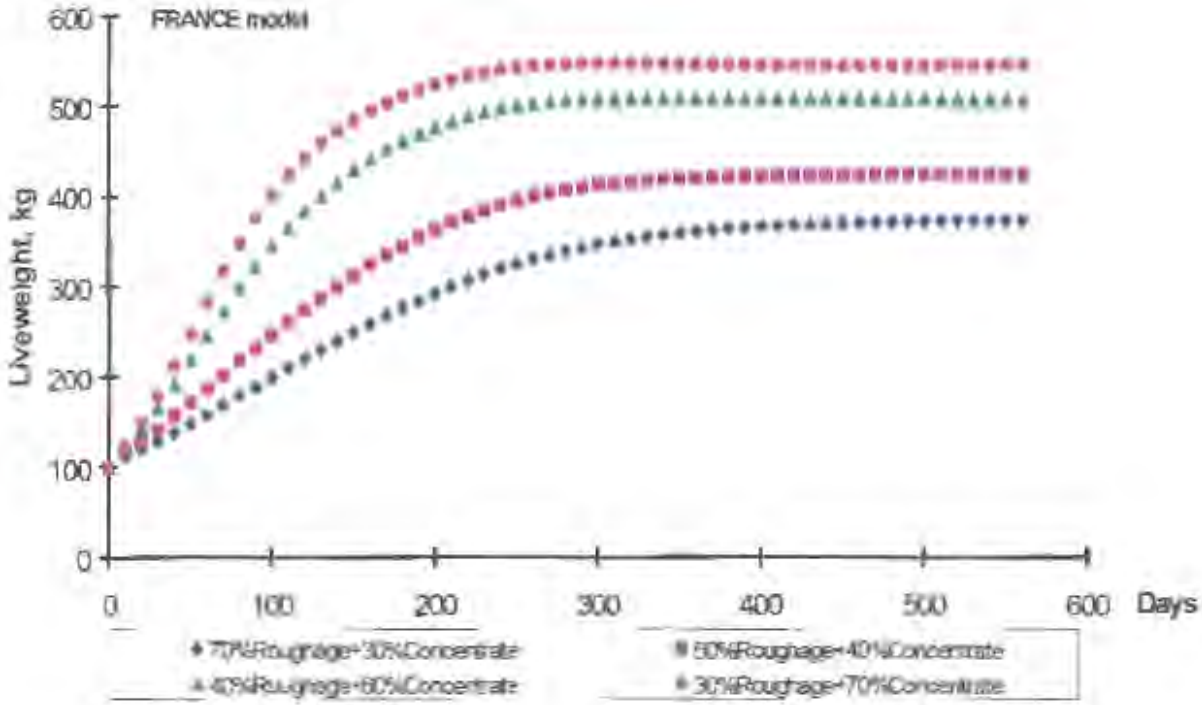


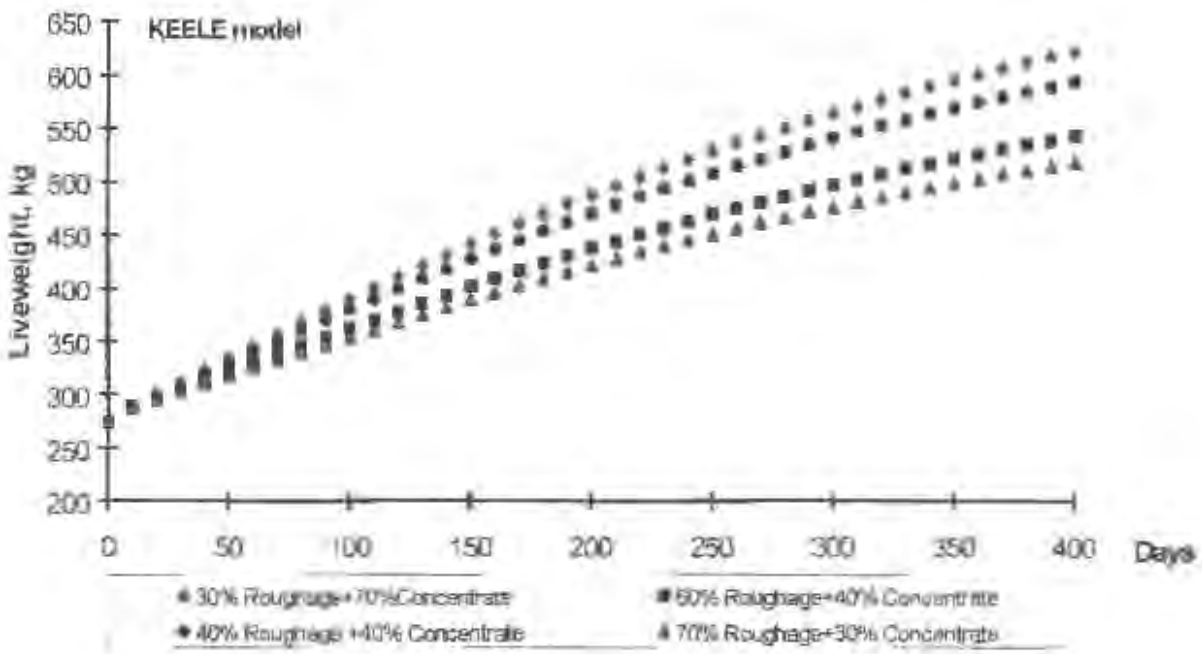
Figure 6.17: The **(a)** liveweight (kg) **(b)** dry matter intake (kg/day) **(c)** protein (kg) and the **(d)** lipid (kg) of the simulated models.

The RUMET model predicted the heaviest liveweight for the 600 days of simulation (Figure 6.17a). For the first 350 days, the FRANCE model predicted a heavier liveweight than the KEELE model but after 350 days of simulation the KEELE model predicted a heavier liveweight. The rate of liveweight gain of both the FRANCE and KEELE models decreased with time (Figure 6.17a).

For a continuously growing animal we would expect the dry matter intake to increase but the rate to decrease as the animal reaches maturity and then the dry matter intake should decrease because of the decrease in rumen volume due to the accumulation of fat around the rumen (Ryan *et al.*, 1993). The KEELE and RUMET models exhibit this phenomenon well. The FRANCE model shows an increase in dry matter intake and then a relatively constant intake when the animal reaches maturity (Figure 6.17b). This is because the rumen volume is kept constant in their model, whereas the dry matter intake is related to liveweight. The simulated lipid of the FRANCE and RUMET models were similar (Figure 6.17d) and the simulated protein by the KEELE and FRANCE models were similar (Figure 6.17c). The KEELE model predicted a continuous increase in the fat pool as an animal matures. As mentioned in section 6.2 and 6.3, the FRANCE and RUMET models do not predict the fat gain of an animal correctly, unless the rate of lipolysis (v_l) is decreased as the animal reaches maturity, whereas the KEELE model does (Figure 6.17d). This is in agreement with the primary

objective of the KEELE model to predict the empty body fat content. Simulating the effects of varying the proportions of roughage and concentrate diet on the liveweight change of the three models is shown in Figure 6.18. All the models simulated the expected result: an animal should have a greater liveweight gain when the diet contains a greater proportion of concentrate than roughage (Steen, 1990).





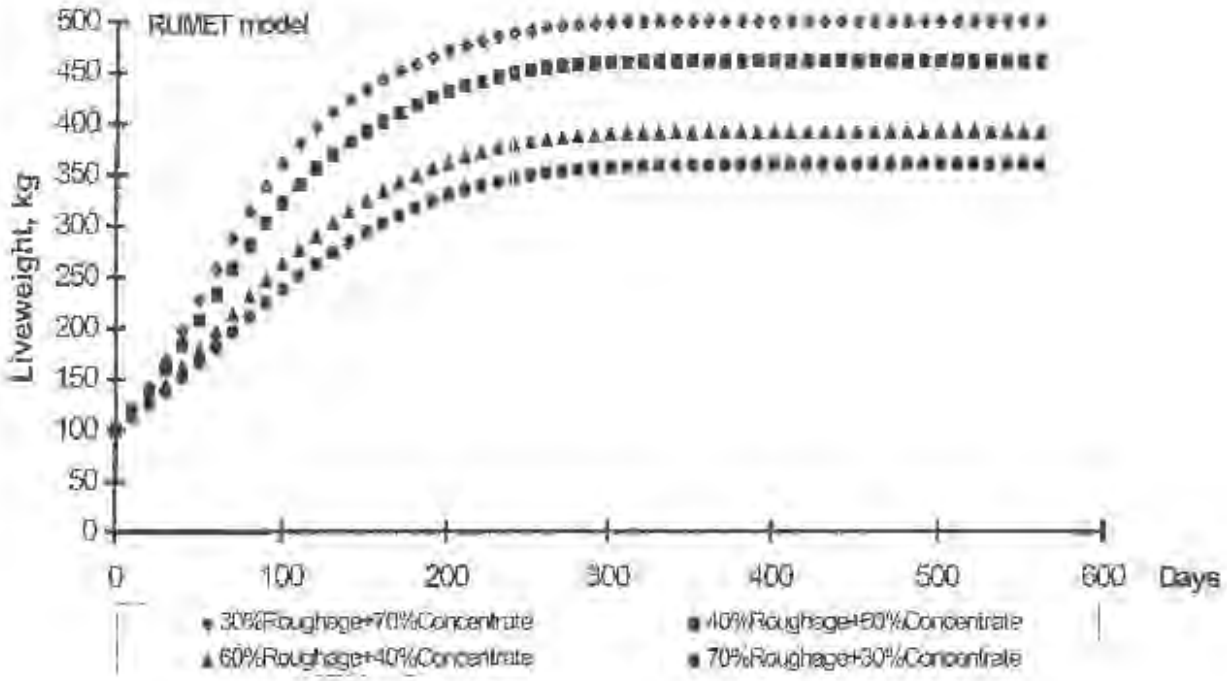


Figure 6.18: Simulating the effects of varying the diet proportions of roughage and concentrate on the weight change predicted by the three models.

6.5.3 Challenging the models with the data of Foot and Tulloh (1977)

The three models were challenged with the dietary regimes imposed by Foot and Tulloh (1977) and Ledger and Sayers (1977). The estimated diet composition and hydrolysis rates of the experiments are shown in Table 6.1. In the experiment of Foot and Tulloh (1977), steers were grown continuously to 330 kg and then fed to lose about 0,5 kg/day for 100 days and then refed ad libitum to reattain 330 kg (see Figure 6.12). The body weight and composition of the steers were recorded for the weight-loss and realimentation phase in Table 6.11 and for the constant weight phase in Table 6.12.

Table 6.11: Results of the simulated and experimental data of Foot and Tulloh (1977) of the body weight and composition during the weight-loss and realimentation phase.

Weight loss/Weight gain

	A				B				C			
	Foot and Tulloh (1977)	KEELE model	FRANCE model	RUMET model	Foot and Tulloh (1977)	KEELE model	FRANCE model	RUMET model	Foot and Tulloh (1977)	KEELE model	FRANCE model	RUMET model
Days	0	0	0	0	100	100	100	100	142	150	140	144
Weight	330*	275	276	279	——	230	209	228	330	2769	285	278
Dry Matter intake (g/day)	6780	5549	5564	5625	——	1513	1406	1503	7180	6709	7021	6745
Body Composition:												
Ash (kg)	11.6	——	12.4	10.8	——	——	15.4	16.3	12.1	——	20.6	16.8
Total protein (kg)	49.2	——	59.9	66.5	——	——	47.1	50.1	48.7	——	64.4	61.2
Liver protein (kg)	4.4	——	——	2.4	——	——		0.2	3.5	——	——	1.9
Muscle protein	——	——	——	41.7	——	——	——	41.7	——	——	——	44.6
Water (kg)	——	——	173.6	185.6	——	——	150.9	159.4	——	——	203.6	187.1
Lipid (kg)	24.2	30.8	30.8	23.0	——	8.1		7.5	20.7	18.4	1.8	18.5
Energy Retention	——	-22.81	-22.32	-5.93	——	-7.94	-15.23	-12.20	——	17.35	10.560	13.08
Maintenance energy expenditure (MJ ME/$W^{0.75}$)	——	0.368	0.341	0.417	——	0.421	0.352	0.270	——	0.367	0.344	0.378

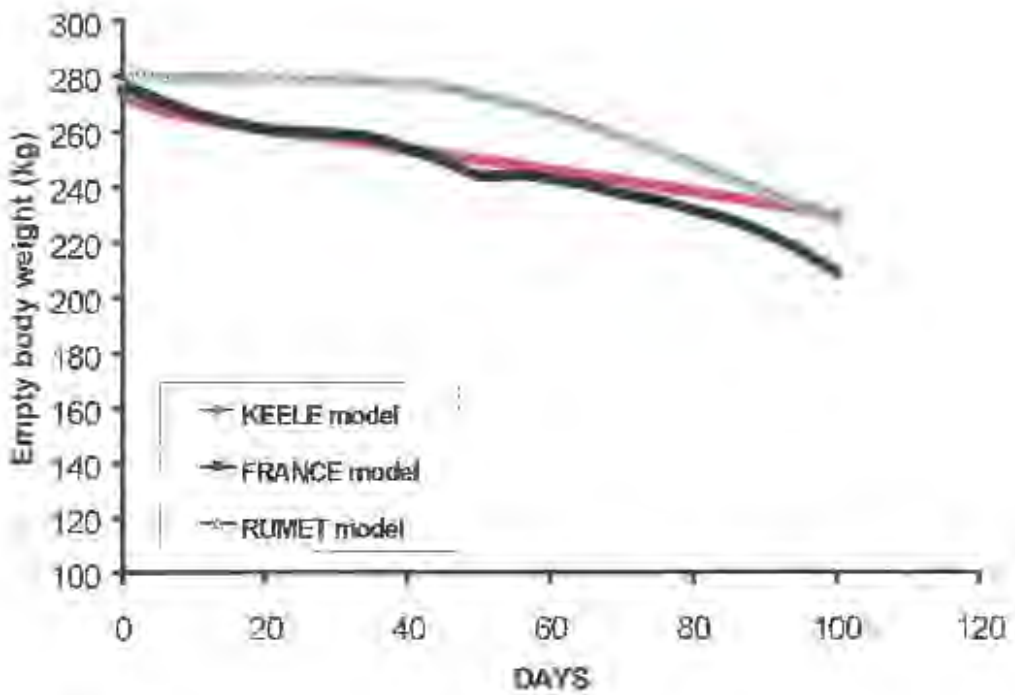
*Liveweight (kg)

Table 6.12: Results of the simulated and experimental data of Fiot and Tullon (1977) of the body weight and composition during the constant weight phase.

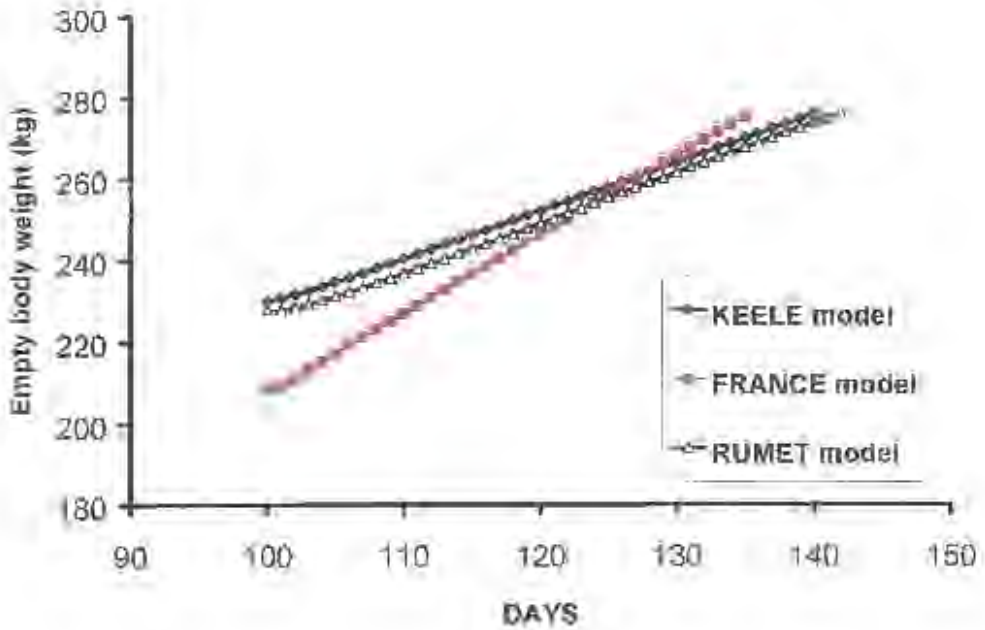
	A				B			
	Fiot and Tullon (1977)	KEELE model	FRANCE model	BUNNET model	Fiot and Tullon (1977)	KEELE model	FRANCE model	BUNNET model
Days	0	0	0	0	100	100	100	100
Weight	330*	275	277	279	—	274	282	283
Dry Matter intake (g/day)	6780	5549	5564	5625	—	4473	3825	4147
Body Composition:								
Ash (kg)	11.6	—	12.4	10.8	—		16.4	17.2
Total protein (kg)	49.2	—	59.9	66.5	—		55.4	62.4
Liver protein (kg)	4.4	—	—	2.4	—			1.3
Muscle protein	—	—	—	41.7	—			43.6
Water (kg)	—	—	173.6	185.6	—		172	191
Lipid (kg)	24.2	30.8	30.8	22.9	—	34.1	13.4	15.8
Energy Retention (MJ/day)	—	-22.31	-22.32	-5.93	—	2.97	-4.76	-3.44
Maintenance energy expenditure (MJ ME/$W^{0.75}$)	—	0.368	0.341	0.417	—	0.368	0.347	0.344

When the model animal reached point A in Figure 6.12, model body composition values were set equal to the body composition values achieved by the animals in their experiment.

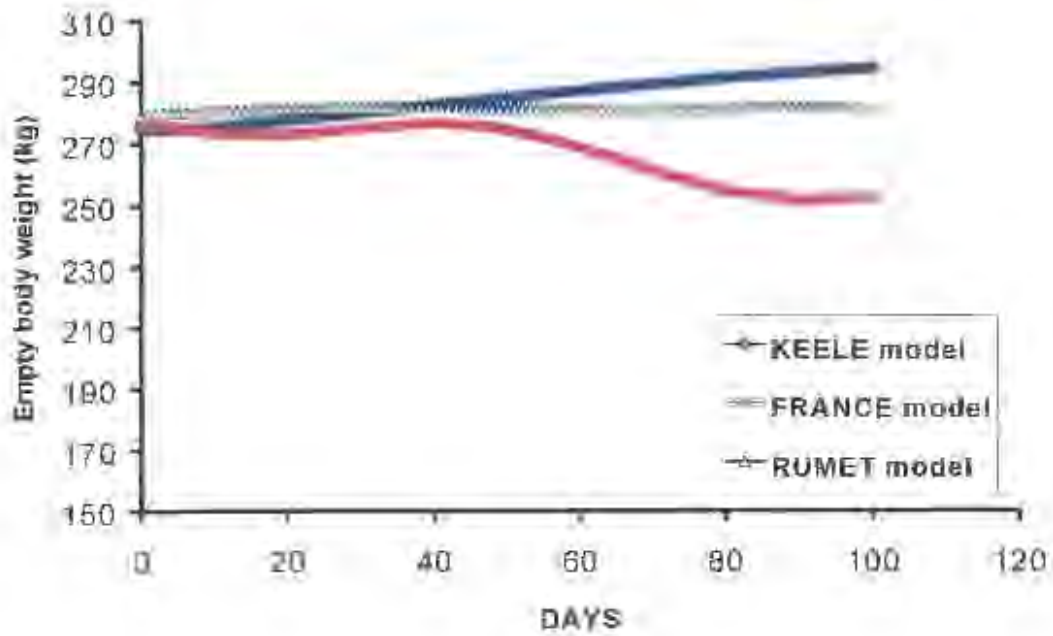
The dry matter intake (kg/day) of the weight loss group was given in their paper and as equation 6.4. Foot and Tulloh (1977) showed that the dry matter intake of the steers at the beginning of the experiment (i.e., at the beginning of the weight-loss phase) was an average of 7.36 kg/day and at the end of the experiment it was 2.1 kg/day. Figure 6.19a shows the weight loss predicted by the three models. All three models predicted a weight loss of about 15% in 100 days as presented by Foot and Tulloh (1977).



(a)



(b)



(c)

Figure 6.19: Simulated liveweight (kg) for the **(a)** weight loss animal, **(b)** the realimented animal, and **(c)** the constant weight animal.

6.5.4 Challenging the models with the data of Ledger and Sayers (1977)

In part of the experiment of Ledger and Sayers (1977), *Bos indicus* steers were fed to maintain a constant liveweight of 275kg. They showed that the feed intake and metabolizable energy expenditure of the steers decreased with time indicating that the efficiency of nutrient use increased with time. The dry matter intake (g/week) of the constant weight animal is given in their paper and in equation 6.5. Figure 6.20 shows the liveweight predicted by the models.

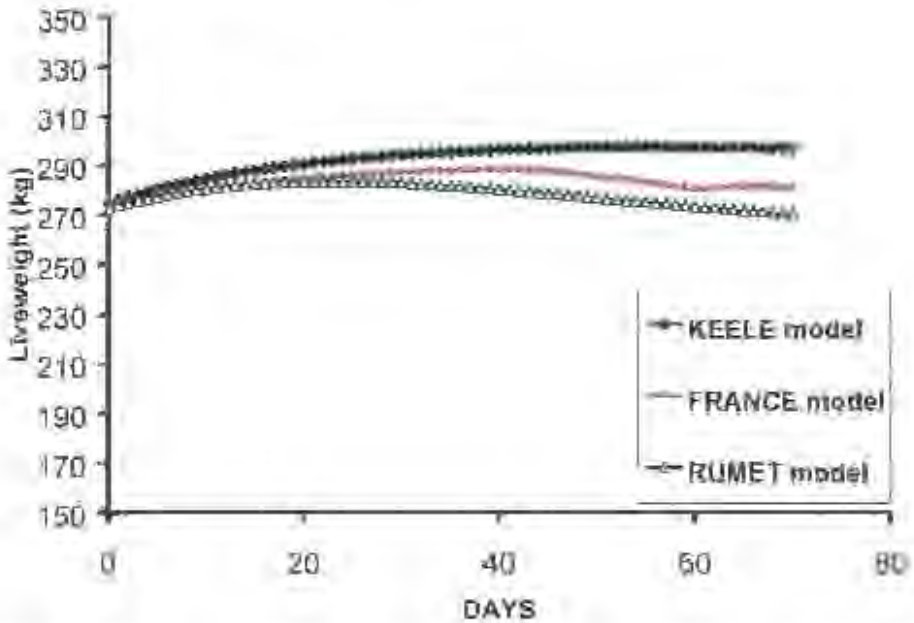


Figure 6.20: Simulated liveweight for the three model animals subjected to the experimental diet of Ledger and Sayers (1977).

6.6 Discussion

The simulation results of the three models relative to the experimental data of Foot and Tulloh (1977) is given in Table 6.11. Because the weights of the body components at point **B** on Figure 6.12 was not given by Foot and Tulloh (1977), model results will be compared with experimental data at the end of the experiment, that is, after the realimentation period. At point **A** the feed intake pattern of Foot and Tulloh (1977) was used as input to the models (till **B**). Thereafter, the models simulated the *ad libitum* feeding to restrain 330kg liveweight.

At the end of the restricted period, the KEELE model predicted the heaviest body weight and fat content. After realimentation, the KEELE model predicted the shortest rehabilitation time of 34 days (Figure 6.19b) but the fat content was predicted correctly. The realimentation time of the FRANCE and RUMET was close to the

realimentation time of 42 days in the experiment of Foot and Tulloh (1977) (42 days and 44 days, respectively). However, unlike the FRANCE model, the RUMET model predicted the lipid content correctly but both models overestimated the ash and protein content. The underprediction of the lipid content by the FRANCE model could be explained by the fact that the model was used to simulate younger and potentially larger animals (bulls of large breed). All the models correctly predicted the continuous increase in the ash pool during the restricted phase. The liver weight was correctly predicted by both the KEELE and RUMET models.

All the models correctly predicted a decrease in the maintenance energy expenditure when simulating a constant weight steer. The important implication is that the changes in energy expenditure by the liver and small intestine can be calculated. However, the protein content was overpredicted and the lipid content underpredicted by both the FRANCE and RUMET model. It could be that the rate of lipolysis (v_l) is not the same for an animal fed to maintain a constant weight or for an animal fed to lose weight. This could be due to the effects of a number of hormones controlling the partitioning of energy between fat and protein.

The linking of the models with the rumen model had a major effect on the results of the models: it allowed for the simulation of a realistic feeding response during realimentation and the improved estimation of the availability of absorbed nutrients and, therefore, energy absorbed. France *et al.* (1987) suggested that their model be linked with a rumen model and that this may improve the prediction of carcass composition.

Comparing models does not determine if any of the models are more accurate than the others, but it does show that results are not similar. Thus if incorporated into a production system model, these models would be expected to produce different results. Therefore, management decisions based on these models could differ depending on which model is used. However, challenging the models with the data of Foot and Tulloh (1977) and Ledger and Sayers (1977) assists us in the evaluation of these models. Generally, it was shown that the RUMET model predicts the experimental data of Foot and Tulloh (1977) and Ledger and Sayers (1977) most closely.

APPENDIX 6.1

Model equations of the OLTBLAX model for a 650 kg steer

$$\frac{dENERGY}{dt} = Hb \times \left[Bke \times \frac{}{(1+e^{(-p)})} \right] \quad \text{(Blaxter and Boyne (1978)} \qquad 6.22$$

where $Hb = Fm \times EBW^{0.67}$ (Basal metabolism) 6.23

$$Bke = \frac{EGM}{EGM + EGG}$$

$$p = EGM \times Ln\left(\frac{EGM}{EGG}\right)$$

$$g = \frac{Gb}{Hb}$$

$$EGM = 0.503 \times Qen - 0.35 \times (Qen)^2$$

$$EGG = 0.006 \times Qen + 0.78 \times (Qen)^2$$

$$Qen = 0.82 \times \text{Digestible energy}$$

$$\frac{dDNA}{dt} = K1 \times (DNAMX - DNA) \times NUT1 \qquad 6.24$$

$$\frac{dPROT}{dt} = SYNTHESIS - DEGRADATION \qquad 6.25$$

where $SYNTHESIS = K2 \times DNA^{0.73} \times NUT2$ 6.26

$$DEGRADATION = K3 \times PROT^{0.73} \qquad 6.27$$

$$\frac{dFAT}{dt} = \frac{\frac{dES}{dt} - 23.6 \times \frac{dPROT/dt}{dt}}{39.3} \qquad 6.28$$

$$\frac{dEBW}{dt} = 4.252 \times \frac{dPROT}{dt} + \frac{dFAT}{dt} \qquad 6.29$$

$$r' = r\left(\frac{A'}{A}\right)^{0.73} \qquad 6.30$$

$$P = \frac{MNJ}{MEINORM} \qquad 6.31$$

$$MEINORM = (1.83 - 1.094 \times \frac{EBW}{A}) EBW^{0.75} \qquad 6.32$$

$$NUT1 = -0.7 + 1.7P \qquad 6.33$$

$$NUT2 = 0.83 + \frac{0.2P}{0.15+P} \qquad 6.34$$

Chapter 7

Dynamic control of ammonia and urea by diffusion across the rumen wall and saliva

7.1 Nitrogen recycling in the ruminant

It is well recognized that ruminants are especially well adapted to survive under unfavourable environmental conditions. This ability is of particular interest under poor nutritional conditions. The ability of the ruminant to conserve body nitrogen or to use different sources of nitrogen is important for survival. Nitrogen is a critical nutrient in the ruminant since it is the key component in protein (amino acids). The ruminant can only use amino acid nitrogen as a nutrient at the tissue level, so bacteria in the rumen can convert non-protein nitrogen (primarily as ammonia) to bacterial protein. These bacteria are subsequently digested by the animal and their protein is used to supply amino acids for production: deposition in milk, wool or animal tissue.

If the diet is deficient in protein or if the protein resists degradation by microorganisms, the concentration of rumen ammonia will be low and the growth of microorganisms slow. Consequently, the breakdown of carbohydrates will be retarded. On the other hand, ruminal bacteria cannot produce enough protein to meet the demand for maximum production of the animal (Bergen and Merkel, 1991). In such cases, the productivity of the animal depends on the selection of feeds and supplements to maximize bacterial production and, if required, to supply protein that will escape digestion in the rumen and pass to the small intestine to supply additional amino acids.

If production of ammonia in the rumen from the food (such as non-protein nitrogen or feed protein) exceeds the capacity of the bacterial population to use ammonia, ammonia will accumulate in the rumen fluid and be absorbed in the blood and converted to urea in the liver. Urea may return to the rumen either by diffusion across the rumen wall (Coenraad and Leng, 1961) or via saliva (Schmidt-Nielsen, 1957), but a greater part will be excreted in the urine (Figure 7.1)

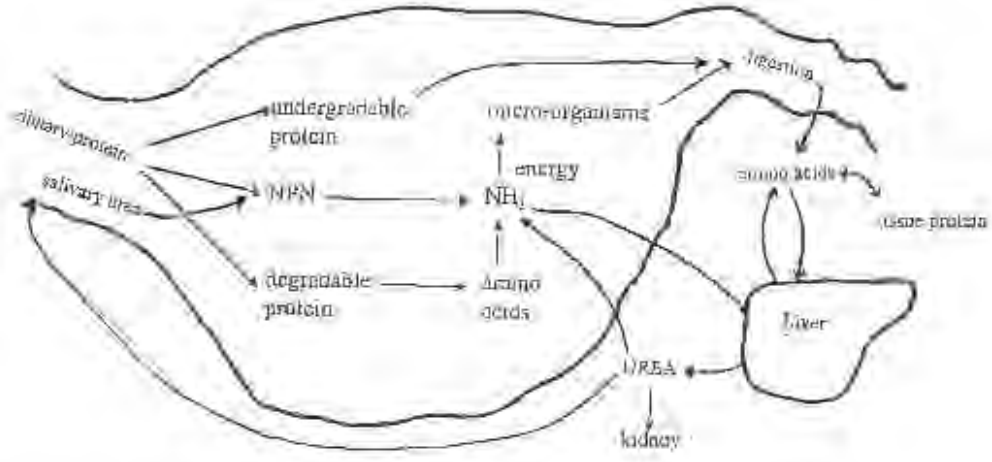


Figure 7.1: Nitrogen recycling in the ruminant.

Houpt (1970) hypothesised that urease is essential in the facilitation of the transfer of urea-N from the blood to rumen. In Houpt's (1970) proposal, nitrogen transfer to the rumen is dependent on a favourable ammonia concentration gradient between epithelial tissue and rumen contents and implies a continual production of ammonia from urea, and the movement of ammonia into the portal bloodstream when rumen ammonia is high. Cheng and Wallace (1979) showed an inverse relation between ammonia concentration and the urease activity by the wall-adherent bacterial population, which implies a reduction in urea hydrolysis through the partial or complete inhibition of urease when rumen ammonia concentration increases. The experiments of Whitelaw *et al.* (1992) supports the experiments of Houpt (1970) and suggests that diffusion of ammonia into the portal blood must limit the transfer of nitrogen into the rumen when rumen ammonia concentration increases. Kennedy (1980) suggested that both the concentration of the plasma urea and rumen ammonia affect the transfer of urea to the rumen of cattle. His experiments confirmed his previous work (Kennedy and Milligan, 1978) that low urea transfer to the rumen was limited by high concentrations of rumen ammonia or low intake of digestible organic matter.

The objective of this chapter is to extend the RUMET model to examine the mechanisms of nitrogen recycling to the rumen via the saliva and diffusion across the rumen wall. This chapter is organised in the following manner. In section 7.2 the results of a sensitivity analysis of the RUMET model (before modifications) will be presented. In this section the possible effects of changing seven parameters, thought to be important in the recycling of nitrogen across the rumen wall and via the saliva, on

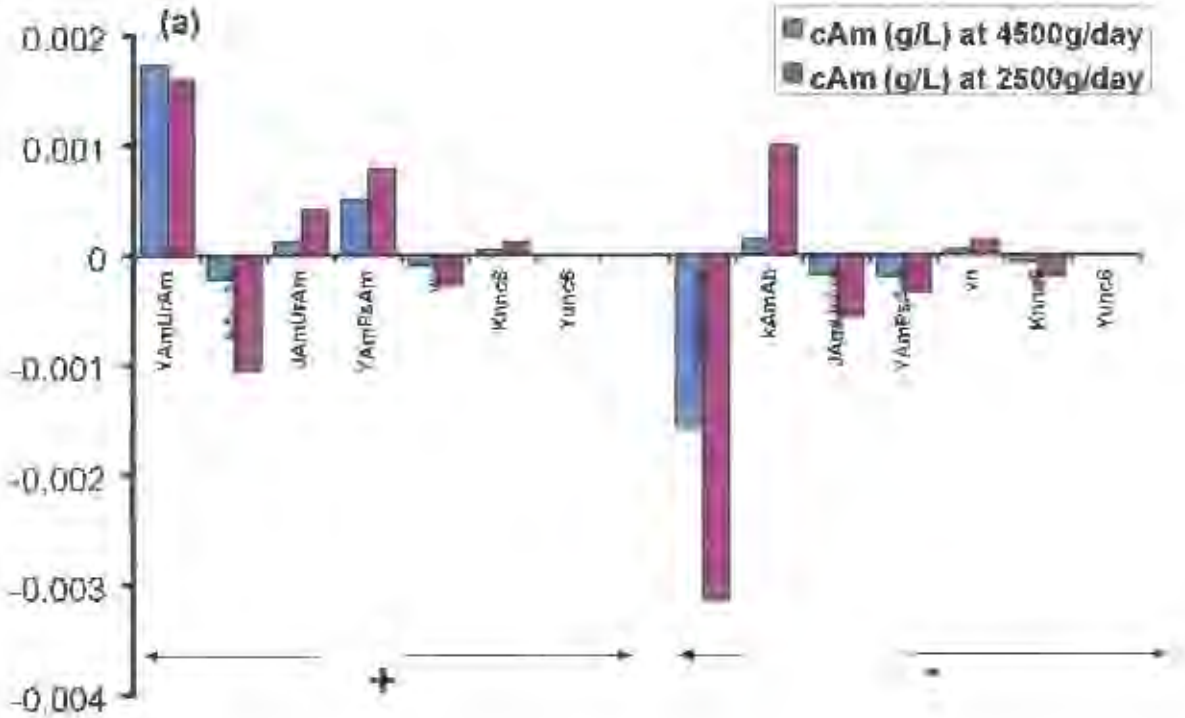
four variables will be examined. The sensitivity analysis was done before the RUMET model was extended to examine whether the addition of the nitrogen recycling mechanisms affected rumen function and energy partitioning in intermediary metabolism. In section 7.3, the modifications of the RUMET model will be described and additional parameter values explained. The notation and symbols used will be the same as in chapter 3 and chapter 5. In section 7.4 the application of the model will be described and the results of the model will be presented in section 7.5. Conclusions will be given in section 7.6 and model equations of the nitrogen recycling component are listed in Appendix 7.1

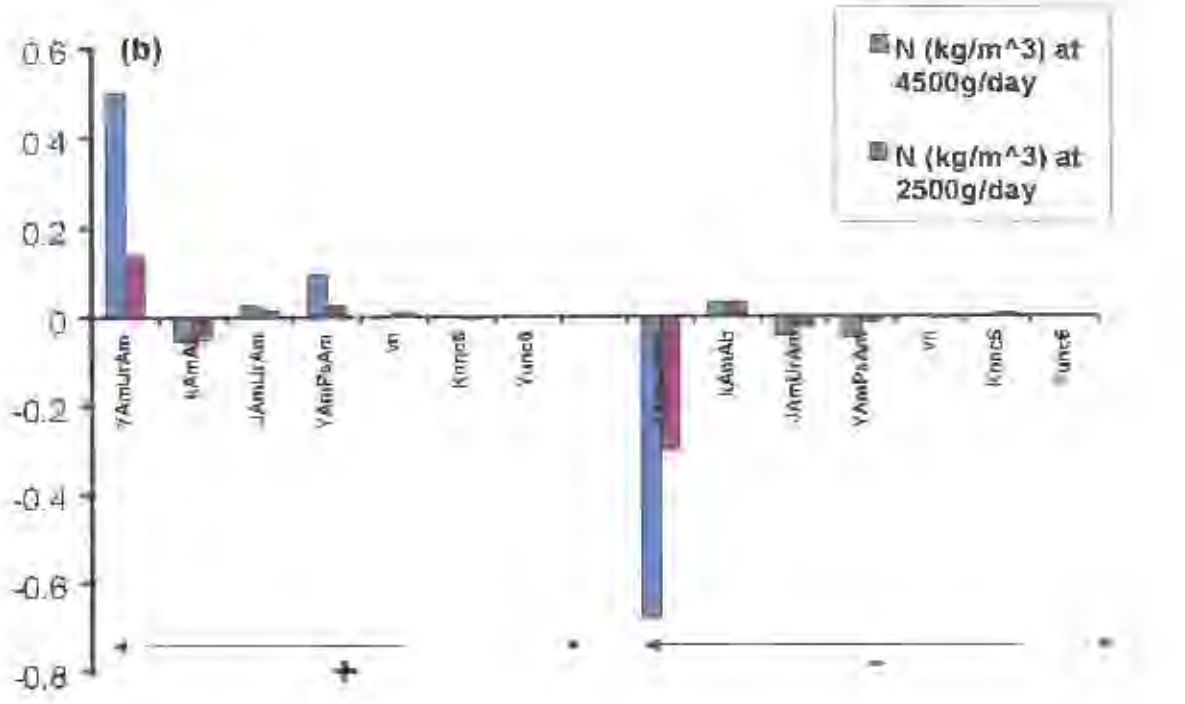
7.2 Motivation for model changes: sensitivity analysis

Seven parameters ($Y_{Am/UrAm}$, k_{AmAb}, $J_{Am/UrAm}$, $Y_{Am/PsAm}$, V_N, K_{NC_6}, Y_{Ur/NC_6}), which are described in Table 7.1, were doubled and halved so that the effects on the concentration of ammonia in the rumen (cAm, $\frac{g}{L}$), the concentration of amino acids in the blood (N, $\frac{kg}{m^3}$), the production of urea (Urea, kg), and the amount of total tissue protein (Protein, kg) could be examined. The metric used for the sensitivity analysis is the absolute difference of the values of the variables when no parameters were changed with the values of the variables when the parameters were changed after 20 days of simulation time. The model was run with two constant amounts of food (4.5 kg/day and 2.5 kg/day). Figure 7.2 below shows the results of the sensitivity analysis: a positive sign (+) indicates the effect of doubling the parameter values and a negative sign (-) indicates the effect of halving the parameter values.

Table 7.1: Description of the parameters and variables used in the sensitivity analysis

Notation	Description	Units
$Y_{Am/UrAm}$	Yield of ammonia from urea to ammonia	g Am/g N
k_{AmAb}	rate of absorption of ammonia across the rumen wall	/day
$J_{Am/UrAm}$	Inhibition of ammonia on urea transport across the rumen wall	g/L
$Y_{Am/PsAm}$	Yield of ammonia from soluble protein to ammonia	g Am/g Ps
V_N	rate of gluconeogenesis from N→C_6	/day
K_{NC_6}	Affinity constant for N from N→C_6	kg/m^3
Y_{Ur/NC_6}	Yield of Urea from N→C_6	kg Urea/kg N
cAm	Concentration of ammonia in the rumen	g/L
N	Amino acid pool	kg N/m^3
Ur	Production of urea	kg
Protein	Total body protein	kg


(a)
cAm (g/L) at 4500g/day
cAm (g/L) at 2500g/day
0.002
0.001
0
-0.001
-0.002
-0.003
-0.004
YAmUrAm
kAmAb
+
-


(b)
N (kg/m^3) at 4500g/day
N (kg/m^3) at 2500g/day
0.6
0.4
0.2
0
-0.2
-0.4
-0.6
-0.8
YAmUrAm
+
-

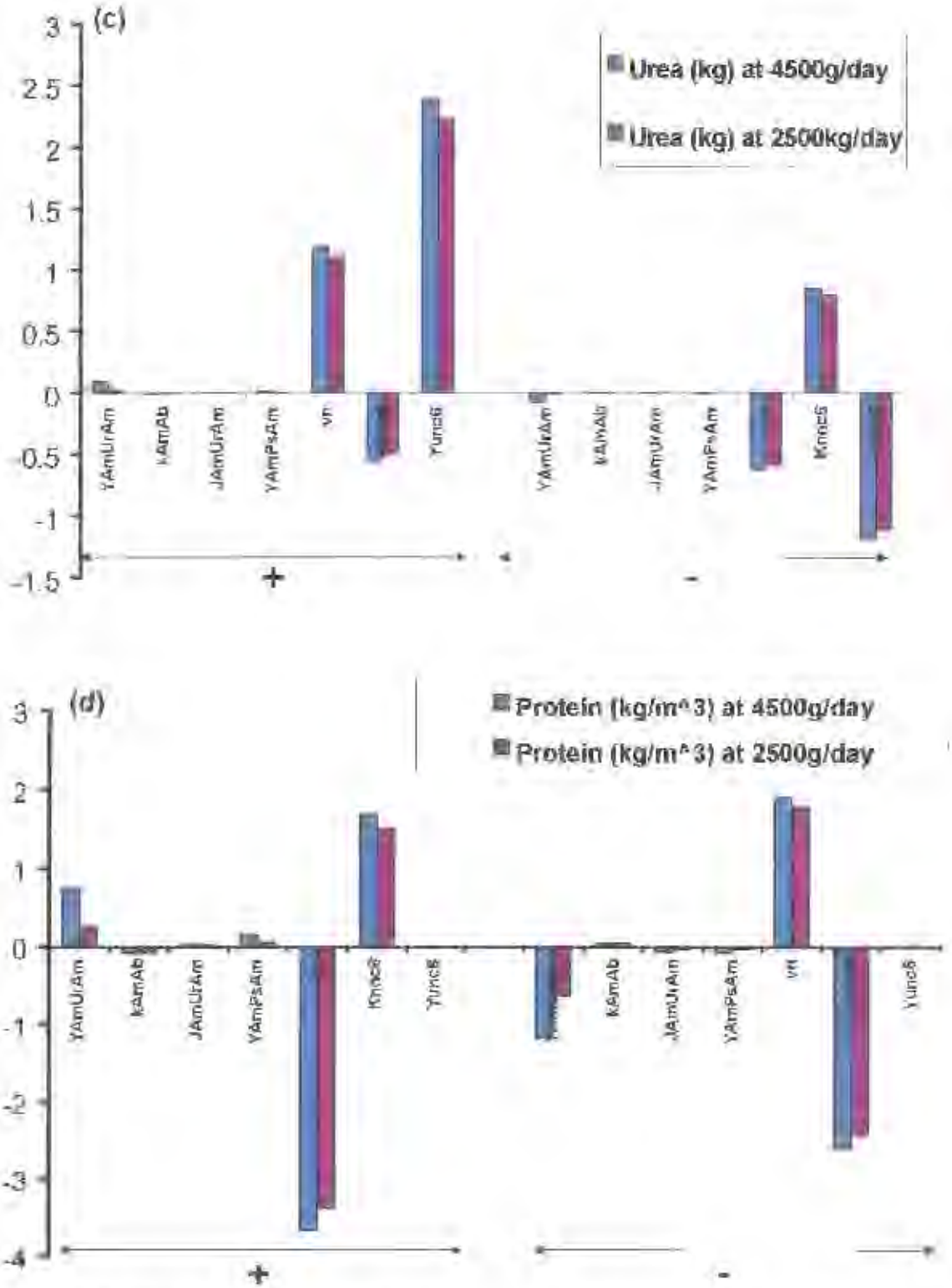


Figure 7.2: Effect of doubling and halving seven key parameters ($Y_{Am/Urea}$, $k_{Am/Ab}$, $J_{Am/Urea}$, $Y_{Am/PsAm}$, V_M, $K_{Nucl_{Deg}}$, Y_{Urea}) on the (a) concentration of ammonia in the rumen (cAm, g/L), (b) amino acid (N, kg/m^3) concentration in the blood (c) urea production (Urea, kg), and (d) total body protein (Protein, kg/m^3).

increasing and decreasing $Y_{Am/AAm}$ had the greatest effect on the amino acid (N) concentration in the blood (Figure 7.2b). This is evidence of an increase in absorbed amino acids to metabolism as a result of the greater yield of urea nitrogen transport to the ammonia pool in the rumen (Figure 7.2a). This indicates the importance of representing this mechanism of nitrogen recycling more explicitly. An interesting result is that the effect of doubling $Y_{Am/UrAm}$ on the blood amino acid concentration was greater on the 4.5kg/day diet whereas the effect of halving $Y_{Am/UrAm}$ on the blood amino acid concentration was greater on the 2.5kg/day diet. Doubling and halving $Y_{Am/PAm}$ had the greatest effect on the concentration of rumen ammonia, which indicates the need to account for the recycling of nitrogen to the rumen via the saliva. Also, changing $Y_{Am/PAm}$ had a larger effect on the 2.5 kg/day diet. Increasing the rate of gluconeogenesis had the obvious result of decreasing the tissue protein concentration and increasing the blood urea production but also decreased the concentration of ammonia in the rumen (Figure 7.2a). This initial analysis supports the need to include a nitrogen recycling component via the saliva and transport from the blood to the rumen.

7.3 The Simulation Model

The scheme of the isolated nitrogen recycling components is shown in Figure 7.3 and the nitrogen recycling components included in the RUMET model is shown in Figure 7.4.

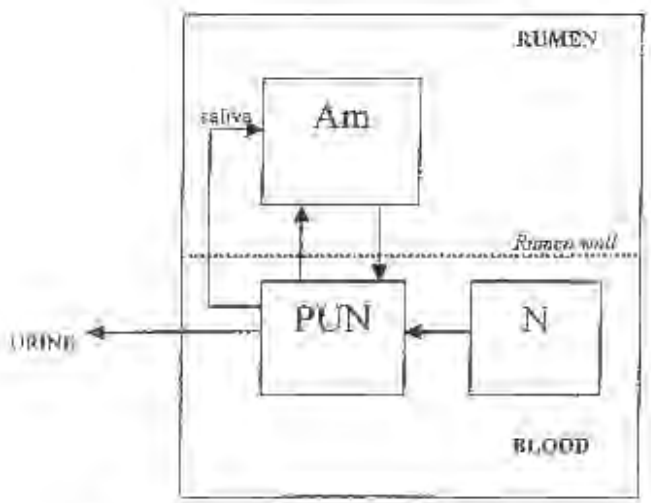


Figure 7.3: Scheme of the N recycling model

The RUMET model was modified to account for the nitrogen recycling to the rumen via the saliva and absorption through the rumen wall. The extra parameters and variables are described in Table 7.2. For convenience, all urea nitrogen excreted is represented as urea, even though a proportion of urinary nitrogen is in the form of purine bases.

Table 7.2: Description of the parameters and variables used in the model

Notation	Translation	Units
P_{Ureamilk}	Rate of production of PUN in Am-PUN transaction	kg Ur/day
P_{UrPUN}	Rate of production of urea in N-PUN transaction	kg urea/day
V_{UreAm}	velocity for PUN-Am transaction	kg/day
k_{Amabs}	Fractional rate constant for ammonia absorption	/day
U_{UreUAm}	Rate of utilization of urea nitrogen by PUN-Am transaction	kgN/day
U_{UreUrea}	Rate of utilization of urea nitrogen by PUN-Am transaction	kgN/day
U_{Ure}	Rate of urinary urea-N excretion	kgN/day
C_{urdif}	N concentration difference between PUN and Am pools	g or kg N
K_{UreUrea}	Affinity constant for PUN-Am transaction with respect to PUN	gN/gPUN
SalN	Concentration of urea nitrogen in mixed saliva	mg/100ml
Salday	Rate of salivary secretion	L/day
Y_{AmUAm}	Yield of Am for PUN-Am transaction	g N/gAm
U_{AmAmAm}	Rate of utilization of Am by PUN-Am transaction	gN/day

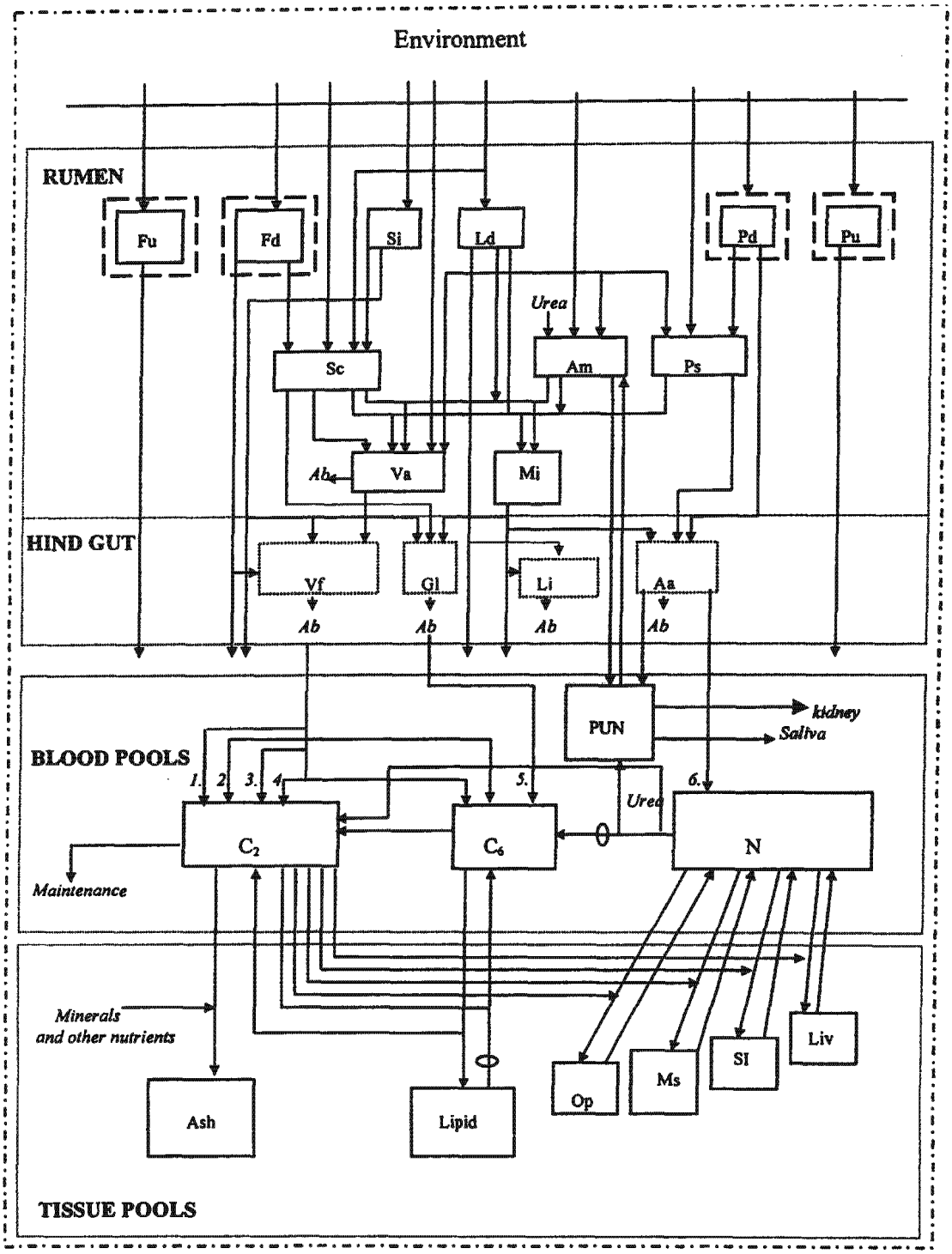


Figure 7.4: The RUMET model, an integrated rumen and intermediary metabolism model, showing the nitrogen recycling components. 1. Acetate, 2. Stearate, 3. Butyrate, 4. Propionate, 5. Glucose, and 6. Amino acids are shown.

Plasma urea nitrogen pool, PUN (kg plasma urea nitrogen per m^3)

There are two inputs to this pool: urea production as a product of amino acid catabolism from the amino acid pool to glucose (P_{Ur,NC_6}: equation 7.1), and the transfer of ammonia nitrogen across the rumen wall ($P_{Ur,AmUr}$: equation 7.2). Urea production (kg urea) was described by France *et al.* (1987) as the yield of urea multiplied by the utilization of nitrogen from the amino acid pool to glucose (U_{N,NC_6}). It is assumed that this also includes the urea-N from glycogenic amino acids. Urea production was converted to kg urea nitrogen by multiplying P_{Ur,NC_6} by a yield factor, $Y_{N,NC_6} = 0.46/1000 = 0.00046$. It has been shown that a high concentration of ammonia in the rumen fluid favours the flux of nitrogen into the bloodsteam (Parker *et al.*, 1995). Free ammonia diffuses readily across biological membranes because of its lipid solubility and lack of charge. Net ammonia flux across the rumen wall has been shown to be proportional (linear) to the rumen ammonia concentration (Remond *et al.*, 1993), but this work has been controversial and there may be some stimulation of ammonia uptake by VFA (Boderker *et al.*, 1992), although the mechanism involved is not clear. It is assumed here that the utilization of ammonia across the rumen wall is a linear function of the ammonia pool (Appendix 3.1: equation 3.31g). The fractional absorption rate (k_{AmAb}) is assumed to be 12/day (Siddons *et al.*, 1985).

There are three outputs from this pool: the excretion of urea nitrogen via the urine (U_{UEx}, equation 7.3), the transfer of urea nitrogen across the rumen wall ($U_{Ur,UrAm}$: equation 7.4), and the transfer of urea nitrogen to the rumen via the saliva ($U_{Ur,UrSal}$: equation 7.6). There is experimental evidence (see for example, Thornton, 1970) that shows an inverse relation between the amount of urea entering the rumen and that excreted in the urine. However, Harrop and Phillipson (1974) suggested that the inverse relation was due to a reduced plasma urea level when entry of urea into the rumen was high (for example, during periods of feeding), rather than to special conserving mechanisms in the renal tubules (Schmidt-Nielsen *et al.*, 1957; 1958). Urea excretion has also been found to be directly proportional to plasma urea concentration (Richardson and Wright, 1984). McIntire and Williams (1970) and Harrop and Phillipson (1971) suggested that it was unlikely than the renal excretion of urea played an important role because animals on different diets had plasma levels above 20 mg urea N/100ml. Richardson and Wright (1984) showed that urinary urea excretion and plasma urea nitrogen concentration increased with an increase in protein content of the diet. They showed that urinary urea excretion (U_{UEx}) declines with an increase in the amount eaten, but that the effect on plasma urea nitrogen was small.

They found that urinary urea excretion was not a simple function of plasma urea nitrogen concentration (PUN) and that urinary urea excretion was a linear function of nitrogen intake among animals given the same amount of food per unit of metabolic size. The following equation was derived from data in Richardson and Wright (1984) for the utilization of urinary urea nitrogen excretion (U_{UEx}):

$$U_{UEx} = Uu1 \times e^{(Uu2 \times PUN)} \tag{7.3}$$

where $Uu1^* = 8.1123$, $Uu2^* = 12.114$.

Cheng and Wallace (1979) proposed a conceptual model for the transport of urea from the blood across the rumen wall which was essentially similar to that of Houpt (1970). They proposed that bacterial urease activity associated with the epithelium serves to maintain a localised concentration gradient of urea across the rumen wall. They extended this model to include the control of urea flux by rumen ammonia concentration. In addition, the results of Harrop and Phillipson (1974) suggested that an upper limit exists to the transfer of urea from the blood to the rumen. The following equation is proposed for the utilization of urea nitrogen from the PUN pool to the rumen:

$$U_{Ur,UrAm} = \frac{v_{UrAm} \times C_{diff}}{1 + \frac{K_{Ur,UrAm}}{C_{diff}}} \tag{7.4}$$

$$\text{where } C_{diff} = PUN - 0.82 \times C_{am}. \tag{7.5}$$

The parameters v_{UrAm} and $K_{Ur,UrAm}$ were estimated using the given values for the utilization of plasma urea ($U_{Ur,UrAm}$, g N/day) into the rumen and the concentration of rumen ammonia and concentration of plasma urea nitrogen, for an animal fed two roughage diets (Australian pasture-hay) in the experiments of Kennedy (1980). Having two sets of these values enabled solving the equations for the two unknown parameters. The value of v_{UrAm} was estimated to be 0.644/day and the value of $K_{Ur,UrAm}$ was estimated to be 5.702g/L.

Bailey and Balch (1961) showed that urea nitrogen constituted a high (but variable) constituent of the total nitrogen in saliva (SalN). The non-urea nitrogen fraction was found to be relatively constant at 1-2 mg/100ml, irrespective of the diet, but the saliva urea nitrogen content varied from 1.3 to 14.4 mg/100ml. They showed that both the quantity of total nitrogen in saliva and urea nitrogen in saliva depends on the nature of

*derived by nonlinear regression analysis

the diet and that urea nitrogen constituted a large proportion of the total nitrogen in saliva: the overall mean (for the diets in their experiments) being 77.2% of the total nitrogen in saliva. Bailey and Balch (1961) found the following relation:

$$SalN = s1 \times PUN^{*} - s2 \qquad (7.7)$$

where $s1 = 0.7083$ and $s2 = 0.8328$ and where SalN is the concentration of urea nitrogen in mixed saliva (mg/100ml) and PUN^{*} is the concentration of urea nitrogen in the blood (mg/100ml). PUN was multiplied by 100 to convert the units from kg/m^3 to mg/100ml. To convert SalN (mg/100ml) to g/L, it is required to divide SalN by 100. What was ultimately required is the utilization of urea nitrogen from PUN to the ammonia pool ($U_{Ur,UrAm}$) and it was therefore necessary to multiply the concentration of urea nitrogen in mixed saliva (SalN) by the rate of saliva secretion (Salday, L/day). Because the units of ammonia in the rumen are in g/L and the units of the PUN pool are in kg/m^3, $U_{Ur,UrAm}$ was divided by 1000.

Bailey (1961) found a mean salivary secretion of 229 ml saliva per minute, however, the rate of secretion varied from 108 ml/min to 250 ml/min on their experimental diets. They found that for most foods the rate of secretion is between 200-300 ml saliva/min. The rate of saliva secretion was assumed a linear function of the sum of the degradable fibre of the two components of the diet:

$$Salday = 0.332 \times 1000 \times (cFd1 + cFd2) + 116.9 \qquad (7.8)$$

so that the rate of secretion varied predominantly between 200-300ml saliva/minute.

Ammonia pool, Q_{Am}(g).

There are three inputs to this pool: input of Am with the diet ($P_{Am,InAm}$: equation 7.11), the production of Am from urea transported across the rumen wall ($P_{Am,UrAm}$: equation 7.12) and the production of Am by fermentation to Ps and via the saliva ($P_{Am,PsAm}$: equation 7.13). The amount of urea-N transferred across the rumen wall is calculated as the yield of urea nitrogen from the PUN pool ($Y_{Am,UrAm}$) multiplied by $U_{Ur,UrAm}$. Because the units for the PUN pool is kg/m^3 and the units for the concentration of rumen ammonia is g/L, $Y_{Am,UrAm} = 1000$ gN/gAm. The amount of urea-N transferred from the saliva is calculated as the yield of urea nitrogen from the PUN pool ($Y_{Am,PsAm}$) multiplied by $U_{Ur,UrSal}$. Because the units for the PUN pool is kg/m^3 and the units for the concentration of rumen ammonia is g/L, $Y_{Am,SalAm} = 1000$ gN/gAm. The other inputs to this pool have been described in chapter 3.

There are four outputs from this pool: Am is used for microbial growth with Ps or Am ($U_{Am,PsMi}$, $U_{Am,AmMi}$), the absorption of Am through the rumen wall ($U_{Am,AmAb}$) and the outflow of Am with the rumen fluid ($U_{Am,AmEx}$). These are described in chapter 3.

7.4 Simulation Protocol

The simulation modelling approach allowed for the evaluation of the recycling of nitrogen to the rumen via the saliva and across the rumen wall. The extent to which nitrogen metabolism adapts to changes in body condition and intake was also examined. The extended RUMET model was used to simulate the growth of a steer (initial values: EBW = 195kg, Protein = 45.6kg, Lipid = 20.3kg) fed a roughage diet which was supplemented with increasing amount of protein as cottonseed meal (equivalent to 0g, 10g, 25g, 50g, 75g, 100g, 125g urea). The objective was to examine the effects of increasing the amount of nitrogen supplement on the recycling of nitrogen to the rumen and on the body weight and composition of growing cattle. In addition, a sensitivity analysis was done on some of the parameters of the nitrogen recycling component to examine the possible mechanisms involved in the adaptation of cattle of different breeds on arid and semi-arid rangeland. The composition of the diet is given in Table 7.3.

7.5 Results and Discussion

Table 7.4 shows the model results of the effect of increasing the amount of urea on the recycling of nitrogen to the rumen. The roughage intake increased as the urea supplementation increased (Figure 7.5). This confirms experimental evidence that the DMI intake increases with increasing urea supplementation and that the rate of DMI intake decreases with increasing urea supplementation (Chase and Hibberd, 1987). The plasma urea nitrogen and the urinary urea nitrogen excretion rate decreased with the 0g, 10g and 25g urea nitrogen supplementation and increased with the 50g, 75g, 100g and 125 g urea nitrogen supplement (Figure 7.6). However, the rumen ammonia concentration decreased on the 0g, 10g, 25g, 50g, 75g and 100g urea supplementation quantities and increased on the 125g of urea supplement (Table 7.4). This is probably because as the amount of nitrogen supplement was increased, more nitrogen was required to support the exponential growth of the microbial population, which in turn, used these nitrogen sources to provide the host with a greater molar quantity of VFA (Table 7.4). The model also simulated the loss of empty body weight (initial empty body weight = 200kg) on the 0g, 10g and 25g urea nitrogen supplementation but the empty body weight and body composition increased as the urea nitrogen increased.

Table 7.3: Feed Composition and hydrolysis rates of roughage diet and urea supplement.

Feed	Feed Composition (g/kg)										Hydrolysis rate (/day)		
	Fd	Fu	Sc	Si	Ps	Pd	Pu	Am	Ld	Ash	Fd	Pd	Si
Roughage	462	176	160	0.0	2	81	32	0	8	50	0.950	0.0	0.84
Urea	0	0	0	0	0	0	0	549	0	0	0.0	0.0	0.0

Fd = Degradable fiber, Fu = Undegradable fiber, Sc = Soluble sugars and starch, Si = Insoluble starch and sugars, Ps = Soluble protein, Pd = Degradable protein, Pu = Undegradable protein, Am = Ammonia, Ash, Ld = Fat.

Recycling of nitrogen has been shown to be inversely related to dietary nitrogen content (Mugerwa and Conrad, 1971), although the data must be interpreted with caution because these experiments have been done with animals at maintenance or in positive energy balance. An animal losing weight (in negative energy balance) catabolizes fat and protein, which increases the plasma concentration of urea, which has been shown to be correlated with a decrease in the rate of urea recycling (Mugerwa and Conrad, 1971).

Table 7.4: Effects of increasing the quantity of protein supplementation (equivalent to 0g, 10g, 25g, 50g, 75g, 100g, 125g) on the recycling of nitrogen to the rumen and on body composition of growing cattle.

Days	20	20	20	20	20	20	20
Roughage DM (g/day)	4566.11	4660.46	4817.9	5055.21	5280.68	5455.15	5587.99
Supplement (gDM/day)	0	68	169	338	507	676	846
Starch and soluble sugars (g/day)	730.58	978.79	1032.29	1116.39	1198.1	1269.15	1331.84
NDF (g/day)	3378.92	3448.74	3565.25	3440.86	3907.7	4036.81	4135.11
Total N (g/day)	30.69	36.03	44.09	57.41	70.64	83.53	96.2
Recycling component:							
Plasma urea nitrogen (g/L)	0.1039	0.1001	0.0999	0.1036	0.1083	0.1131	0.1181
Concentration of rumen ammonia (g/L)	0.00160	0.00138	0.00119	0.00102	0.00094	0.00093	0.00100
Rate of nitrogen transport to rumen (kg/day)	0.0012	0.0011	0.0011	0.0012	0.0013	0.0014	0.0015
Rate of nitrogen recycling via saliva (kg/day)	0.0249	0.0239	0.0239	0.0249	0.0261	0.0274	0.0288
Urinary urea-N excretion rate (kg/day)	0.0219	0.0211	0.0212	0.0225	0.0241	0.0259	0.0278
Cdiff	0.1025	0.0990	0.0989	0.1028	0.1075	0.1123	0.1173
Saliva secretion rate (L/day)	382.17	382.17	382.17	382.17	382.17	382.17	382.17
Rate of nitrogen absorption from the rumen (g/day)	1.13	0.98	0.84	0.73	0.68	0.68	0.74
Concentration of microbal population in the rumen (g/L)	7.19	7.94	9.42	12.14	14.80	17.41	19.97
Body Composition:							
Empty Body Weight (kg)	194.66	196.55	198.88	202.35	205.63	208.72	211.67
Ash (kg)	7.12	7.15	7.16	7.17	7.18	7.18	7.18
Total protein (kg)	45.57	46.07	46.71	47.66	48.55	49.40	50.22
Lipid (kg)	20.29	20.39	20.52	20.73	20.97	21.19	21.38
Maintenance expenditure (MJ ME/W^0.75)	0.370	0.367	0.364	0.360	0.355	0.351	0.348
Absorption of VFA from the rumen (moles)	16.27	17.40	19.34	22.17	24.39	25.98	27.08

This has been shown in sheep (Ford and Milligan, 1970, Nolan and Leng, 1972), cattle (Mugerwa and Conrad, 1971) and in deer (Robbins *et al.*, 1974). Model results (Table 7.4) show that for an animal losing weight the rate of nitrogen recycling via the saliva and transport across the rumen wall decreases and increases slightly for animals above maintenance. Figure 7.7 shows that for animals losing weight the model suggests that just below maintenance the nitrogen recycling rate will continue to increase even when the rate of empty body weight change decreases and then increases. The increase in the rate of nitrogen recycling as the rate of empty body weight change increases may indicate the change in the utilization from fat to protein.

For animals losing weight, fat and protein is catabolised and the urinary urea excretion rate decreases, which in turn increases the plasma urea concentration. The model also suggests that the nitrogen recycling rate (divided by dietary nitrogen) is inversely related to the quantity of dietary nitrogen (Figure 7.8).

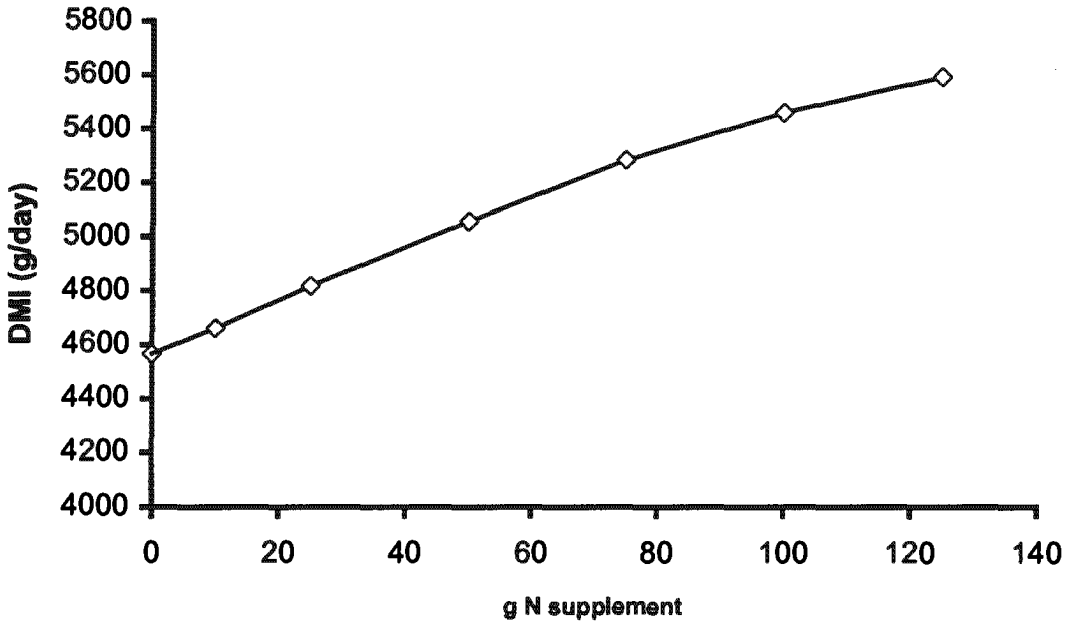


Figure 7.5: The effect of increasing the urea nitrogen supplementation on the intake of a basal roughage diet.

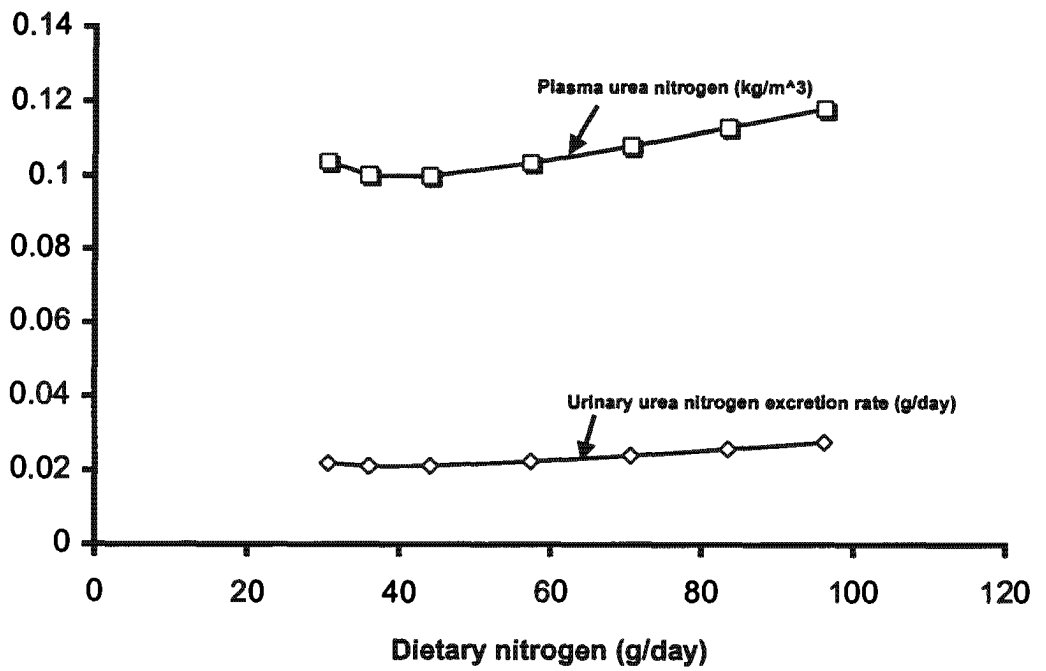


Figure 7.6: The effect of increasing the urea nitrogen supplementation on the plasma urea nitrogen concentration and the urinary urea excretion rate.

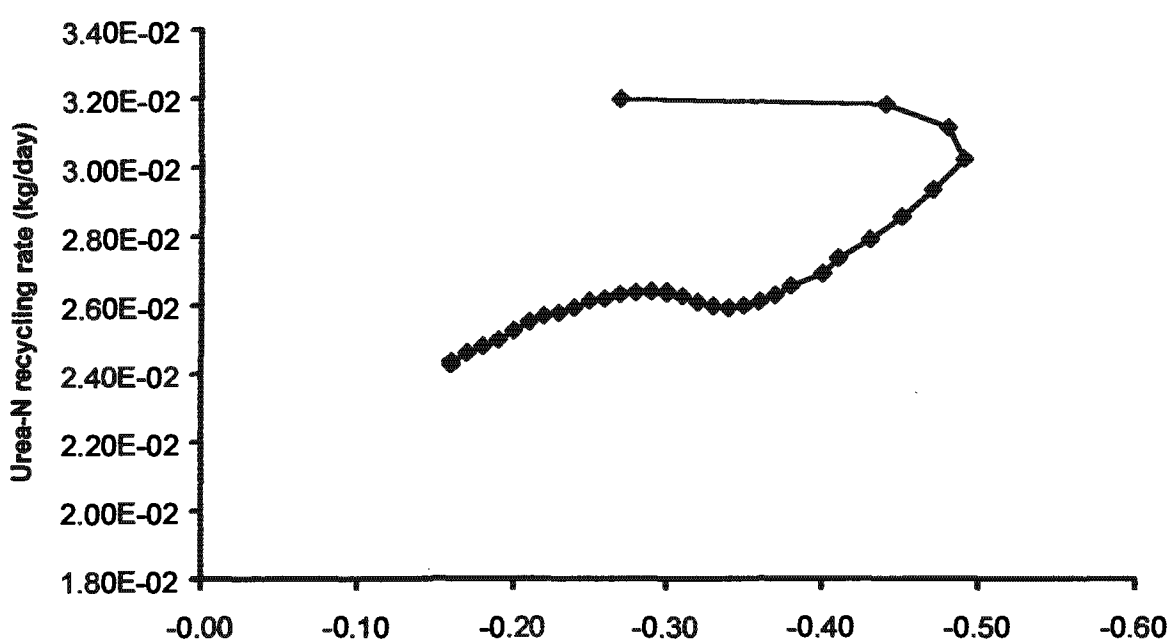


Figure 7.7: The effect of the rate of change of empty body weight (kg/day) on the urea-N recycling rate via the saliva and absorption across the rumen wall.

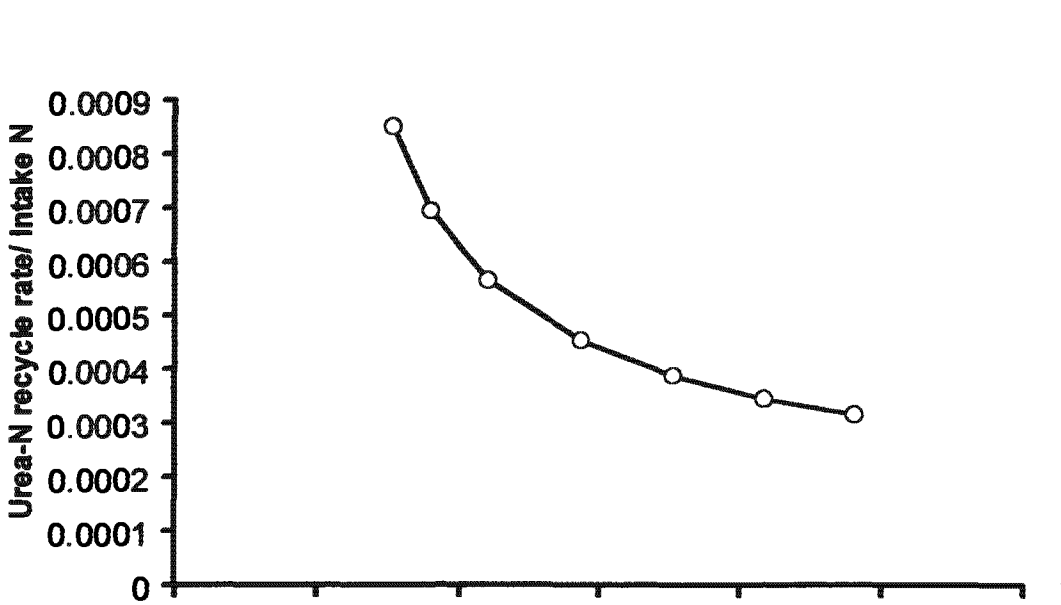
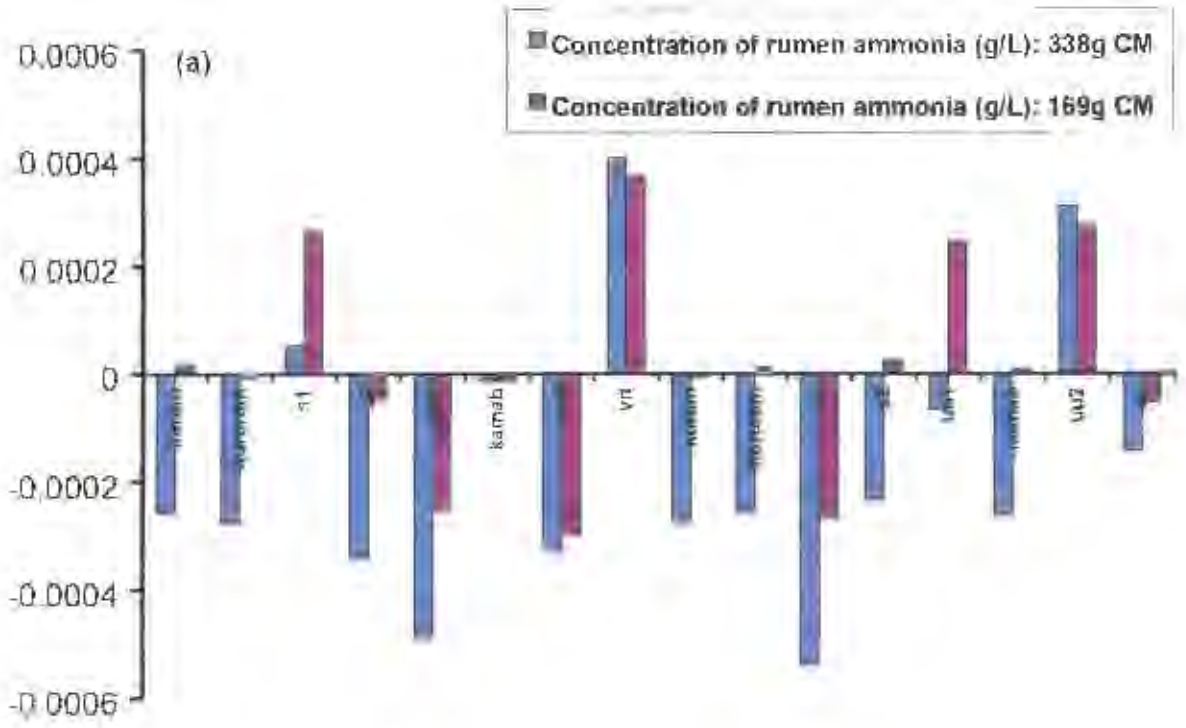


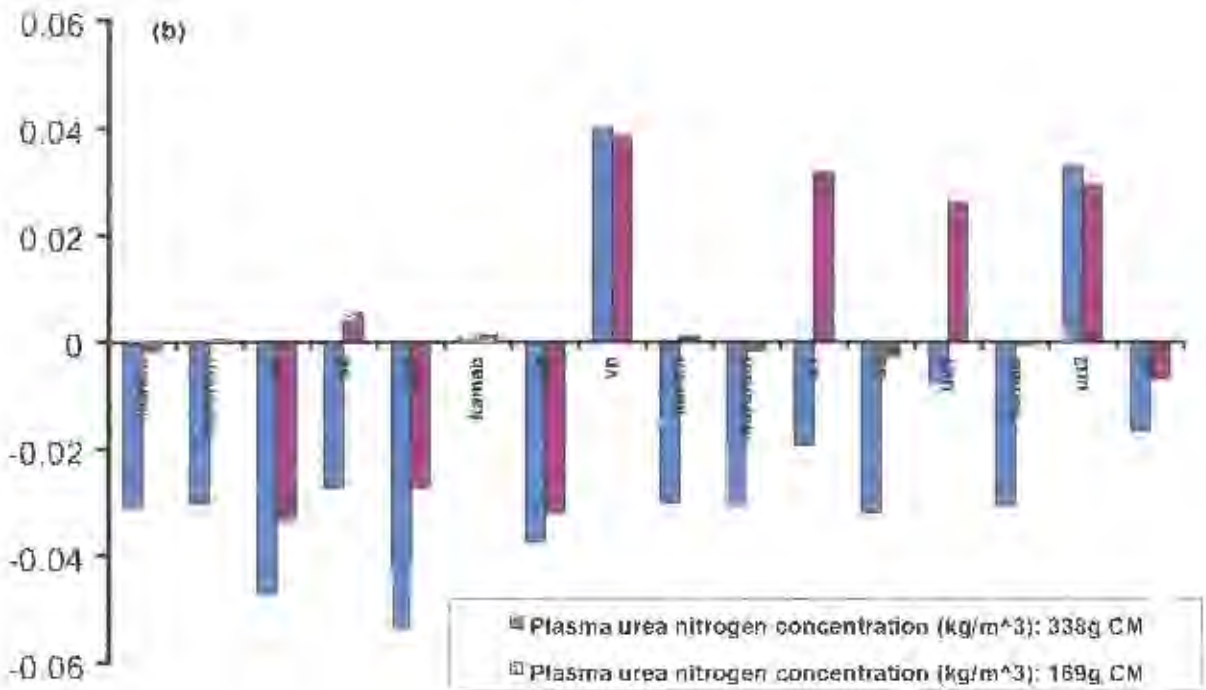
Figure 7.8: The effect of increasing the nitrogen supplementation on the urea-N recycle rate divided by the intake of N.

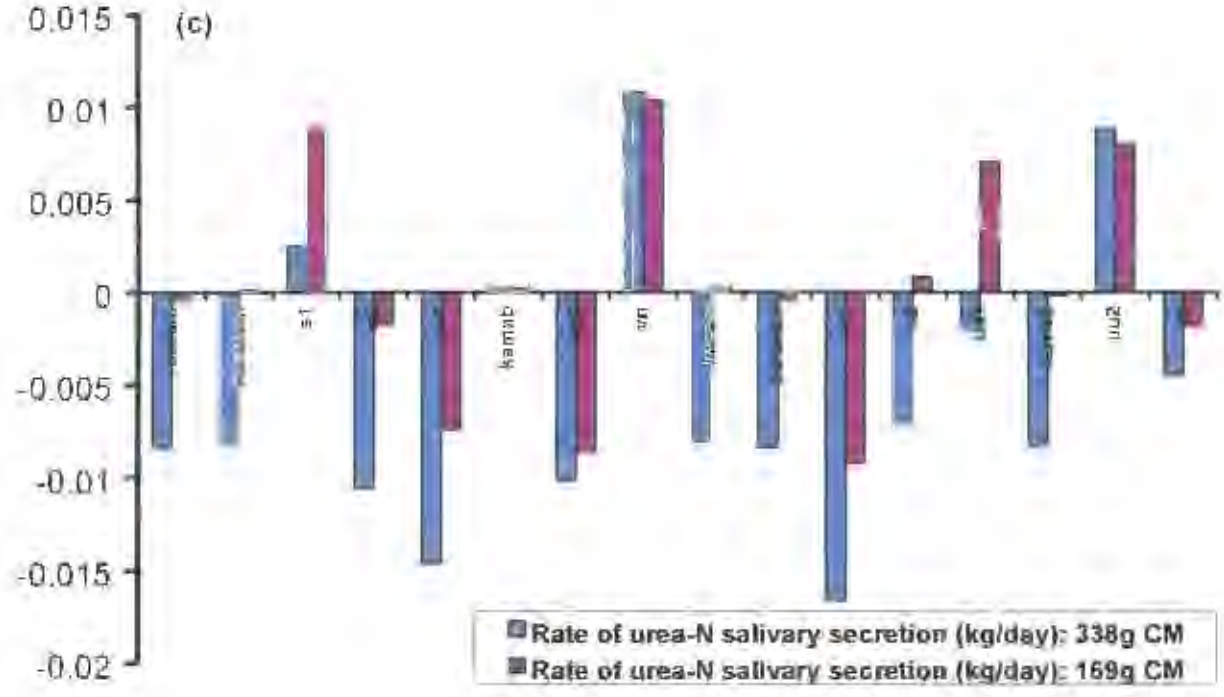
7.5.1 Sensitivity Analysis

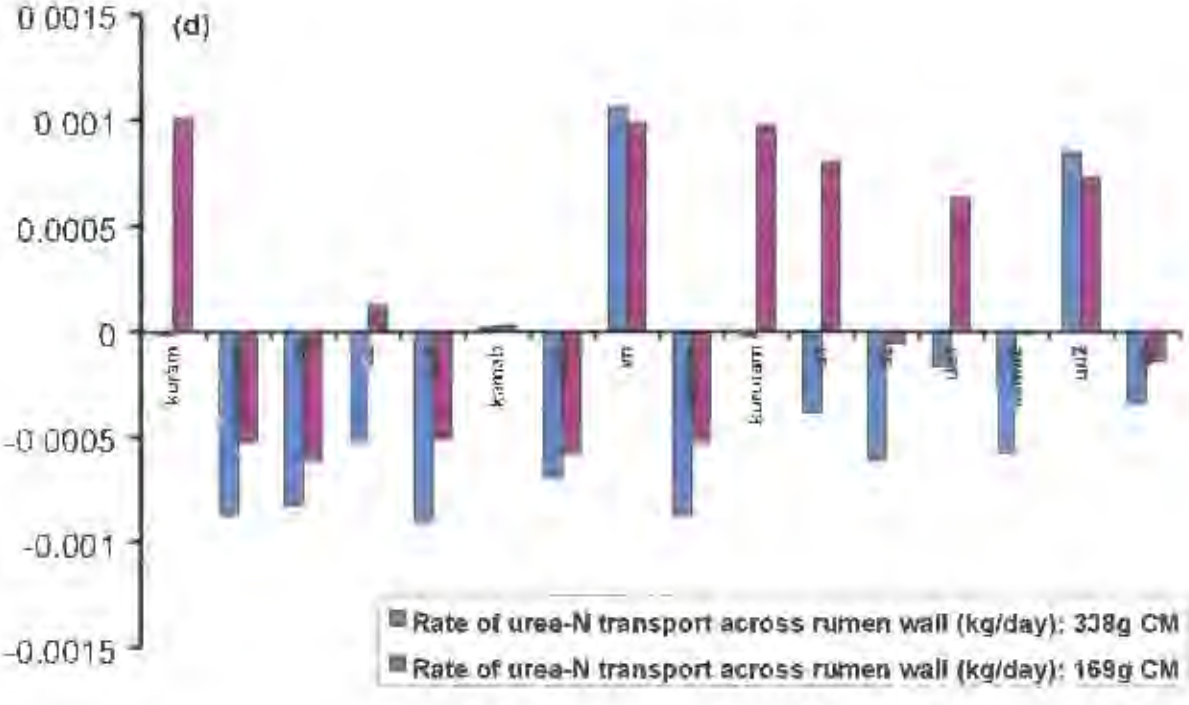
Eight parameters (v_{UrAm}, $K_{Ur,UrAm}$, s1, s2, Uu1, Uu2, k_{AmAb}, v_N), which are described in the text, were doubled and halved so that the effects on the concentration of ammonia in the rumen (cAm, $\frac{g}{L}$), the concentration of plasma urea nitrogen (PUN, $\frac{kg}{m^3}$), the rate of urea-N salivary secretion ($U_{Ur,UrSal}$, $\frac{kg}{day}$) and the rate of urea-N transport across the rumen wall ($U_{ur,uram}$, $\frac{kg}{day}$) could be examined. The metric used for the sensitivity analysis is the absolute difference of the variables after 20 days of simulation when no parameters were changed with the values of the variables when the parameters were changed after 20 days of simulation time. The model was run with two different urea-N quantities of cottonseed meal (25g, 50g) for 20 days. Figure 7.9 below shows the results of the sensitivity analysis: a positive sign (+) indicates the effect of doubling the parameter values and a negative sign (-) indicates the effect of halving the parameter values.

Hunter and Siebert (1985) reported in their experiments that rumen ammonia concentration was higher in Brahman that in Hereford steers when each was offered a low-N tropical hay in pens. The Brahman cattle represent a genotype with more adaptive traits and required a lower supplement input for maintenance and production when grazing low quality pastures. The aim of the sensitivity analysis is to examine the possible mechanisms that give rise to these adaptive traits of cattle. Figure 7.9a shows that increasing s1, v_N and decreasing Uu2 increased the concentration of rumen ammonia. These parameters are important because an increase in the concentration of rumen ammonia has been shown to be a good indicator of an increased adaptive animal fed low quality roughages (Hunter and Siebert, 1985; Hennessy *et al.*, 2000). Increasing s1 increased the concentration of nitrogen in mixed saliva and therefore the recycling of nitrogen to the rumen via the saliva (Figure 7.9c). The effect of increasing s1 was also seen to be greater with the 25g urea-N supplement than with the 50g urea-N supplement. Increasing the rate of gluconeogenesis (v_N) increased the concentration of plasma urea nitrogen and, in turn, increased the concentration of rumen ammonia. Decreasing Uu2 decreased the urinary urea excretion and therefore would increase the concentration of plasma urea and rumen ammonia concentration. An interesting result is that increasing k_{UrAm} one would expect an immediate increase in the concentration of rumen ammonia, however, this only occurred on the low N supplement. In addition, Figure 7.9d show that the rate of transport of nitrogen from PUN to the rumen only increased on the low N supplement and decreased slightly on the higher N supplement. This could be because of the sufficient supply of nitrogen for microorganisms on the higher N supplement.


(a)
Concentration of rumen ammonia (g/L): 338g CM
Concentration of rumen ammonia (g/L): 169g CM
0.0006
0.0004
0.0002
0
-0.0002
-0.0004
-0.0006


(b)
0.06
0.04
0.02
0
-0.02
-0.04
-0.06
Plasma urea nitrogen concentration (kg/m^3): 338g CM
Plasma urea nitrogen concentration (kg/m^3): 169g CM


(c)
0.015
0.01
0.005
0
-0.005
-0.01
-0.015
-0.02
Rate of urea-N salivary secretion (kg/day): 338g CM
Rate of urea-N salivary secretion (kg/day): 169g CM


(d)
0.0015
0.001
0.0005
0
-0.0005
-0.001
-0.0015
Rate of urea-N transport across rumen wall (kg/day): 338g CM
Rate of urea-N transport across rumen wall (kg/day): 169g CM

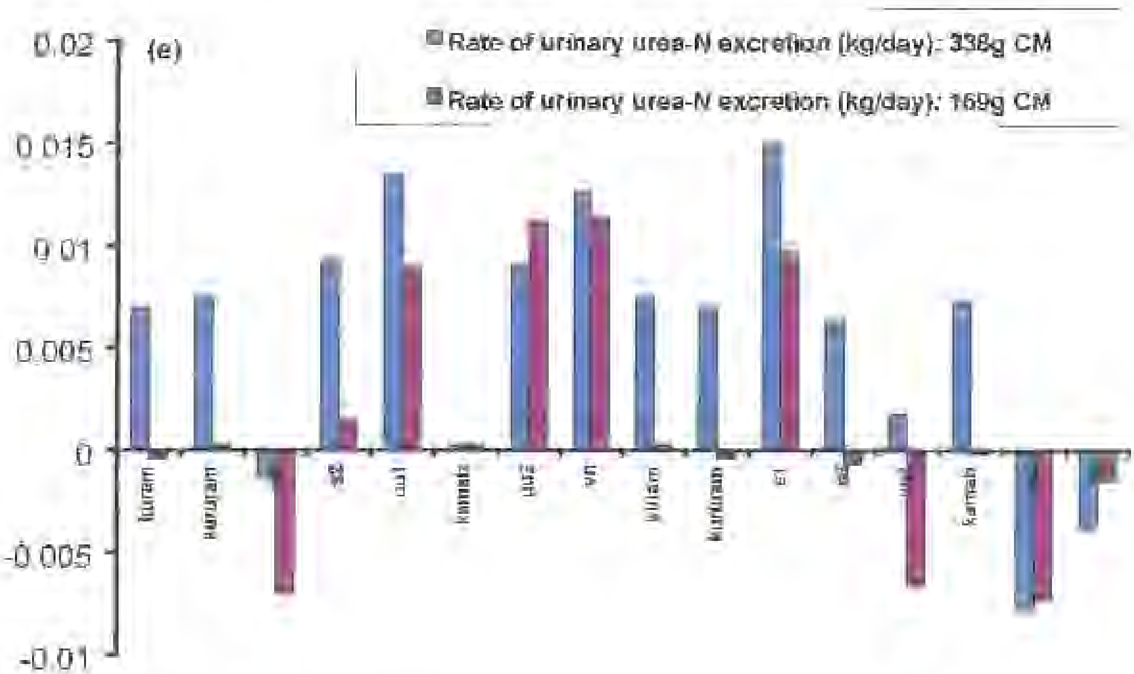


Figure 7.9: Effect of doubling and halving eight key parameters (v_{DeAm}, $K_{M(UrAm)}$, S1, S2, Uu1, Uu2, k_{AmAu}, v_0) on the (a) concentration of ammonia in the rumen (cAm, g/L), (b) plasma urea nitrogen concentration (PUN, kg/m^3), (c) rate of urea-N salivary secretion ($U_{Pl,Sal}$, kg/day), (d) rate of urea-N transport across the rumen wall ($U_{Pl,Ru}$, kg/day), and (e) rate of urinary urea-N excretion (U_{Ur}, kg/day).

7.6 Conclusions

The model suggests the possible mechanisms that contribute to the recycling of nitrogen to the rumen and, therefore, the mechanisms that contribute to the adaptation of cattle on arid and semi-arid rangelands. Although Hennessy *et al.* (2000) did not examine the extent of N recycling to the rumen, finding higher rumen ammonia concentration in the Brahman steers than in the Hereford steers or Brahman × Hereford steers suggests a greater nitrogen recycling ability in Brahman steers. For example, Hunter and Siebert (1985) found that *B. indicus* steers recycled more body nitrogen to the rumen than did *B. taurus* steers. They suggested that this ability may provide them with an advantage over *B. taurus* cattle grazing low quality subtropical/tropical pastures (Frisch and Vercoe, 1977). However, in spite of the apparent ability to maintain a high concentration of rumen ammonia, Brahman steers did not express production advantages over the Hereford or Brahman × Hereford steers confined to pens during their study.

The model suggests that factors that affect the concentration of urea-N in mixed saliva and the rate of gluconeogenesis from $N \rightarrow C_6$ are important for controlling the recycling of N to the rumen. In particular, model results suggest that factors affecting $s1$ increases nitrogen recycling without affecting animal production, whereas, factors affecting v_0 increases nitrogen recycling and animal production. In addition, the presence of a diverse population of microbes in the rumen enhances the potential to utilize N sources, which is an important component of ruminant N economy. The utilization is difficult to quantify experimentally (Nolan and Leng, 1972; Siddons *et al.*, 1985). However, using the model developed in this thesis may assist to quantify the utilization of N sources.

APPENDIX 7.1

MODEL EQUATIONS

Plasma urea nitrogen pool, PUN (kg plasma urea nitrogen per m^3)

Input:

$$P_{Ur,NC_6} = Y_{N,NC_6} \times U_{N,NC_6} \quad (7.1)$$

$$P_{Ur,AmUr} = k_{AmAb} \times Q_{Am} \quad (7.2)$$

Output:

$$U_{UEx} = 8.1123 \times e^{(12.114 \times PUN)} \quad (7.3)$$

$$U_{Ur,UrAm} = \frac{V_{UrAm} \times C_{diff}}{1 + \frac{K_{Ur,UrAm}}{C_{diff}}} \quad (7.4)$$

$$\text{where } C_{diff} = PUN - 0.82 \times C_{am} \quad (7.5)$$

$$U_{Ur,UrSal} = SalN \times Salday \quad (7.6)$$

$$\text{where } SalN = 0.7083 \times PUN^{*} - 0.8328 \quad (7.7)$$

$$Salday = 0.332 \times 1000 \times (cFd1 + cFd2) + 116.9 \quad (7.8)$$

Differential Equation:

$$\frac{dPUN}{dt} = \underbrace{P_{Ur,NC_6}}_{\text{gluconeogensis}} + \underbrace{P_{Ur,AmUr}}_{\text{diffusion of ammonia across rumen wall}}$$

$$- \underbrace{U_{UBx}}_{\text{excretion of urea}} - \underbrace{U_{Ur,UrAm}}_{\text{transport of urea across rumen wall}}$$

$$- \underbrace{U_{Ur,UrSal}}_{\text{transport of urea via saliva}} \quad (7.9)$$

Ammonia pool, Q_{Am} (g).

Concentration:

$$C_{Am} = \frac{Q_{Am}}{Rvol} \quad (7.10)$$

Input:

$$P_{Am,InAm} = D_{Am1} + D_{Am2} + D_{Am3} \quad (7.11)$$

$$P_{Am,UrAm} = Y_{Am,UrAm} \times U_{Ur,UrAm} \quad (7.12)$$

$$P_{Am,PsAm} = Y_{Am,PsAm} \times U_{Ps,PsAm} + Y_{Am,SalAm} \times U_{Ur,UrSal} \quad (7.13)$$

Output:

$$U_{Am,PsMi} = R_{Am,PsMi} \times U_{Ps,PsMi} \tag{7.14}$$

$$U_{Am,AmMi} = \left[v_{AmMi} \times Q_{Mi}\right] / \left[1 + \left(\frac{M_{Am,AmMi}}{C_{Am}}\right) + \left(\frac{C_{Ps}}{J_{Ps,AmMi}}\right) + \left(\frac{M_{Sc,AmMi}}{C_{Sc}}\right) + \left(\frac{M_{Ld,AmMi}}{C_{Ld}}\right)\right] \tag{7.15}$$

$$U_{Am,AmAb} = k_{AmAb} \times Q_{Am} \tag{7.16}$$

$$U_{Am,AmEx} = k_{FlEx} \times Q_{Am} \tag{7.17}$$

Differential Equation:

$$\frac{dQ_{Am}}{dt} = \underbrace{P_{Am,UnAm}}_{\text{diet input}} + \underbrace{P_{Am,UrAm}}_{\text{transport of urea}} - \underbrace{P_{Am,PsAm}}_{\text{fermentation of Ps}}$$

$$- \underbrace{U_{Am,PsMi}}_{\text{microbial growth}} - \underbrace{U_{Am,AmMi}}_{\text{microbial growth}} - \underbrace{U_{Am,AmAb}}_{\text{absorption}}$$

$$- \underbrace{U_{Am,AmEx}}_{\text{outflow with fluid}} \tag{7.18}$$

Chapter 8

General Discussion

In ruminants, the profile of nutrients available for absorption generally differs substantially from that present in the ingested feed. The type and amount of feed nutrients absorbed can significantly affect the amount and composition of liveweight change. Thus, there is an obvious need to understand the mechanisms responsible for the transformation of ingested to absorbed nutrients (Gill and Oldham, 1993). Whilst research on various aspects of this transformation has yielded valuable information, the concentration of research on individual components, rather than on the integration of knowledge, has resulted in insufficient information on many important mechanisms which link the individual components, and thereby hampers adequate predictions of the supply of nutrients to carcass components. France *et al.* (1987) concluded that their intermediary metabolism model did not predict body composition in all situations and suggested that the model be linked with a model of rumen digestion to improve the estimation of nutrient inputs. This was successfully done (FRANCE model) in chapter 6. The FRANCE model exhibits compensatory growth and predicts the body weight and composition of a steer during and following a period of nutritional restriction. However, a few modifications were made to the FRANCE model to improve its behaviour and predictions of body weight and composition. This resulted in the RUMET model which contributes to our understanding and improved predictions of body weight and composition during and following a period of undernutrition.

Briefly stated, several ideas have been proposed to model ruminant digestion and metabolism in order to understand the mechanisms of adaptation. These include the linking of a modified rumen and intermediary metabolism model, adding an intake component to predict intake, including a delay in the digestion of forage due to the attachment of microorganisms to food particles, and explicitly representing the effect of the size of the small intestine and liver on total basal metabolism and representing the dependence of liver and small intestine size on feeding level. In addition, a mathematical model based on a chemostat-type model of competition between functional groups of microbes in the rumen was developed and the extent of nitrogen recycling via the saliva and the rumen wall was examined.

During nutritional restriction, the RUMET model shows that tissues are differentially mobilized and that the greatest losses occur in the liver and small intestine, which has been shown in chapter 6 to agree with the results of many experiments. Model predictions support the following two hypotheses: firstly, the amount of nutrients processed by these tissues are decreased during nutritional restriction and, secondly, these tissues are extremely active metabolically and have a high maintenance requirement. The visceral organs exhibit quick responses to changes in nutrition whereas the peripheral tissues (for example the muscle) shows a lag in its response to undernutrition. During nutritional restriction the rumen volume does not decrease. At the end of the restricted period the animal has a large rumen relative to its body size. At this point the highly active visceral tissues are small and therefore the maintenance requirement is low. As a consequence, when more dry matter intake is available, the feed intake relative to body size increases dramatically. Also, the enhanced food intake in relation to size and the reduced maintenance requirements will imply that proportionately more nutrients will be available for tissue synthesis. In practice, however, this may not be the case, food intake will slowly increase and not as suddenly as the model predicts. This sudden increase in the rate of growth may also have an affect on the body composition at the end of the realimentation. The model thus increases our understanding of the mechanisms involved in compensatory growth. An important need for further investigation is whether the model can simulate the body composition after different degrees of nutritional restriction and also to examine the effects of different rates of weight-loss and weight-gain.

There is growing information on the co-ordination of nutrient use by the peripheral tissues (Eisemann, 1994). The co-ordination has been shown to involve key metabolic hormones and changes in tissue sensitivities and nutrient partitioning during growth can be manipulated by altering homeostatic and homeoretic mechanisms. With the increased focus on the mechanisms involved, there will be a need to assimilate the data using mathematical models.

The models developed in this thesis have focused on ruminants (steers) on arid and semi-arid rangeland. This thesis arose from the need to integrate the mechanisms of adaptation within a quantitative framework. However, the models developed can be modified to include, for example, animals of different breeds and stage of maturity. This will require new data sets to parameterise and evaluate the models, which is still one of the greatest constraints in model building. The models, in their present form,

may be adapted to different situations, however, it may be best to develop simplified models that attempt to incorporate the proposed mechanisms and their relatedness.

Applications of the models developed in this thesis may include examining the effects of environmental changes (for example, changes in vegetation) on the survival of different wild ruminants. Changes in diet composition at different times of the year have different effects on animals of different sizes. A model examining these effects may be combined with a routine that predicts the changes in outflow rate of digesta with body size (Illius and Gordon, 1991).

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