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Environmental stochasticity and density dependence in animal population models

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Abstract

Biological management of populations plays an indispensable role in all areas of population biology. In deciding between possible management options, one of the most important pieces of information required by population managers is the likely population status under possible management actions. Population dynamic models are the basic tool used in deriving this information. These models elucidate the complex processes underlying the population dynamics, and address the possible consequences/merits of management actions. These models are needed to guide the population towards desired/chosen management goals, and therefore allow managers to make informed decisions between alternative management actions.

The reliability that can be placed on inferences drawn from a model about the fate of a population is undoubtedly dependent on how realistically the model represents the dynamic process of the population. The realistic representation of population characteristics in models has proved to be somewhat of a thorn in the side of population biologists. This thesis focuses in particular on ways to represent environmental stochasticity and density dependence in population models.

Various approaches that are used in building environmental stochasticity into population models are reviewed. The most common approach represents the environmental variation by changes to demographic parameters that are assumed to follow a simple statistical distribution. For this purpose, a distribution is often selected on the basis of expert opinion, previous practice, and convenience. This thesis assesses the effect of this subjective choice of distribution on the model predictions, and develops some objective criteria for that selection based on ecological and statistical acceptability. The more commonly used distributions are compared as to their suitability, and some recommendations are made.

Density dependence is usually represented in population models by specifying one or more of the vital rates as a function of population density. For a number of reasons, a population-specific function cannot usually be selected based on data. The thesis develops some ecologically-motivated criteria for identifying possible function(s) that

could be used for a given population by matching functional properties to population characteristics when they are known. It also identifies a series of properties that should be present in a general function which could be suitable for modelling a population when relevant population characteristics are unknown. The suitability of functions that are commonly chosen for such purposes is assessed on this basis.

I also evaluate the effect of the choice of a function on the resulting population trajectories. The case where the density dependence of one demographic rate is influenced by the density dependence of another is considered in some detail, as in some situations it can be modelled with little information in a relatively function-insensitive way.

The findings of this research will help in embedding characteristics of animal populations into population dynamics models more realistically. Even though the findings are presented in the context of slow-growing long-lived animal populations, they are more generally applicable in all areas of biological management.

Preface

A population model is a mathematical expression that exhibits the unknown biological processes in nature in a simple form such that the underlying dynamics are easily understandable. Any 'good' model should be simple to understand, realistic enough to represent the dynamics of the population, general or flexible enough to cover a range of possibilities in nature, and sufficiently applicable so that it can be used with the available information to produce the required information as outputs.

The aim of this thesis is to explore the possibility of expressing some of the important population characteristics in models in a more realistic way. I was motivated to investigate this when I first noticed an unconventional distribution being used to represent the environmental stochasticity in a model proposed for the New Zealand Sea Lions. Environmental stochasticity and density dependence are two characteristics commonly required in population models. This research reviews how these two characteristics can be incorporated into population dynamics models, and evaluates the relative merits of alternative approaches. The evaluation is based on the success of different approaches in elucidating the underlying biological mechanisms and in representing them realistically in the model. A few drawbacks in some of the present approaches have been identified and suggestions are made for improvements. The findings are summarized in the last chapter, and some recommendations are made for future research on the topics covered.

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Thanks to the Department of Conservation for the opportunity of working with New Zealand Sea Lion population modelling, which motivated me to research some of the areas covered by this thesis.

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Table of contents

Abstract iii

Preface v

Table of Contents vii

List of Tables xiii

List of Figures xiv

1 Introduction 1

1.1 Population dynamics 1

1.1.1 Population modelling 2

1.1.2 Brief history of population modelling 3

1.1.3 Nature of population models 3

1.1.4 Approaches for 'building in reality' into population models 4

1.2 Broad research objectives 5

1.3 Significance of study 5

1.4 Research approach 6

1.5 The structure of the thesis 7

2 Motivating Example 8

2.1 Introduction 8

2.2 New Zealand Sea Lion (NZSL) 9

2.3 Fishing related mortality of New Zealand Sea Lions 10

2.3.1 Maximum Allowable Fishing Related Mortality (MALFiRM and Potential Biological Removal (PBR) 11

2.4 Research in progress towards developing a species specific model 13

2.4.1 Population models proposed 13

2.4.2 Ongoing research on estimating demographic parameters 14

2.5 A basic population dynamics model for the NZSL 16

3 A General Overview of Animal Population Modelling 21

3.1 Introduction 21

3.1.1 Importance of modelling and aims of modelling 21

3.1.2 The flow of the chapter 22

3.2 Applications of modelling 23
3.2.1 Harvest management models 24
3.2.2 Pest and disease control models 25
3.2.3 Conservation management models 26
3.3 Different types of population models 27
3.3.1 Continuous-time models and discrete-time models 28
3.3.2 Population based models and individual based models 30
3.3.3 Single-species models and multi-species models 32
3.3.4 Count based models and demographic models 33
3.3.5 Unstructured models and structured models 33
3.3.6 Metapopulation models and multistate models 38
3.4 Characteristics of populations represented in population models 41
3.4.1 Stochasticity 43
3.4.2 Density dependence 45
3.4.2.1 Representing DD in population models 47
3.4.3 Other characteristics 48
3.4.3.1 Human activities 48
3.4.3.2 Catastrophic events 49
3.4.3.3 Heterogeneity in population 49
3.4.3.4 Senescence 50
3.4.3.5 Correlation 53
3.4.4 Remark on representing population characteristics 54
3.5 Model assessment and interpretation 56
3.5.1. Model verification and Validation 57
3.5.2. Uncertainty analysis 59
3.5.3. Sensitivity analysis 63

4 Environmental Stochasticity 67
4.1 Introduction 67
4.1.1 Importance of environmental stochasticity in population models 67
4.1.2 Stochasticity as distinct from uncertainty 68
4.1.3 Importance of modelling stochasticity beyond mean and variance 70
4.1.4 Correlated and iid stochastic vital rates 70
4.1.5 Properties to consider when selecting a distribution to

represent environmental variation 72
4.1.6 Maximum possible variability for a given range 74
4.1.6.1 Maximum possible variability under any finite distribution 75
4.1.6.2 Maximum possible variability under any finite unimodal distribution 76
4.2 Statistical distributions in use in representing environmental variation 77
4.2.1 Normal distribution 78
4.2.2 Lognormal distribution 80
4.2.3 Beta distribution 82
4.2.4 Logitnormal distribution 85
4.2.5 Probitnormal distribution 86
4.2.6 Lognormalpower distribution 87
4.3 Comparing suitability of distributions 89
4.3.1 Ability to specify parameters using available information 90
4.3.2 Generating vital rates from the distributions 92
4.3.3 Ability to use all sensible combinations of mean and sd 92
4.3.4 Reliability of parameter specification 94
4.3.5 Ability to simulate variability as intended 94
4.3.6 Suitability of Delta method approximation in specifying Parameters 96
4.3.7 Shape of the distribution 98
4.4 Summary 103

5 Effect of Stochastic Distribution on Population Predictions 104
5.1. Introduction 104
5.2. The population model and the Monte Carlo simulation 105
5.3. Effect of distribution on stochastic growth rate 110
5.4. Effect of distribution on extinction risk 110
5.5. Effect of distribution on elasticity of growth rate 114
5.6. Discussion 117
5.6.1 Other possible approaches in modelling stochasticity 117
5.6.2 Choice of distribution 118
5.6.3 Parameter estimation and speed of implementing

population models 119
5.6.4 Reliability of beta random number generators 120
5.6.5 Flexibility and suitability of shapes 121
5.6.6 Possibility of explaining simulation results from shapes of distributions 123
5.6.7 Modelling vital rates over other intervals 127
5.6.8 Possibility of generating correlated vital rates 128
5.6.9 Suitability of variance to represent variability of vital rates in population models 129
5.6.10 Relationship between U shaped distributions and risk of extinction 132
5.7. Conclusion 134

6 Density Dependence 136
6.1 Introduction 136
6.1.1 Introduce the chapter 136
6.1.2 Introduce density dependence 136
6.1.3 Representing density dependence in population models 138
6.1.4 Assumptions for the rest of the chapter 139
6.1.5 Graphically representing density dependent data 141
6.1.6 The flow of the chapter 143
6.2 Desirable properties of a DD function 144
6.2.1 Shape of the DD function 146
6.2.2 Nature of density dependent competition (Contest vs Scramble) 147
6.2.2.1 Method 1 150
6.2.2.2 Method 2 151
6.2.2.3 Method 3 152
6.2.2.4 Interrelationship between methods 2 and 3 152
6.2.3 Population stability 153
6.2.3.(a) Stability at multiple time period 156
6.2.3.(b) Relationship of stability to other characteristics 156
6.2.4 Asymmetric population growth 157
6.2.5 Summary of properties 159

7 Properties of Commonly Used Density Dependent Functions **161**

7.1 Introduction 161

7.2 Logistic function 162

7.3 θ–logistic function 166

7.4 Ricker function 171

7.5 θ–Ricker function 175

7.6 Haldane function 189

7.7 Beverton and Holt function 182

7.8 Maynard Smith and Slatkin function 186

7.9 Hassell function 191

7.10 Usher function 194

7.11 Linear transformation of functions 198

7.12 Summary of functions 200

8 Effect of Density Dependence Modelling on Population Predictions **204**

8.1 Introduction 204

8.1.1 Objective of the chapter 204

8.1.2 Short list of functions 206

8.1.3 The approach 207

8.1.4 The flow of the chapter 208

8.2 Generating same level of density dependence from various functions 208

8.3 Effect of DD function on model predictions 212

8.3.1 The population model and the simulation 212

8.3.2 Simulation results 215

8.3.3 Discussion on comparing functions. 223

8.4 Interactive density dependent (IDD) model 231

8.4.1 Introduction to IDD model 231

8.4.2 Interactive Density Dependent model 233

8.4.3 Calculation parameters for IDD models 235

8.4.4 Simulation 238

8.4.5 Simulation results 240

8.4.6 Discussion on IDD model 244

8.5 Conclusion 252

9 Conclusion and Further Research **254**

9.1 Environmental stochasticity 255

9.2 Density dependence 266

9.3 IDD model 279

9.4 Summary 281

Bibliography **284**

Appendices

Appendix 1 Maximum possible variance that can exist in a finite interval under any distribution 304

Appendix 2 Probability density functions for distributions used for Environmental stochasticity 306

Appendix 3 Parameters for statistical distributions in terms of their mean and standard deviation 309

Appendix 4A Intended and realised variability from beta distribution 314

Appendix 4B Intended and realised variability from logitnormal distribution 314

Appendix 4C Intended and realised variability from lognormalpower distribution 315

Appendix 4D Intended and realised variability from probitnormal distribution 316

Appendix 5 Relationship between mean and sd for differently shaped beta distributions 317

Appendix 6A Interrelationship between two methods used to investigate the stability at equilibrium population density 322

Appendix 6B Stability properties at multiple time steps 327

Appendix 7 Population stability and asymmetric growth in demographic models 324

Appendix 8 Population level corresponds to maximum productivity under the Richer function 326

Appendix 9 Deriving the shape of the MS&S function from its functional form 328

Appendix 10 Form of competition represented by MS&S function 329

Appendix 11 Curves from DD function with same strength density dependence 330

Appendix 12 Estimating parameters for DD functions for IDD models 334

Appendix 13 Smallest level of vital rate in IDD models 344

List of Tables

Table 4.1 Parameter specification for stochastic distributions using mean and variance, and variates generation from those distributions 91

Table 4.2 Summary of shapes of stochastic distributions 100

Table 4.3 Summary of characteristics of statistical distributions relevant to stochastic variation 102

Table 5.1 Stage specific mean survival rates effective in the simulation at three Levels of C 109

Table 6.1 Evaluating neighbourhood stability at equilibrium population level 155

Table 6.2 Summary of methods to evaluate properties of DD functions. 160

Table 7.1 Summary of properties of DD functions 203

Table 8.1 Parameter values used for the MS&S function at the lowest DD strength of simulation 213

List of Figures

Figure 2.1	The location of Aucklands Islands	10
Figure 2.2	Life cycle diagram for the basic population model for the NZSL	18
Figure 3.1	Prebreeding and postbreeding models.	30
Figure 4.1	Probability density functions of stochastic distributions	85
Figure 4.2	Comparison of intended and achieved variability from statistical distributions	95
Figure 4.3	Shapes of distributions estimated from delta method and numerical integration	96
Figure 4.4	Reliability of the delta method approximation for specifying parameters of distributions	97
Figure 4.5	Change of shapes of stochastic distributions with *m* and *s*	101
Figure 4.6	Relationship between mean and standard deviation for differently shaped standard beta distributions	103
Figure 5.1	Life cycle diagram for the population model used in the case study	105
Figure 5.2	Effect of C on the survival rates	108
Figure 5.3A	Stochastic population growth rates predicted by different distributions showing effect of the mean	111
Figure 5.3B	Stochastic population growth rates predicted by different distributions showing effect of variability	112
Figure 5.4	Probabilities of quasiextinction under different distributions	113
Figure 5.5	Elasticity of stochastic growth rate with respect to survival rate	116
Figure 5.6	Comparison of beta generators	122
Figure 5.7	Probability density functions of distributions for *m* and *s* combinations used in simulation	126
Figure 5.8	Beta probability density functions representing variability by θ	131
Figure 5.9	Probability of quasiextinction curves for different mean levels	133
Figure 6.1	Different ways of graphically representing density	143
Figure 6.2	A sketch of N_{t+1} vs N_t relationship for contest and scramble competitions	151
Figure 7.1	Density dependence under the logistic function	163
Figure 7.2	Density dependence under the θ-logistic function	167

Figure 7.3	Density dependence under the Ricker function	172
Figure 7.4	Density dependence under the θ-Ricker function	176
Figure 7.5	Density dependence under the Haldane function	180
Figure 7.6	Density dependence under the B&H function	183
Figure 7.7	Density dependence under the MS&S function	187
Figure 7.8	Density dependence under the Hassell function	192
Figure 7.9	Density dependence under the Usher function	195
Figure 8.1	Same DD function with different strengths, and different DD functions with same strength	210
Figure 8.2	Trajectories under the DDS model	216
Figure 8.3	Trajectories under the DDS model scaled by K	216
Figure 8.4	The DD functions used for each vital rate with or low and high DD levels	218
Figure 8.5	Trajectories from DDR and DDSR models	220
Figure 8.6	Declining trajectories under DD functions	222
Figure 8.7	The sequence of re-calculating DDS and DDR curves in IDD models	235
Figure 8.8	Population trajectories from DD functions under the IDD-DDSR model	241
Figure 8.9	Variation of vital rates realized from IDD models with time	242
Figure 8.10	Population trajectory from the logistic function under the IDD-DDSR model	242
Figure 8.11	Population trajectories from DDS and DDR models	244
Figure A.5.1	Relationship between the mean and standard deviation for differently shaped standard beta distributions	321
Figure A.11.1	Different DD functions with same strength	330

1 Introduction

1.1 Population Dynamics

The 'dynamics' of animal populations - the ways in which their numbers grow and shrink as time goes by - depends on a number of factors. They include both internal and external factors such as the availability of food, water and shelter, reproduction, mortality, inward and outward movement. This thesis deals with population dynamics by addressing the influence of internal and external factors on the dynamic process.

The factors that impact upon a population can be classified under inputs and outputs of a dynamic model. The size of a population depends on the balance between the inputs and outputs. In a simple scenario, the two basic inputs are birth (natality) and immigration, and outputs are death (mortality) and emigration. The population size may fluctuate with time depending on the effects and the balance of inputs and outputs to the equation. Mathematical expressions that elucidate the dynamics of populations using such inputs and outputs, and as realistically and simple as possible, are termed population models. In this study of population dynamics, the principle focus is on the population modelling.

Detailed field experiments can, within practical limitations, unveil the status and trends of a system, but they cannot elucidate the underlying biological process of a system as done by population models. Building a 'good' model of a biological system is the one sure way of understanding the workings of the system, or pinpointing areas

of ignorance (Starfield et al. 1981). Models provide an explicit description of one's conceptualization of a process for system, and the status of the population.

1.1.1 Population modelling

This research reviews different approaches of deriving population models, aimed at elucidating a more effective approach for understanding the biological process underlying population dynamics. Hence, a brief introduction to population modelling is timely at this point to enhance the readers' understanding of the forthcoming sections.

Population modelling is used in the field of population dynamics in attempting to resolve such issues as: How could population dynamics influence the viability of a population? Are there any factors that regulate population stability? How likely is a population of a vulnerable species to survive without extinction, say for another 100 years? How can we predict the fate of a threatened population? Is it more likely to recover or go extinct due to natural dynamics? What corrective measures will be most appropriate to save the population if it is likely to go extinct in the near future? These are some biologically and economically important issues that the studies of population modelling would address.

In practice, animal population modelling is employed for pest control, harvest management, and conservation activities. Wade (1999) identifies three purposes of modelling in conservation; to estimate the possible fate(s) of the population under current status (such as the probability of extinction of a vulnerable population), to explore the possible consequences of future management options, and to estimate the depletion level of a population that has experienced anthropogenic mortality. In this research population modelling is examined in the light of conservation of marine animal species. However, the study is not confined only to these species, and therefore its outcomes are applicable to a wider range of modelling applications.

1.1.2 Brief History of Population Modelling

It was Kepler (1571 – 1630) who first recognized the link between biological phenomena and mathematical relationships (Edelstein-Keshet 1988). Since then mathematical models have been developed with the view of understanding the biological phenomena. Verhulst's work in 1830s may be viewed as the first formal study of dynamics of populations of large mammals (Smith and Fowler 1981, p1). About a century ago the earliest fisheries models were built, and marine mammal population models followed soon (Chapman 1981). Basic theoretical ideas of population modelling were also developed during the same period. The earliest models were of a descriptive nature; more recently models have been built to understand the driving forces underlying the biological mechanism, rather than just to describe their behavior.

1.1.3 Nature of Population Models

Population models are quite diverse. They may range from simple to very complex[1] ones. Furthermore, population models may vary widely in the way they are constructed (e.g., count-based or demographic, unstructured or structured, single populations or metapopulations, etc[2]), depending on their intended use and availability of data. Not all population models represent the characteristics[3] of populations to the same extent or in the same manner. In addition, available knowledge about population characteristics is often inaccurate (biased) and/or imprecise (uncertain). Therefore all these issues need to be taken into account in constructing population models and in making predictions from such models for species management.

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[1] Simple population models have only an empirical fit of a mathematical relationship to observations. Therefore, they can be used for projecting the population to future without a clear understanding the factors behind its constitutions. Complex population models have more realistic model structures to elucidate the 'real phenomena' and therefore can be used to explore the natural processes of the population dynamics and hence more useful for the purposes of management decision making.

[2] These terms are detailed in Chapter 3

[3] Density dependence, dispersal, stochasticity, and senescence are certain common characteristics of populations.

The reliability of predictions from a particular model depends on how well the model can portray the dynamics of the real population. In order to derive a realistic population model the characteristics of natural processes need to be built in. It is clear that building the natural processes exactly as it operates in nature is very complex, if not impossible. Further, an objective of modelling is to present these processes in a simplified way. However, a good population model should represent the features of natural processes at least to the level demanded by the objectives that the model is used for.

Mathematical models provide perhaps the best techniques for exploring the consequences of conceptualizations (Horwood 1981). Levins (1968) identified generality, precision, and realism as three essential features of models constructed to represent natural systems. Ayala et al. (1973) suggested a model should describe the implicit population mechanism adequately (i.e., it is realistic), while having as few parameters as possible (i.e., simple and parsimonious), with each parameter having a plausible biological interpretation (i.e., applicable). A 'good' model should simultaneously maximize all the above features. This criterion can be used in evaluating the successfulness/reliability of a model or comparing various approaches taken in modelling (Starfield et al. 1981). Levins (1968) pointed out the difficulty (if not impossibility) of obtaining strength in all these attributes in one model. For example, in an attempt to enhance the realism of a model one might increase the number of model parameters, inevitably increasing its complexity (i.e., reducing the simplicity). This would also compromise the practicality of the model because it is unlikely that we have enough data to estimate large number of parameters, especially if the model refers to an endangered species (Ralls et al. 2002). Therefore, one or more of these features has to be sacrificed to improve the others, hence each model possesses some of these characteristics more than others.

1.1.4 Approaches for 'building reality' into population models

A major part of 'building in reality' involves representing any known information about population characteristics (which are usually contained in raw data) in

population models as precisely as possible. This includes two major groups of work; building the population model (including its components that describe each characteristic), and estimating its parameters. There are different approaches of doing this. The suitability of one approach over another depends on how well the dynamics of the real population are represented in the model. It is not clear on how best to represent those important characteristics in population models so that models are realistic and applicable in managing populations. This research aims to investigate how population characteristics can be realistically represented in population models.

1.2 Broad Research Objectives

This research reviews how certain characteristics of population, such as stochasticity and density dependence, can be built into population dynamics models. Then the relative merits of different approaches are evaluated. The evaluation is based on the successfulness of different approaches in elucidating underlying biological mechanisms and representing them realistically in the model. In particular this study examines the relative merits of those approaches in the context of marine mammal population models, and aims to recommend more appropriate approaches in doing so for a given population by identifying some of previously unseen shortcomings in present approaches.

1.3 Significance of the study

Globally, a vast number of animal species, including most marine mammal populations, are in a state of conservation concern. Nevertheless, human activities continue to directly or indirectly modify their dynamics. In many such situations the underlying 'natural' dynamics of the population have been modified by a human-induced increase in mortality, even if the populations are not exploited or harvested directly. Such activities invariably accelerate the rate of extinction of such species, raising concerns on their viability and biodiversity.

This study reviews the existing approaches of embedding population characteristics into models, and recommends objective criteria after identifying some weaknesses in present approaches. Findings of this research will help in modelling the dynamics of such animal populations. Even though the analysis is presented in the context of marine mammal models, it has wider applicability as the methods and findings are useful anywhere in the area of conservation of long-lived vertebrates.

1.4 Research Approach

This research reviews present approaches in representing characteristics of populations in population models. The review is based on the basis of their ability to elucidate underlying biological mechanisms and represent them realistically with respect to biological data or biological plausibility. Special focus is given to density dependence and environmental stochasticity, two characteristics commonly represented in models.

First I developed an ecologically motivated criterion to identify qualities that should have in any approach used to represent those characteristics in population models. Then an analytical approach is used to qualitatively investigate the applicability of present approaches on the basis of adhering to above qualities. At this stage I grouped approaches as 'poor' in representing realism, or as 'worthy' of assessing further. The approaches that appear to be most appropriate (and also that are commonly used in practice) are investigated further to compare their performances using simulation methods. Here, various approaches were used to represent the same characteristics in a given (generic) population model so that the resultant model predictions reflect the differences between approaches. Unless otherwise stated, all the computer programmes used throughout the thesis were written in MATLAB®. Those programs are available from the author on request.

1.5 The structure of the thesis

This thesis has nine chapters. In chapter two I present the New Zealand Sea Lion population (NZSL), and a basic model for that population. This model does not reflect the characteristics of a realistic model as discussed in chapter three in terms of appropriately reflecting characteristics of the NZSL population. In fact this thesis does not aim to develop a population model for the NZSL which would involve various aspects of model building that are not within the scope of the thesis. Instead NZSL is used to provide a context for the later chapters. Chapter three gives an overview of important characteristics of a population model. This overview does not limit itself to the two characteristics (i.e., density dependence and environmental stochasticity) that the thesis is focused on, but rather describes how those two characteristics fit into a general modelling framework.

Chapter four discusses various ways of building environmental stochasticity into population models, and compares the suitability of commonly used parametric distributions on analytical (theoretical) grounds. Chapter five compares the performance of the most appropriate candidate distributions in a population model in terms of their ability to represent the intended stochastic variation.

The next three chapters discuss modelling density dependence. The main focus in chapter six is to identify an objective criterion that can be used to choose one density dependent function against others by matching population characteristics to functional properties. Chapter seven evaluates a spectrum of presently used density dependent functions on the basis of above criterion. Chapter eight first evaluates possible population dynamics consequences of such alternative density functions. It then presents a new way of modelling density dependence that can be useful in some situations where more than one vital rate is density dependent but no sufficient information is available to estimate separate density dependent functions. The final chapter summarise the findings and gives general conclusion and generalization. Possible influences on the modelling and management of NZSL by the findings of the thesis are briefly mentioned in individual chapters, and in the last chapter.

2. Motivating Example

2.1 Introduction

The previous chapter introduced the subject of population modelling and the aim of the thesis. Because of the considerable degree of variation in procedures used in population modelling, a general discussion on this subject would be too lengthy and not focused. Instead, this chapter introduces a specific population, the New Zealand Sea Lion population (NZSL), and the related management problem, as the background for discussion in later chapters. The motivation for choosing this particular population is that the nature of the population and the related management problems are very similar to what are found in other marine mammal populations. Further, as described later, measures that could be taken to conserve NZSL pose conflicts with the economic interests of New Zealand fishing industry, and so any decision with regards to management of this population has to be taken very carefully, and will be subjected to heavy scrutiny by all concerned. Also management decisions are likely to be a compromise between recommendations based on conservation biology and social, economic, and political interests. These are common features of most marine mammal management problems.

The key objective of this chapter is to present a focused but still fairly general context for the discussions in later chapters. However, it should be emphasized that this thesis does not aim to model NZSL population, or to provide solutions to its management problems, as that would require attention to population characteristics that are not discussed in this

thesis with sufficient details. A basic (generic) population model is given in the context of NZSL to facilitate the discussion. This model is simple, fairly flexible, and can be enhanced by incorporating the other modelling aspects discussed in later chapters.

2.2 New Zealand Sea Lion (NZSL)

NZSL, *Phocarctos hookeri* (Gray 1844) is the only endemic New Zealand pinniped and, globally, is one of the least abundant sealion species. They were widely distributed and abundant around New Zealand and were heavily exploited before European settlement in New Zealand (Childerhouse and Gales 1996, Heinrich 1998). The current population is about 12000-15000 (Childerhouse et al. 2005), and is highly localized around the Auckland islands (Figure 2.1), where 95% of the pupping occurs (DOC 1999, Gales and Fletcher 1999). Although a few females breed on the New Zealand mainland (at Otago Peninsula and Catlins) (McConkey 1997, Heinrich 1998) those numbers are insufficient to treat them as established breeding sites. Based on the low number of breeding sites, all situated within a few kilometers of each other, the International Union of the Conservation of Nature (IUCN)[1] classified NZSL as a 'vulnerable' species (Reijnders et al. 1993, IUCN 2004). In 1997, New Zealand Department of Conservation (DOC) also classified it as a 'threatened' species under the marine mammal protection act 1978 (DOC 1999).

Because of the highly localized distribution, the impact of a catastrophic event on the population, such as a disease or an oil spill, could be very significant. As an example, at least 57% of pups (Baker 1999) and about 20% of adults (Wilkinson et al. 2005) died from a disease outbreak in the 1997/98 pupping season. In order for this species to achieve non-threatened status, the level of human-induced mortality has to be minimized, allowing the population to establish more breeding sites outside Auckland Islands. The most significant impact of human activities is the accidental bycatch of NZSL in mid-

[1] Updated versions of the species classification criteria and species lists are available at http://www.iucn.org/themes/ssc/red_list_2004/GSA_book/Red_List_2004_book.pdf

water trawl fishery for squid (*Nototadrus sloanii*) around Auckland Islands. Under the Marine Mammal Protection Act 1978 (MMPA), the ministry of fisheries has a legal obligation to manage the fishing related mortality of a threatened species at a level that allows the species to gain non-threatened status as soon as possible. Thus, minimizing the fishing related mortality to a level that does not significantly delay the recovery of the population, while allowing the continuation of fishing activities has become essential.

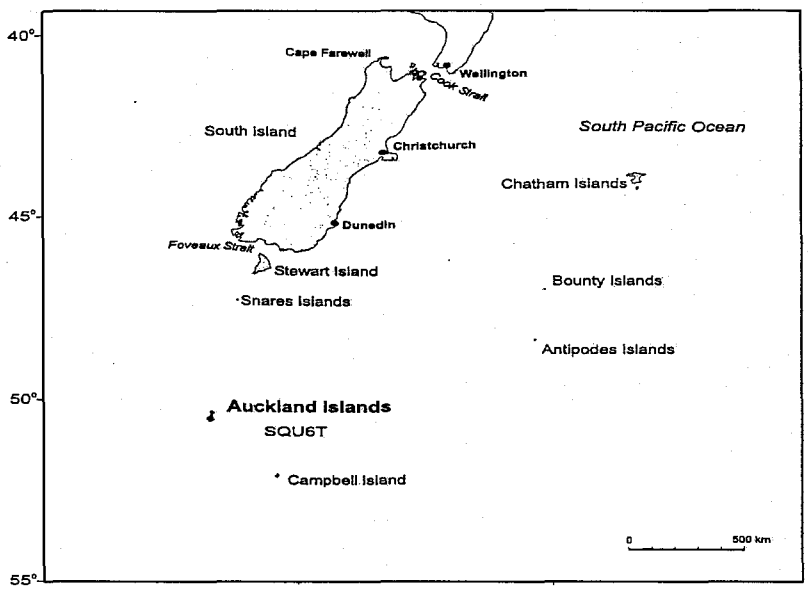

Figure 2.1: The location of Aucklands Islands where NZSL population and mid water trawl fishery for squid are concentrated around.

2.3 Fishing related mortality of New Zealand Sea Lions

The southern squid trawl fishery around Auckland Islands represents 25% of the New Zealand squid fishery, having a present catch limit of 32369 tonnes (MinFish. 2005). The foraging range of NZSL overlaps considerably with this fishing area, resulting in a accidental bycatch in fishing nets (DOC 1999). The level of impact on the population by this bycatch is subject to debate. The fishing industry argues that the bycatch level could still be increased substantially without having a significant impact on the population. Conservation groups, independent scientists, and most government scientists on the other

hand argue the present level of bycatch prevents the population reaching non-threatening status. To mitigate the interaction of NZSL with squid trawls, the New Zealand government has imposed several restrictions on the fishing industry. They include;

- Establishment of a fishing exclusion zone around Auckland Islands in 1982, which was upgraded to a marine mammal sanctuary in 1994.
- Delaying the start of the squid fishing season to avoid interaction with NZSL breeding season.
- Establishment of a Maximum Allowable Limit of Fishing Related Mortality level (MALFiRM) in 1992 where fishery is supposed to close once the bycatch reached the MALFiRM.

Under the Fisheries Act of 1966, the Minister of Fisheries has a legal obligation to close the fishery for the year when the bycatch is reached the MALFiRM. However, due to various reasons, fishery continues in most years even after the MALFiRM was reached (DOC 1999). For example, during the 2002/2003 fishing season, the fishing industry obtained a court injunction against closing the fishery, disputing the bycatch estimated by DOC. Further, in the following year, another injunction was obtained disputing the way the MALFiRM was fixed. To understand the relationship between this management problem, population modelling, and model predictions, we need to explain the MALFiRM, how it is calculated, and how bycatch is estimated.

2.3.1 The Maximum Allowable Limit of Fishing Related Mortality (MALFiRM)

MALFiRM is defined by section 3 of Marine Mammal Protection Act 1978 as, "the maximum number of animals that can be removed by fishing while allowing species to achieve non-threatened status as soon as reasonably practicable, and in any event within a period of not exceeding 20 years". MALFiRM does not include deaths related to human activities other than fishing. According to this definition, the level of MALFiRM cannot be determined without assessing the time to reach non-threatened status under a range of

bycatch scenarios, which essentially requires a population model (a species specific model or otherwise). Presently, in the absence of a species specific population model, MALFiRM is fixed using the Potential Biological Removal (PBR) approach of Wade(1998) developed by National Marine Fisheries Service of United States.

The PBR approach recognizes the common problem of setting a suitable catch limit for a population when a species specific model is unavailable[2]. According to this (generic) formula, the number of marine mammals that can be removed from the population in a year without adverse effects on the population is calculated as;

$$MALFiRM = N_{\min} F_r \left(\frac{\lambda_{\max} - 1}{2} \right), \qquad \textbf{(2.1)}$$

where, N_{min} is the minimum abundance estimate, λ_{max} is the maximum possible rate of population increase, and F_r is the 'recovery factor', a value ranged from 0.1 to 1.0, which ensures threatened populations get the protection needed to recover. In the case of NZSL population, all parties generally agree to use the 20th percentile of the estimated population distribution for N_{min}, and 1.08 for λ_{max}. There is a considerable debate between fishing industry and conservation groups on the appropriate value for F_r. However, a value of 0.15 is used at present. This formula is based on a generic population model for marine mammals therefore it provides an opportunity of conservative management even in the absence of detailed species-specific demographic information. It is simple to use because its inputs are easily measurable (relative to parameters in a species-specific population model).

To implement the MALFiRM the bycatch level has to be estimated in each fishing season. Government observers have been placed on sample of commercial fishing vessels to monitor the bycatch and other activities. The estimation of total bycatch, including those from unobserved vessels, was based on observer reports using the ratio

[2] The underlying principle behind this approach is simple. Even in optimal demographic conditions a population cannot increase by a proportion higher than $(\lambda_{\max} - 1)$; the sustainable incidental mortality cannot exceed $N(\lambda_{\max} - 1)$, where N is the population size and λ_{max} is the maximum rate of populatiin increase. To assess the maximum mortality limit in non-optimum situations, Robinson and Redford(1991) and Wade(1998) proposed the formula $NF(\lambda_{\max} - 1)$, where F is a number smaller than one that takes in to account the effect of density on the growth rate. The PBR formula is formulated by finding optimum values for N, F, and λ_{max} (Wade 1998).

estimation method of Manly et al.(1996), with confidence intervals obtained using the binomial distribution.

2.4 Research in progress towards developing a species-specific model

The PBR formula does not directly address the issue of forming new breeding sites away from Auckland Islands which was the primary reason for classifying NZSL as a threatened species. Therefore, most parties agree that a species-specific population model would be more useful in providing an appropriate mechanism of bycatch control that ensures the restrictions to the fishery is kept at a minimum possible level while population is managed to non-threatened status. There are several population models proposed.

2.4.1 Population models proposed

In 1997, fishing industry proposed a species specific population model, known as the Hilborn model (Hilborn et al. 1997). It emphasizes the effect of bycatch on the probabilities of new breeding colonies being established (rather than targeting a larger population size). This model was subjected to heavy criticism because it does not address the stochastic variation, it relies heavily on unjustified assumptions about the animals' pattern of dispersing, it is more complex than available information can support, and it does not address the time frame of population recovery set out in the legislation. These criticisms caused the model to be further modified, particularly to introduce stochastic variation and to ignore the metapopulation structure, (Hilborn and Wade 1999).

Another model, which is known as the Breen-Kim model, was proposed in 2003 (Breen et al. 2003). This model has been employed to assess the relative merits of using different fishing rules in setting bycatch limit. The objective of these assessments was to evaluate the possible consequences of a range of different bycatch rules, so that management can

choose an appropriate catch rule that deem to be compromising between economic loss and species recovery.

All these models are subjected to criticism on one ground or another. All concerned parties (e.g., department of conservation and ministry of fisheries, fishing industry, conservation groups, and independent consulting agencies) are still trying to agree on an appropriate population model which can achieve the management objective(s), and provide workable management option(s) including a MALFiRM level.

2.4.2 Ongoing research on estimating demographic parameters

Whatever the population model that is used to manage the population, the demographic information plays a significant role, and hence the estimation of demographic parameters is given a priority. Since an unknown proportion of this population always lives in water, and is not able to be detected, the population is studied mainly by observing the portion of animals found on land – specifically, the pups, breeders, and hauling-out animals. Since the 1994/95 pupping season, reasonably precise and comparable pup production estimates are available from a mark-recapture study conducted at the two largest breeding colonies, Sandy Bay and Dundas Islands, and from direct counts at other smaller colonies. The number of pups was estimated to have slightly increased until about 1997 (Gales and Fletcher 1999), to have remained relatively stable for few years (DOC 2002, Breen et al. 2003), and to have decreased since about 2001 (Chilvers 2005). It is uncertain whether the trend in pup estimates is a true indication of the status of population level. Available demographic information from the above major study and other studies include:

- adult survival and pupping interval data from tagged adult females,
- pups and juvenile survival data from tagged pups,
- reproductive rate and age at sexual maturity data from tagged pups,
- bycatch data from observer coverage,
- sex ratio data from pup counts and from bycatch,

- age at maturity data from bycatch carcasses and from growth ring studies of extracted teeth.

The breeding (pupping and subsequent mating) takes place mainly at four breeding sites in Auckland Islands beaches (Figure 2.1), during December and January each year (DOC 1999, Gales and Fletcher 1999). The females may become sexually mature at an age as early as three years old and may produce their first pup at age four (Dickie 1999, Gales and Fletcher 1999). But the majority of females do not produce pups until about six years old (Chilvers 2005). Adult females give birth to a single pup at a time, with a pupping interval of one or two years. Pups are totally dependent on mothers for about 8 months, during which time females alternate between foraging trips at sea and periods on land suckling their pups (McConkey 1997). Pups become independent of their mother at age one if the mother becomes pregnant, or later otherwise.

It has been observed that about 4% to 22% of pups (about 12% on average) die within the 4 – 6 weeks long breeding season (Chilvers 2005). The survival rate estimates from different sources are quite variable. For pups it ranges from about 0.7 (Gales and Fletcher 1999) to 0.8 (Breen and Kim 2005b). Juvenile survival has been approximated to be about 0.8 (Unpublished information from Ian Wilkinson, DOC, Wellington) to 0.85 (Breen and Kim 2005b). Survival rate for adult females has been estimated to be from about 0.73 to 0.9 (Chilvers 2005). Females up to the age of 21 have been observed (Dickie 1999).

Information about dispersal is very limited, although some pups tagged at Auckland Islands have been seen in other locations. The only information available on catastrophic events is the severity estimates of the major disease epidemic in 1997 and few repeat outbreaks in later years.

In the next section I present a simple population model for this population. This model does not encompass all of the present knowledge about this population, but rather it includes basic features of the population as a focused context for discussion in future chapters. Thesis does not aim to produce a management model for NZSL population.

2.5 A basic population dynamics model for the NZSL population

A simple population model is presented here for the NZSL population described above. The structure of the model was designed to represent the main features of current knowledge about the population. It does not represent some characteristics of the population that are known to exist (e.g., dispersal patterns), but it does allow further development by incorporating more features. Even though the model is presented in the context of the NZSL population, the main purpose of it is to present a 'generic model' that provides the context for the discussions in later chapters, specifically to discuss issues related to modelling density dependence and stochasticity.

The model is summarised by the life cycle diagram in Figure 2.2. The main features of the model are:

- Single population with no metapopulation structure because information about dispersal patterns is not available at present, and is not expected to become available in the near future. This thesis does not aim to discuss modelling of metapopulations (see section 2.4.1).
- Structure of three stages (pups, juveniles, and adults). All newly born animals are pups for the first year of life, and all surviving pups enter the juvenile stage next year. Of the surviving juveniles, those who become sexually mature progress to the adult group at the end of the year, whereas those who are immature remain in the same stage. Similarly, all surviving adults remain in the adult stage, so there is no maximum age specified for these animals[3]. Age structure is not used because neither the maximum age nor the age specific vital rates are known. Pups are the only single-aged group in the model (because it is the only age-group that we know vital rates with some confidence).
- Birth pulse model, and post-breeding census (i.e.; the main data, the pup counts, are taken soon after breeding).

[3] This may seem unrealistic because, depending on the survival rates, there is a possibility of considerable proportion of animals passing beyond the observed maximum age of 21. I will discuss this issue in section 3.4.3.4.

- Female only model with assumed 1:1 sex ratio. Because of harem nature of breeding, the fate of the population is not sensitive to the number of males unless males fall to very low numbers.
- Reproduction is by animals in the adult group only[4].
- Stage specific survival rates are subjected to stochastic variation while the reproductive rate is subjected to density dependence effect.
- Animals in the juvenile and adult stages are subject to bycatch, in proportion to their abundance. Bycatch is based on 'at the beginning of the year population' because the fishing season is at the beginning of the year, immediately after the census. Pups are not subject to bycatch as they live on land during whole fishing season.
- There are no catastrophic events, regime shifts, or reproductive senescence in this model, because data on these features is neither available nor can be expected in near future.
- Unlike in age-structured models, age at first maturity is not explicit in this model. A fraction (γ_t) of surviving stage2 animals become mature in year t and enter stage3 as adults while the other surviving juveniles recycle in stage2, hence the duration of stage2 is variable. This allows the time spent in stage2, and thus the age at first maturity, to follow a statistical distribution.

[4] In stage-structured postbreeding models, because the census takes place only once a year after the breeding, some of the subadults in juvenile stage may grow into the adult stage by the time of the census. They must contribute to the reproduction. Thus, as consistent with (Crowder et al. 1994), the corresponding projection matrix in equation 2.2 could include a small value in the first row second column. 'Reproduction by adults only' implies the assumption that no subadults move to the adult group prior to census.

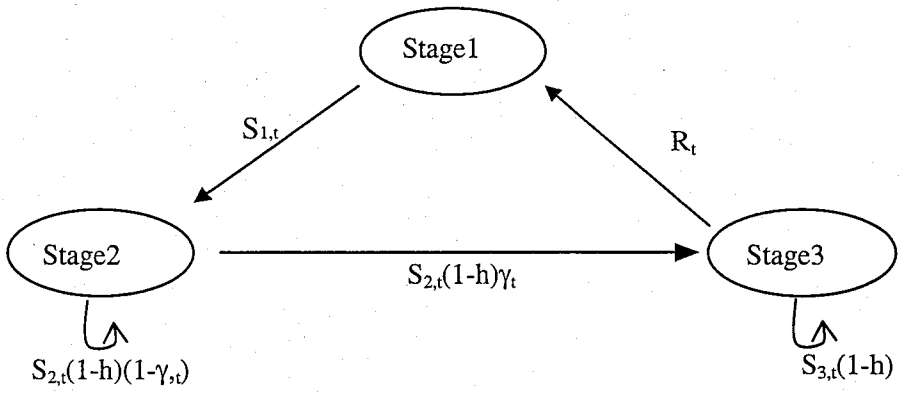

Figure 2.2: Life cycle diagram for the stage-structured population model. $S_{1,t}$ to $S_{3,t}$ are stage-specific survival rates for the year t, R_t is the reproductive rate defined as the number of female pups per adult female for the year t, γ_t is the proportion of individuals that move from stage2 to stage3 at the end of the year t, and h is the bycatch as a proportion of abundance at the beginning of the year.

The corresponding population projection equation for this model is given by the equation 2.2, where $N_{i,t}$ is the number of individuals in stage i at time t.

$$\begin{bmatrix} 0 & 0 & S_{3,t}(1-h)R_t \\ S_{1,t} & S_{2,t}(1-h)(1-\gamma_t) & 0 \\ 0 & S_{2,t}(1-h)\gamma_t & S_{3,t}(1-h) \end{bmatrix} \begin{bmatrix} N_{1,t} \\ N_{2,t} \\ N_{3,t} \end{bmatrix} = \begin{bmatrix} N_{1,t+1} \\ N_{2,t+1} \\ N_{3,t+1} \end{bmatrix} \qquad \textbf{(2.2)}$$

The density dependence in recruitment rate is represented using θ-logistic function as

$$R_t = d\left\{1-\left(\frac{N_t}{a}\right)^b\right\}$$

where $N_t = N_{2,t} + N_{3,t}$. The parameters d represents the value of R_t at the lowest density of N_t, while a is the density when R_t become zero due to density dependent effects. The b is a parameter that determines the curvature of the density dependent relationship.

The environmental stochasticity in survival rate is represented as

$$S_{i,t} = S_{i,t}^{*\,\exp(x)}$$

where $x \sim N\left(0, \sigma^2\right)$, $S^*_{i,t}$ is the survival rate for stage i in year t in the absence of stochastic variation, and parameter σ represents the severity of the stochastic variation. This particular stochastic distribution and density dependent function are discussed in detail later in the thesis (section 4.2.6 and 7.3 respectively). These specific models are selected for stochasticity and density dependence because they were used by Breen et al (2003) in the latest management model proposed the NZSL, and they provide a basis for the discussion in later chapters. In fact the motivation for the present investigation on the suitability of stochastic distributions (chapters 4 and 5) was generated by noticing this particular distribution been proposed to represent the stochastic variation in survival rate of this population.

Here the parameter γ_t represents how fast the surviving juveniles become sexually mature. This information has not been estimated from data, and hence the projection matrix cannot be constructed until γ_t is estimated or specified in terms of other parameters. Under the assumption of stable stage structure, γ_t can be expressed as

$$\gamma_t = \frac{1}{\bar{t}} \exp\left[-\ln\left(\lambda / s_{2,t}(1-h) \right) \left(\frac{\bar{t}}{2} - \frac{V(t)}{2\bar{t}} \right) \right] \qquad \textbf{(2.3)}$$

where $\bar{t}$ and $V(t)$ are the mean and variance of time spent in the stage2, and λ is the dominant eigenvalue of the population projection matrix (Caswell 2001, 164). However γ_t cannot be estimated until the projection matrix is constructed, and the projection matrix cannot be constructed without knowing λ. One way to overcome this difficulty is to use an iterative procedure to estimate both of them at once. Alternatively, a simpler approach can be used to estimate γ_t by assuming a stationary population (i.e.; $\lambda = 1$) as done by Crouse et al. (1987), and then estimate the elements in the population projection matrix.

It is obvious that this basic model cannot realistically represent the dynamics of NZSL population, as it does not contain some of the characteristics of a realistic model that will be discussed in chapter 3. However, its simplicity will facilitate the discussion in later

chapters. This model allows modifications described in later chapters, and also facilitates the assessment of the impact of different management options using a range of indices, which I do not intend to do in this thesis. The main focus of the thesis is to discuss some aspects of adding reality to similar population models. In chapter 3 I give an overview of population modelling. In chapter four and five I discuss how the stochasticity can be added to a model, and chapter six to eight focuses on representing density dependence in population models.

3 A General Overview of Animal Population Modelling

3.1 Introduction

3.1.1 Importance of modelling and aims of modelling

Management decisions based on detailed field experiments may be the most thorough means of comparing the relative merits of management alternatives. They have an advantage over management decisions based on population models because the latter are usually subjected to bias due to the assumptions used in model building. Models are used to estimate present population status from life history data when direct field surveys are impossible or impractical for reasons such as limitations imposed by the behaviour of animals, inaccessibility, or severe weather conditions, (e.g., Chivers 1999). Moreover, field surveys or experiments cannot elucidate the underlying biological process, which is vital for management decisions. Such physical manipulations of a species may not be ethically sound (e.g., release of a known pest to simulate an outbreak), politically acceptable (e.g., experimenting with harvest rates of an economically sensitive exploitable population), demographically feasible (e.g., manipulating an endangered population), or practically feasible (e.g., due to limited funding and time for large-scale manipulations). Population modelling (and subsequent population viability analysis in the case of conservation concerned species) is useful in predicting the fate of the population without actually manipulating the population. It can be used to evaluate the relative effects of alternative management options on the population in an otherwise unforeseeable

future (Dixon et al. 1997). However this statement does not imply that population modelling is superior to field experiments or latter can be replaced with former.

In conservation biology, management actions based on field experiments are often triggered by population levels (or their trends) being either detected by population surveys or predicted by demographic studies (Forney 2000). Such detections through surveys cannot be made until a considerable time after the actual decline has occurred (Wade and DeMaster 1999). However, population modelling can be designed to incorporate uncertainty (both in model structure and parameter values) and stochasticity in demographic parameters into model assessment (see section 3.5). They can also be incorporated into simulations or analytical studies to calculate quantities such as the quasiextinction risk (Thompson et al. 2000). They can be used to provide advance warnings that allow management a significant amount of time to take corrective decisions before the actual decline is occurred.

3.1.2 The flow of the chapter

This chapter gives a general overview of population modelling. It is not designed to be a thorough review of all types of population models. The model building is purpose-specific, hence in the next section we look at major areas of modelling applications (namely; harvest management, pest control, and species conservation) where the success of the population management is dependent on model building. In section 3.3 I discuss the basic framework of different types of modelling, such as isolated population versus metapopulation models, count-based versus demographic models, and unstructured versus structured models. In section 3.4 I look at population characteristics that are generally included in population models, such as density dependence and stochasticity. The usefulness of a particular model, its inputs and outputs, and complexity are determined by the type of the model and the characteristics represented in the model. In section 3.5 I discuss how a model can be assessed for appropriateness, and how uncertainty in model structure and input information can be accounted for in model predictions. The sensitivity of model

outputs to the model's structure and inputs is given as a tool that is useful in making more realistic interpretations from model outputs.

The subject area surrounding the population modelling is very wide as reflected in the growing literature, both refereed papers and text books (e.g., Manly 1990, Morris and Doak 2002, Williams et al. 2002, Lande et al. 2003). Hence I do not intend to review this whole area in this chapter, but rather give the general modelling framework where the contents of later chapters of this thesis fit into. In these later chapters I will not discuss all types of the models and population characteristics stated in this chapter. The thesis intends to discuss mainly the modelling of environmental stochasticity and density dependence with special reference to models of long-lived slow-growing animal populations of conservation concern. As an overview of model building, this chapter includes some sections (sections 3.4.3 onwards) that are not directly relevant to the main focus of the thesis. However this general overview provides background information as to where the above two characteristics fit in to the modelling framework. Although this thesis does not consider in great detail how other characteristics can be incorporated in to the modelling process, it in no way implies that their importance is secondary.

Specific reviews with regards to embedding environmental stochasticity and density dependence in population models will be given in chapters 4 and 6 respectively. In those chapters I identify existing knowledge gaps, which will pave the way for discussions developed in the rest of the thesis.

3.2 Applications of modelling

The purpose of animal population modelling is at least threefold, namely harvest management, pest control, and conservation management (Dixon et al. 1997). Model structure, parameterisation, inputs/outputs, and interpretation of outcome depend on, among other things, what purpose the model is being used for. Some aspects of a model are more important for one purpose than another. The specific uses of modelling in each of these areas cover a vast range. For example, in the area of

conservation of marine mammal species, various models have been used to predict the effect of a catastrophe (Reed et al. 1989) or human-induced mortality (Swartzman 1984, Swartzman et al. 1990), to describe seasonal migration (French et al. 1989), to examine the potential effect of marine debris (French and Reed 1989), to investigate possible causes for an observed population trend (Trites and Larkin 1989, Eberhardt 1990), to estimate age specific mortality curves (Barlow and Boveng 1991) and confidence intervals for the stochastic growth rate (Gerrodette et al. 1985). The diversity of uses in other areas of biological management offers no exceptions.

3.2.1 Harvest management models

Harvest management models, such as models used in managing fishery and wildlife resources, generally aim to optimize the yield from a population while maintaining the population size at a desirable level (Dixon et al. 1997). Earlier harvest models appear to rely on a harvest strategy based on a constant harvest level known as the maximum sustainable yield (MSY) (Pitcher and Hart 1982, Williams et al. 2002). These models are built with the central theme; population growth over and above that needed for exact replacement is regarded as a 'surplus' which can therefore be harvested. The population and yield are assumed to remain stationary when the harvest is equal to the surplus production. This assumption has been subjected to criticisms, for example (Hilborn and Walters 1992)[1].

Although one of the major aims of most harvest management models is to maximise the economic benefits, MSY based models are based purely on biological factors only. Non-biological inputs such as socio-economic and political factors are later incorporated in to MSY models to predict what is called an optimum sustainable yield (OSY), (Gulland and Robinson 1973, Hannesson 1993). Support for such analyses is evident in recent literature (e.g., Enriquez-Andrade and Vaca-Rodriguez 2004).

[1] MSY is the maximum yield, measured either by number of animals or by their weight, that can be harvested from the population on a continuous basis without affecting the population size. Due to the changes in population and in the environment, the production capability is continuously changing, and hence it is unlikely for such a constant population level to exist in reality (Hilborn and Walters 1992).

The major practical advantage of these surplus yield models is that they require only easily available data, such as catch and effort data, the sort of information that has been accumulated over many years for most harvestable populations. Nevertheless these models fail to represent populations realistically and as a consequence there are many examples in the modelling literature that did not achieve their intended goals. A well known example of this is the Peruvian anchovy fishery, one of world's largest fisheries that contributed 15% to the world's total fish production. This fishery collapsed in 1972 even though the catch was well within the 'safety zone' as predicted by its MSY based population model. The single most important factor lacking in these surplus yield models, where the models cannot stand much chance of predicting the likelihood of population collapse, is the lack of realism due to ignoring environmental randomness (Pitcher and Hart 1982, p236). MSY and OSY do not appear to be the management goals in recent models. During the last decade attempts have been made to develop harvest and management models that could lead to more robust management decisions even in data rare situations e.g., (Wade 1998).

3.2.2 Pest and disease control models

Pest control models are used to help decide on strategies for eradicate pest populations or to control them at a level that prevents harmful outbreaks (e.g., Mount and Haile 1987). The simpler form of these models focuses only on the dynamics of the pest species. Usually those models evaluate the effects of single-species management actions such as removing individuals from the population or releasing sterilized males to the population. These models generally evaluate a stochastic density-dependent description of population dynamics with the intention of developing optimal control policies that maximise the realised gain (e.g., Mann 1971). More complex multi-species pest control models explicitly consider the interaction between pest and host populations, and generally focus on the balance between the two populations, unless the aim is eradication (e.g., Grasman et al. 2001, Zhang and Chen 2006). Such interaction models are usually more complex for analytical studies, thus a simulation approach is commonly used to make population predictions (Dixon et al. 1997).

Any control action disturbs the balance of nature; the resulting imbalance from an action could possibly be more severe than the initial pest situation. In order to account for this, the model should have a mechanism of balancing gains achieved by control actions against its costs.

3.2.3 Conservation management models

Conservation management focuses on increasing the size of a small population (Wade 1998), reducing the probability of extinction of a rare species (e.g., Slooten and Lad 1991, Heppell et al. 1994, IWC 1994) adjusting the minimum viable population (Soulé 1987), or improving persistence time (Williams et al. 2002). Managers are often required to answer questions regarding threatened and endangered populations like how much harvesting can be allowed (e.g., NZSL population), the value of effort of trying to preserve a particular population, or the relative effectiveness of enhancing the survival of abundant juveniles and minority adults? Answers to such questions demand quantitative measures.

Conservation biology aims to provide answers to these questions through population modelling, population viability analysis (PVA), and sensitivity analysis. In doing so, conservation managers often use age or stage based stochastic matrix models to make long term projections for their populations (Manly 1990, Burgman et al. 1993). A recurring problem in most of these works is that often data are scarce and the time and resources available for the collection of new or additional data is often limited. What actions can the managers take if management actions cannot be delayed until better data are collected? They have to make the best decision on the basis of the available data (until better data are collected). In such situations, population models used in conservation biology are often built using the theme that simpler models based on general assumptions can generate useful management recommendations. Such an approach is recommended by Starfield (1997) and Wade (1998), and seem to be accepted by some managers. For example, in the process of developing a model for NZSL, the Technical Working Group decided to use a simpler model by ignoring the

metapopulation structure that was present in the originally proposed model (i.e., Hilborn et al. 1997, model), simply because it was more complex than the available dispersal data could support even though it was known that dispersal patterns exist and are important for better management decisions. NZSL management cannot be delayed for another 8 - 10 years until new dispersal data are collected. However, it does not mean management should try to find a simpler model to fit the available data whenever good enough data are unavailable. If the available data are insufficient for a realistic model we should attempt to collect more/better data.

Similar to the concept of OSY in harvest models, the 'trade off' between conservation gains and consequent economic losses of management actions has been suggested for conservation models (e.g., Possingham and Shea 1999a). Such an analysis has been done for New Zealand sea lion population (Maunder et al. 2000). However, unlike harvest models, the suitability of such approaches in conservation models is subject to debate, e.g., (Bedward 1999, Hamilton 1999, Possingham and Shea 1999b).

This thesis inclines towards the models aimed at conservation goals, and so the material given is mainly in the context of population models used in conservation biology. However the concepts developed have a general use, and are applicable to models in other areas too.

3.3 Different types of population models

The types of models used in population management depend primarily on the questions being asked (i.e., management goals). The simple models have only an empirical fit of a mathematical relationship to observations hence can be used for projecting the population into the future, but without giving any understanding of or the insight into the population. Such projections reveal the present status of the population rather than possible future fate (Dixon et al. 1997). Confusions arise in the literature when interpreting results from projection models due to not distinguishing them from possible future fates of the population (as clarified in section3.4). More comprehensive models are complemented with added realism; they elucidate the natural process of the dynamics, and they can be used to predict possible responses of

the population to different management options. The suitable compromise between comprehensiveness and simplicity of a model for a given population is governed by the management objectives and available data. In this section I briefly mention common types of population models. None of them will be discussed in detail, but rather presented as an overview of models providing the background for the rest of the thesis.

3.3.1 Continuous-time and discrete-time models

At an early stage of an ecological modelling exercise, a choice must be made between the discrete-time and continuous-time approaches. The choice can sometimes appear obvious. For example, we might be modelling the development of an organism whose growth involves a series of discrete stages associated with sharp demographic changes, or we might be concerned with a population with discrete (non-overlapping) generations. Even in such apparently straightforward cases, however, there should still be some pause for thought. Discontinuous growth is the culmination of a continuous physiological process, and discrete generations are caused by continuous processes within a single time period.

The development of continuous-time models preceded that of discrete-time models, with the latter frequently viewed as approximations to the former. Goodman (1967) and Keyfitz (1968) attempted to reconcile these two schools of approaches for simpler (early) models that existed about forty years ago. No such attempt has been made for recent models (Caswell 2001). Moreover, continuous and discrete versions of the same model can yield quite contrasting results in some situations, thus the selection of an appropriate version is an important preliminary step in model development[2].

Continuous-time models are often described through differential equations. They are generally easier to treat analytically, and there is a large body of mathematical literature concerning their properties. However, they are much less well adapted to

[2] For example, the discrete-time version of the logistic count-based model can produce chaotic behaviour whereas the continuous-time version of the same model with comparable input data cannot (Alsop 1998).

numerical realisations, especially when the model is more comprehensive with structural and stochastic model components.

Discrete-time models on the other hand make some approximation to the outcome of these processes over a finite time interval. Difference equations, which constitute discrete-time models, are intuitively appealing and simpler for numerical calculations (Gurney and Nisbet 1998). Structured, discrete-time population models are perhaps the most widely used population models, especially for populations with seasonal behaviours (e.g., annual breeding cycles), sharp demographic changes, or a series of development stages in the life cycle. Assuming that a discrete-time model provides a reasonable approximation to the continues-time dynamics of the species concerned, their only known serious drawback is that they sometimes pose a considerable challenge to analytical treatments. For example, for stochastic structured discrete population models, there is no way of analytically calculating the time needed to recover a depleted population (i.e., time to reach the population level to a given percentage of its carrying capacity), or the probability of extinction, under a certain management action.

Discrete-time models with seasonal breeding patterns (i.e., birth pulse breeding) can either be 'prebreeding' or 'postbreeding'. The number of animals in a prebreeding model refers to the number alive just before breeding starts. These models reflect the situation where the population census is done prior to breeding. In contrast, postbreeding models refer to the number of animals alive immediately after breeding occurs, and therefore reflect the situation where the population censuses are done after the breeding occurs (Figure 3.1). The number of animals in a given year is different between prebreeding and postbreeding models even if all the demographic parameters are the same in the two models, because they refer to two different points of time within the year. The sequence of survival and reproduction events within a year is also different in the two types of models. In prebreeding models, reproduction occurs prior to survival, while in postbreeding models reproduction occurs after survival.

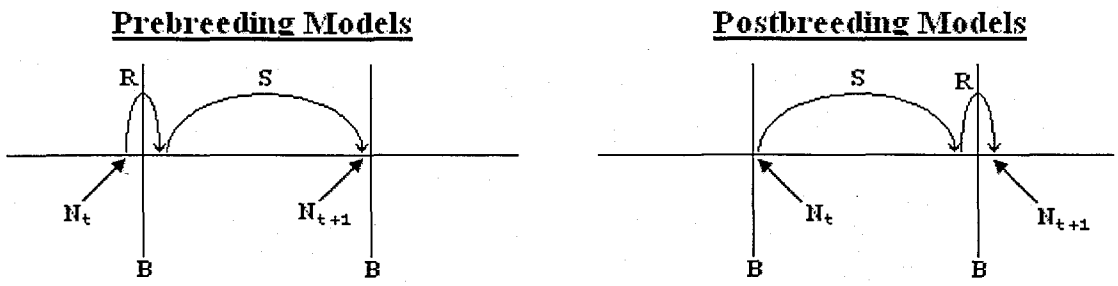

Figure 3.1: Prebreeding and postbreeding models. The horizontal axis denotes the time, and B denotes the points of time that annual breeding events occur. R and S are the reproduction and survival events respectively. N_i is the population size at time *i*.

Most large mammal species have seasonal patterns of behaviour; they breed seasonally, they are subjected to seasonal predation and bycatch, and food availability dispersal etc are correlated to seasonal weather patterns. The information on most populations is also gathered on discrete time intervals (e.g., annual census, annual bycatch estimates, etc). Therefore the later chapters of the thesis focus on discrete-time models. Both prebreeding and postbreeding models are examined because for a given population, available information will more closely resemble one type of model than the other.

3.3.2 Population based models and individual based models

Individual-based population models (IBPMs) apply a bottom-up approach to modelling population dynamics in time and space. IBPMs define a population by the characteristics of its constituent individuals. These models follow each individual throughout its life, tracking all events such as birth, growth, reproduction, and death (e.g., Chivers 1999). IBPMs contained a detailed account of the process of the dynamics, and explicitly take in to account the individual differences (demographic stochasticity). Since individuals with different characteristics (age, stage, size, etc.) may behave differently, population dynamics can only be predicted by integrating over all individuals what happened to them after they had interacted with each other and the environment. Individual-based models have been shown to be especially useful as tools for understanding the interplay between behaviour, population dynamics, and evolutionary processes.

Individual-based models are growing in interest in ecological modelling (Grimm 1999). An explanation of this growing interest is that individual based models can incorporate very detailed and refined representations of the individuals and their behaviour. It is expected that this additional level of detail brings more realism into the model, which becomes a more faithful representation of reality. However, such detailed descriptions lead to heavier computational models which are sometimes very difficult to understand due to loss of simplicity. In conservation biology, these models are used to manage very rare species for which there are often very detailed data on the few remaining individuals (Gross et al. 1992).

In contrast to IBPMs where the individual is used as the modelling unit, models that use aggregated variables to describe the dynamics often attempt to use the whole population as the modelling unit. Even though less realistic, aggregate models have some advantages over their IBPM counterparts; they require shorter computation times, their mathematical form provides theoretical insights on their possible behaviour even before any simulations are performed, they contain useful elements to explain the results, and they require much less data than IBPMs demand. Moreover, administrators and managers are often interested on aggregate variables such as the number of individuals, their distribution of ages, and trends in these variables. Therefore, the bottom-up approach alone is insufficient for management decisions at system level; top-down approaches are needed to provide an appropriately integrated view at the population level (Grimm 1999).

Edwards et al.(2005) show that the results of the IBPMs and aggregate models can be very similar or very different, depending on the chosen parameters. For some parameters, the aggregate models are trapped in local minima, whereas the individual based models, because of their stochasticity, always get out from these local minima.

It is therefore important to understand in which conditions the development of an individual based model will actually bring some additional insight to the study population. For instance, if the additional complexity of the individual based model does not bring any (or considerable) difference in the global behaviour, then simpler

aggregate models are certainly more appropriate. That could be the case for most populations when the population size is not very small.

Even in situations where IBPMs are more appropriate, they can be beyond practicality for most populations due to the heavy demand of data. Therefore population based models are more common in all areas of biology. The rest of this thesis is focused on population based models. Consideration of whole population as a single unit in population based models is similar to assuming every individual in the population is identical, which is generally an unrealistic assumption. Structured population models provide a compromise between these two extremes by recognising the inherent heterogeneity as well as the limitations in distinguishing individuals explicitly (Manly 1990).

3.3.3 Single-species and multi-species models

Single-species models implicitly assume that the effects of all interactions between other species can be represented in the model by embedded deterministic and stochastic changes to its vital rates. For example, none of the models proposed for NZSL directly consider the variability in available food resources (i.e., dynamics of squids and other prey species). This assumption is relaxed in multi-species models. The interaction between species, say through competition for resources or predator/prey relationships, are explicit in multi-species models (e.g., the Lotka Volterra equation[3]). In some situations one of the main objectives of modelling is the examination of the nature of this interaction in order to identify appropriate management actions (e.g., Zhang and Chen 2006). Such models are very common in some areas of ecology such as biological pest control. Reasonably realistic species interaction models are usually more complex for analytical studies, and so a

[3] The general form of Lotka Volterra equation that describes the dynamics of species *i* interacting with other *m* species is $\frac{dN_i}{dt} = r_i N_i \left(1 - \frac{N_i}{K_i} - \sum_{j=1}^{m} \alpha_{ij} \frac{N_j}{K_j}\right)$, where K_j is the carrying capacity of species *j*, α_{ij} is the coefficient of interaction between *i* and *j* species (May 1973, eq 17). When the interaction is with one species only, above equation reduces to $\frac{dN_i}{dt} = r_i N_i \left(1 - \frac{N_i}{K_i} - \alpha_{ij} \frac{N_j}{K_j}\right)$, for $i,j = 1,2, \quad i \neq j$ (Gilpin and Ayala 1973, eq1). When there is no interaction with another species, this equation further reduces to $\frac{dN}{dt} = rN\left(1 - \frac{N}{K}\right)$. This is the continuous-time logistic equation of Verhulst (1838). We consider the discrete-time version of this equation in section 7.2.

simulation approach is commonly used to make population predictions (Dixon et al. 1997).

Most species interaction models suffer one way or another from the lack of reliable data on the nature of the interaction. As a consequence they are often subjected to unverified assumptions, especially when applied to small and endangered populations. Although more realistic and comprehensive, they are rarely used in conservation biology due to their complexity and heavy demand of data. In conservation modelling, one is usually interested in populations that are 'data poor', therefore multi-species modelling is usually inappropriate. The focus of the rest of this thesis is on single-species models.

3.3.4 Count-based and demographic models

Any population model can be identified either as count-based or demographic. The term count-based is used to imply that the dynamics of the population is measured on the basis of the total number of individuals in the population, without explicitly accounting for their demographic parameters. Demographic models account for the demographic parameters explicitly, and represent the dynamic as a demographic process. They provide more insight into the underlying population processes, and permit more meaningful management actions to be identified.

The rest of this thesis is given in the context of demographic models. However, the contents are equally applicable to count-based models too.

3.3.5 Unstructured and structured models

Models can also be grouped under the headings of unstructured and structured. Unstructured models consider the whole population as a single homogeneous unit (i.e., assume all individuals in modelled population are identical). All count-based models fall into this group. Structured models, on the other hand, recognise the

heterogeneity in the population; they divide the heterogeneous population into a series of subgroups where individuals in each subgroup are assumed homogeneous. Demographic models can either be unstructured or structured.

3.3.5.1 Unstructured models

As stated later in sections 6.1.2 and 7.2, unstructured population models were introduced to ecology by Malthus, Gompertz, and Verhulst in 1798, 1825, and 1838 respectively. But they were not used in biological management. They were redeveloped (or redesigned) for the management of fishery resources about 50 years ago (e.g., Ricker 1954, Schaefer 1954, Beverton and Holt 1957, Pella and Tomlinson 1969). These models were designed to focus on density dependent relationships, and to estimate the suitable harvest levels and the sustainability of the population. Later they were modified for use in managing other ecological resources. A few important drawbacks with unstructured models have been identified. First, they are based only on the most general (i.e., over-simplified) biological principles, and therefore lack biological realism, meaning they provide mainly an empirical fit to data (Ragen and Fowler 1992). Second, many possible dynamic forms could empirically represent the observed relationship (Pascual et al. 1997), so it is difficult to prefer one model against another. Third, they poorly demonstrate the mechanism of the dynamics, and therefore cannot elucidate the relative merits of alternative management options. Fourth, because they treat all individuals identically, they are incapable of dealing with heterogeneous populations even when information about the heterogeneity is available. When the population is highly heterogeneous, unstructured models tend to ignore the extreme (undetectable) segments of the population and focus only on the other segments. For example, surplus production models in fisheries focus only on the fishable part of the population ignoring the larval (and sometimes juvenile) stages where individuals are subjected to heavy mortality, (Hilborn and Walters 1992)[4].

[4] These models make adjustments to model parameters to reflect the effect of ignored portion of the population. This is similar to the implicit representation of immigration and emigration in single population models using adjustments to model parameters, as opposed to explicit representation of them in metapopulation models (section 3.3.6).

Irrespective of these drawbacks, unstructured models are widely in use because of their simplicity, analytical ability, and the heavy demand of data by alternative (structured) models. Less realistic unstructured models can often give more useful predictions than by more realistic structured models when data are rare (Ludwig and Walters 1985, Dixon et al. 1997).

3.3.5.2 Structured models

Structured models deal with heterogeneity of individuals by forming them into subgroups, so that all individuals in a single subgroup are homogeneous. Subgroups can be formed based on any demographic character that varies over the life of individuals (e.g., age, stage, size, ... etc), so the individuals pass through different classes over their life. Age-structured models are simpler because all animals in one age class move to the next age class in following year if they don't die or migrate. The homogeneity assumption within each age class is equivalent to assuming that all properties relevant to individuals' demographic fate are highly correlated with age (Caswell 1989, p19). This assumption is generally not true as the characteristics of individuals within an age class could be dissimilar than those in different age classes. Also, because age is difficult to measure in wild animals, the age-specific vital rates required by age-classified models are difficult to estimate in the field. Therefore other forms of classification could be more practical with those populations.

Stage-structured models classify individuals based on a more easily observable character, the stage. Unlike age structured models, only a portion of the surviving animals progress to the next stage over a year in stage-structured models. Therefore, these models require estimates of transition rates from stage-to-stage. Except in rare occasions, animals progress along the structure generally in one direction, usually from less matured classes to more matured classes. Population models that contain combinations of age structure and stage structure are common for marine mammal species.

Size structure is similar to stage structure, but can be more complicated in populations where individuals can grow as well as shrink in size over time. Shrinking individuals move backward in class structure while growing animals move forward[5]. Size structure is more common than age or stage structure in some areas of modelling where available data classify individuals only on size (e.g., tropical fisheries models based on length frequency data).

Structured matrix models were first introduced by Leslie (1945). They have a characteristic matrix of parameters known as the Leslie matrix or projection matrix. This matrix was later generalized to stage-structured models (e.g., Lefkovitch 1965, Usher 1969), and also to include other population characteristics such as density dependence and stochasticity. They became more popular after about 1970s.

3.3.5.3 Unstructured versus structured models

Having fewer parameters than in structured models, unstructured models (especially the count-based models) are solvable algebraically to estimate specific measures of its dynamic (e.g., growth rate, probability of extinction, optimum harvest rate, etc). More realistic structured models are based on complex variations of the stochastic Leslie matrix (Leslie 1945), and are insolvable analytically for most measures of population performance indicators. For example Tuljapurkar (1990, ch11) proposed an analytical method to estimate the sensitivity and elasticity of the stochastic growth rate to changes in the entries of the stochastic projection matrices. An extension of this result to estimate such elasticity and sensitivity to changes in demographic rates was developed only recently by Caswell (2005). There are no such analytical methods developed to estimate other population performance indicators such as extinction risk and time to extinction for stochastic models. Therefore most analyses of structured models are based on numerical and simulation approaches.

Structured models have four important features. Firstly, they represent the entire population, contribute towards biological realism, and relax certain strict assumptions

[5] Such movements in various directions can be seen in multisite and multistate models too (section 3.3.6).

in unstructured models (homogeneity). Secondly, they require more input data, and are more difficult to formally analyse. Thirdly, they have a greater chance of introducing bias and uncertainty to model predictions when the required input information is not known with appreciable level of accuracy and precision. Finally, they do not replace the realistic features (such as density-dependent growth mechanisms) existing in simpler analytical models, but rather retain them by embedding into the elements of the parameter matrix.

Unstructured and structured models do not always lead to the same population predictions even if comparable data are used[6]. For example, density-independent unstructured models always predict populations growing (or declining) at a constant rate starting from the initial year[7]. The structured models with comparable input data predict a different growth rate in initial years, and asymptotically reach the growth rate predicted by the unstructured model once population structure becomes stable. Further, when density dependence is present, growing populations converge to an equilibrium level which is reached at a constant rate under unstructured models. Such a convergence cannot be guaranteed in structured models; in some cases it is possible to show a cyclic pattern instead. Even in situations where convergence occurred, structured models show a transient behaviour before converging (Caswell 1997). Reduction of survival rates in unstructured models, for example by increasing the predator/bycatch pressure, can predict a higher probability of population extinction than predicted by structured models. This suggests the inherent resilience of the structured models to perturbation; that is, the presence of individuals in different age/stage groups allows some of them to escape the perturbation more than others, hence forming the basis of population recovery[8]. This may be a more realistic depiction of what is actually happening in nature.

Even if no significant or apparent difference exists in the behaviour of populations as projected by structured and unstructured models, structured models have more

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[6] This is similar to the possibility of having different outputs from continuous and discrete versions of comparable models, section 3.3.1.

[7] This is assuming no stochasticity. Stochastic environmental variation can produce irregular fluctuations in growth in both structured and unstructured models.

[8] This is comparable to increased resilience in multi-site models (section 3.3.6). Individuals in multiple sites allow some individuals to escape unfavorable conditions, forming a buffer effect.

advantages. Recognition of these advantages is reflected in the growing literature published in the last two decades. Among those advantages are:

(a) It is possible to identify subgroups that the population is most sensitive to, enabling researchers to identify where the management actions are to be focused for them to be most effective. For example, the best strategy for ensuring the persistent of any endangered species may be conserving the subgroup that makes a disproportionately higher contribution to the overall population growth.

(b) They provide flexibility in comparing the effectiveness of alternative management strategies such as controlling a selective predator or selective bycatch pressure.

(c) Most management actions create perturbations by altering one or more vital rate. The resultant transient behaviour is more appropriately depicted by structured models than by unstructured models.

Although early models used in population biology are unstructured, structured models are now common in all areas of conservation biology. With few exceptions, almost all species-specific models used in the conservation of marine mammal populations are structured. I use structured models as the context for the case studies in later chapters of this thesis, but the concepts are equally applicable to unstructured models as well.

3.3.6 Metapopulation models and multistate models

The closed population models assume no movements in or out apart from through birth and death[9]. The open single-population models relax this assumption by allowing permanent and temporary immigration/ emigration, but do not specifically account for the dynamics of individuals from the time they left the population until (if ever) they return. This spatial effect is incorporated more realistically in multi-site metapopulation models by explicitly accounting for the dynamics of animals in multiple locations, and their movements between locations in any direction.

[9] The term 'closed population' is defined differently in the literature. In mark-recapture studies, closure usually means the size of the population is constant over the period of investigation, i.e., no recruitment (birth or immigration) or losses (death or emigration), (Otis et al. 1978). Here I used the term in the meaning of definition of Hale et al. (1995) as, a population into which there is no gene input from outside, i.e., the only possible genetic change is through mutation.

Demographers of human populations were the first to incorporate multiple locations into projection matrix based population models (e.g., Rogers 1966). These so-called multi-site matrix models are becoming more common in present day animal ecology (Lebreton et al. 2000).

3.3.6.1 Metapopulation models

Dispersal inter-connectivity between subpopulations has important effects on the abundance, strength of fluctuations, and the probability of extinction for both individual subpopulations and for the whole metapopulation (Williams et al. 2002). Even though some observations exist to the contrary (e.g., Hess 1996), it is generally believed that small populations are more vulnerable to extinction in isolation; dispersal links between subpopulations often increase the species stability by working as a buffer against extinction (e.g., Hill et al. 2002). If one subpopulation tends to extinct, other subpopulations feed their resources into it through dispersal, making it recover as a result (DeWoody et al. 2005). Therefore, the models that fail to recognize the dispersal properties of populations lead to over-estimation of population extinction risk.

There are advantages of metapopulation models in a genetic and evolutionary perspective. Small isolated populations are liable to inbreeding depression. In addition some genes are liable to be lost from those populations due to natural selection process. Metapopulations allow the reintroduction of lost alleles from other subpopulations and new combinations can also be formed by occasional mixing. Livestock breeding programmes have found this process is faster and more effective than natural selection for generating adaptive stocks (Wade 1976, Slatkin and Wade 1978). This genetic migration between subpopulations is an important factor for species persistence, which is not allowed for by single population models.

In metapopulation models, animals tend to migrate or return depending on the favorability of local conditions and the level of competition for local resources. Single population models, on the other hand, rely on the implicit assumption that all such

factors can be accounted for by density dependent (and stochastic) adjustments to vital rates of the individuals[10]. Therefore, the density dependence in single populations can be considered as an implicit surrogate for the metapopulation structure.

Conservation management based on metapopulation models is more sensible because the management unit and the unit of evolution are the same. However, even when the actual population is known to be a metapopulation, individual subpopulations become the unit of management for practical reasons, for example when the metapopulation is distributed across political boundaries. Thus, the importance of single population models cannot be undermined in practice.

3.3.6.2 Multistate models

Multistate models generalise metapopulation models beyond the spatial structure. They allow individuals to be distributed across multiple sites as well as among multiple states (Williams et al. 2002). A *state* can be any subgroup of the population, with individuals in each state having state-specific demographical properties and the ability of transiting between states. States can be based on physiological or behavioral characters (e.g., breeding or not breeding, heavy or light), or geographic locations (e.g., habitat types, breeding colonies). Similar to stage structured models, multistate models include a suite of transition probabilities. Although stage structure does not normally allow transition from a mature state to a younger stage, multistate models allow transition between states in any direction. The term multistate model generally refers to models that permit stochastic transitions among states as distinct from deterministic transitions among age/stage classes in other structured models.

Many questions in evolutionary ecology and conservation biology require the investigation of movements between states as stochastic functions. In recent years there have been increasing interest in problems involving both multiple phenotypic states and multiple locations (e.g., Nichols et al. 1992, Hunter 2001, Bradshaw et al.

[10] This is similar to the assumption in single species models that all interactions with other species can implicitly be represented through adjustments to vital rates.

2003). The growing interest in spatial aspects of population dynamics presently contributes to making multistate models a very promising tool for population biology.

The multistate generalisation of metapopulation models, and mark-recapture methods in particular, are an extraordinarily powerful tool in estimating various demographic parameters (Caswell and Fujiwara 2004). Nichols et al. (1992) presented the first explicit application of multistate mark-recapture methods to matrix population models. Lebreton and Cefe (2002) reviewed the interest and the potential of multistate capture-recapture models, in particular when they are used with demographic states as well as geographical sites.

Limited demographic information often compelled conservationists working with long-lived slow-growing animal populations to use single population models even when the advantages of using metapopulation models or multistate models are clear. This thesis is not about metapopulation or multistate models. The following chapters are focused on modelling vital rates, and so the contents are applicable to all models even though they were presented in the context of modelling a single population.

3.4 Characteristics of populations represented in models

Any population model tries to represent the population dynamics as realistically and simply as possible. Even though the model should not be complicated to the extent that the data cannot support, oversimplification will cause loss of realism so that model predictions are of little use. The level of reality represented in a model depends on the management goals and available knowledge. With improvements in the understandings of the underlying processes there is a tendency of turning towards more comprehensive and more realistic models (Ragen and Fowler 1992). With regards to marine mammal populations, this tendency shows a number of trends over time:

- Increasing model complexity through a shift from simple count-based models to complex structured models (Morris and Doak 2002).

- Trend from projection models to predictive models[11].
- Enhancing realism by incorporating additional components to the model such as stochasticity, density dependence, senescence, uncertainty, catastrophe, dispersal and species interactions (Dixon et al. 1997).
- Growing emphasis on adequacy (or limitations) of models through the processes of validation and sensitivity analysis (Mills and Lindberg 2002).
- Use of simulation based methods (Manly 1997) more than analytical methods in drawing inferences from models.

These trends reflect:

- The progress in understanding population regulation process and its limitations.
- The extent (and limitations) of availability of biological information.
- Advancing technology/methodology (Goodman 2002).
- Diversity and changing management goals (Dixon et al. 1997).
- Importance of recognizing limitations in any realistic attempt to manage animal populations.

To build a realistic predictive model we need to include the natural process in the model at least to the level demanded by the objective of the model. In this section I focus on enhancing model realism by incorporating additional components to the model. The natural process may be driven by characteristics of the population such as the presence (or absence) of density dependence and its form, dispersal, heterogeneity, species interactions, human interactions, senescence, and stochastic variations, ... etc. In addition, the available knowledge is often inaccurate (biased) and/or imprecise (uncertain), and these issues also need to be addressed when producing model predictions for species management. In this section I present an overview on how such characteristics are built into models. I neither attempt to present an in-depth discussion on any of these components in this chapter nor to discuss all population characteristics, but rather present only the most commonly used

[11] Projection models extrapolate present qualitative trends into the future to describe what would happen if given conditions persist. They do not reveal the possible behaviour of the population in future (Keyfitz 1972). Predictive models on the other hand forecast about what can happen in future by explicitly accounting for possible changes for present conditions (Dixon et al. 1997). Interpretation of demographic analysis requires distinction between projection, and prediction. Confusion results in ecological literature due to the failure in recognizing this distinction (Caswell 1989, p20).

or most applicable characteristics. Two particular characteristics, namely stochasticity and density dependence, will form the basis of the later chapters.

3.4.1 Stochasticity

In its simplest terms, stochasticity can be described as unpredictable nature in population dynamics, which is often a combined result of various factors such as demographic and environmental. The effect of demographic factors on the dynamics is more prominent in small populations whereas environmental factors have larger influence on the dynamics of large populations. Use of individual based models (section 3.3.2) is an excellent way of representing demographic variability between individuals. However, except in very small populations, this approach is rarely used in practice due to the difficulty of tracking each individual in the model throughout their life. Structured population models provide a compromised alternative. This thesis does not focus on demographic variability. One of the main aims of this thesis is to study the methods of modelling environmental stochasticity. Gaston and McArdle (1994) reviewed the measures, methods and patterns of temporal variability in animal abundance. The variations in abundances are directly correlated with the changes in the underlying vital rates. In section 4.1 I discuss representing environmental changes in vital rates in more detail. Chapters 4 and 5 are devoted entirely to modelling environmental stochasticity.

Deterministic models are easy to construct, simple to understand, possible to analyze for most outcomes using analytical tools. They are often over-simplified by ignoring temporal variations (and individual differences) and by assigning average values for vital rates, and hence lack realism. The management consequences of using such deterministic models are well documented (Bjornstad and Hansen 1994), and one such example is given in section 3.2. However, they can help understand the general behaviour of the population with regards to the underlying mechanism of population dynamics (e.g., whether the population cycles, or is chaotic), (Dixon et al. 1997).

Stochastic models demand more data and more computational power, and suffer from lack of analytical solutions for most performance indices of interest to management.

But, in conjunction with numerical procedures they produce more accurate and realistic predictions than deterministic models. Simulation methods allow assessing any desired outcome of stochastic models, without being confined to some specific outcomes as in analytic methods. These simulation methods are capable of analyzing very complex life cycles and stochastic patterns. For example, analytical treatments will be very difficult, if not impossible, for some performance indicators in life cycle situations when stochasticity allows some individuals to bypass certain stages of the life cycle and/or force them to return to one of the previous stages of the life cycle. However these situations can easily be allowed for with simulation methods.

There are various approaches in the literature to represent environmental stochasticity in population models.

1. One of the ways is by estimating a separate set of vital rates for each time interval. In the case of matrix models, this involves estimating time-specific transition matrices. Dixon et al. (1997) used this approach by estimating a time series of transition matrices to describe the influence of random environmental variation. The advantages of this approach are that, it simultaneously includes both demographic and environmental stochasticity, it avoids the need for numerical methods, and it allows complex stochastic models to be evaluated using available analytical techniques. There are also marked drawbacks in such methods such as involved complexity in analysis, added sampling variability, and the difficulty in interpretation of results. Most importantly, this approach is far beyond practical limits for most populations due to the impossibility of estimating time-specific matrices. A single projection matrix with embedded stochastic components (i.e., stochastic transition matrix model) offers an alternative method (number three below).
2. If a long time-series of estimates is available for various vital rates, then vital rates for each time step of population projection can be sampled from that list. This is analogous to the non-parametric bootstrap. Kaye and Pyke (2003) used such a series of matrices in a simulation study by randomly selecting a matrix at each time step. An added advantage of this method is that any cross correlation reflected in those estimates are effortlessly incorporated in to the population projections. However, this sampling approach discards any autocorrelation present in the time series of vital rates (see section 3.4.3.5). It

also has limited practical value because it is rare for such long lists of vital rates estimates to exist.

3. The most common approach is analogous to the parametric bootstrap procedure. Here, the stochastic variability of each vital rate is expressed by a statistical distribution, with parameters for that distribution estimated explicitly from observed variation. Vital rates for each time step in the population projection are sampled from that estimated distribution.

This most common method requires a priori specifying of a statistical distribution. Modelers often select a statistical distribution that seems reasonable without sufficient data to test its fit (Kaye and Pyke 2003). In this thesis I will examine the influence of this subjective judgement, and develop an objective criterion for making the selection (chapters four and five).

3.4.2 Density dependence

Density dependence (DD) is the dependence of the population performance on its density (or on any other measure of its abundance). Generally this accounts for resource limitations (food, space, mates, etc) or competition among individuals for available resources. Resource limitation results in reduced per capita share at higher population levels, which in most cases leads to reduced performance of individuals (i.e., reduced contribution to population viability). This creates a negative relationship between per capita performance and population density. In general terms, the presence of any form of correlation (positive or negative) between the population density and any demographic process can be called density dependence (Burgman et al. 1993, p160).

Density dependence is a very broadly defined term having a plethora of inconsistent definitions (Murray 1994). The definition of Reddingius (1971) is based on statistical dependency between population density and the demographic process, which does not imply causation or specific direction of correlation (negative or positive). This has

sometimes been labeled as *statistical density dependence*, to avoid confusion with its causation counterpart (Alsop 1998).

In contrary, Royama (1977) defined the density dependence as the dependence of the net rate of change of a population on the present or past density, with some form of negative feedback process, and a reciprocal causal connection between population density and factors determining it. Here 'negative feedback' implies causation and negative correlation. Supporting this, Eberhardt (1970) emphasize the danger of using correlation based techniques (which do not imply causation) in detecting density dependence. A long lists of works based on both these definitions can be seen in the literature, for example in (Royama 1977).

In agreement with a 'negative feedback' mechanism, density dependent models generally imply inverse relationships between population performances and density (Varley et al. 1973). Remarkably this excludes the Allee effect from density dependence[12]. However, by density dependence most ecologists do not mean to exclude the possibility of the Allee effect (Murray 1994). In agreement with this, Williams et al. (2002, p6) defined density dependence as the functional dependence of vital rates on abundance or density.

The term density does not necessarily mean the population size (section 6.1.3). The majority of models assume density dependent population regulation is governed by a specific segment of the population such as the adult population or the portion of the population breeding in the current year (Reed et al. 1989, Ragen and Fowler 1992). It is important to understand whether the density dependence is restricted to a specific age/stage classes. For example, harvesting density independent classes[13] will not activate the density dependent population regulatory mechanisms (DeMaster 1981). Similarly, density indices derived using components of populations that do not contribute towards the density dependent mechanism could result in the model

[12] If the biology of the species is such that certain factors depressing population growth begin to act more strongly when a population's abundance falls to a low level, the population said to experience *Allee effects* (Burgman et al. 1993), or depensation (Hilborn and Walters 1992). The loss of group effect in defense, and difficulty of finding mates could be examples. Excluding this positive density dependence from models (if it is present in nature) can greatly underestimate the extinction risk of small populations.

[13] Here the term *density independent* is related to the degree that these classes show density dependent changes rather than to absolute density independence (Ragen and Fowler 1992).

leading to erratic management decisions (Ragen and Fowler 1992). Since there is no consensus agreement on what is meant by the terms density and density dependence, care should be taken when comparing results from different studies (Murray 1994).

3.4.2.1 Representing density dependence in population models

Krebs (1991) considered the density dependent paradigm as descriptive, a posteriori, and not leading to an understanding of the process, because no mechanisms are specified. Contrary to this, it is commonly accepted that density dependence is an essential component in understanding the underlying process behind population dynamics. Density independent models assume the dynamic process is independent of the changes in population size implying that any changes in the dynamic process can be attributed to other factors such as environmental variation. As a result density independent models will eventually predict higher sensitivity to environmental changes, and higher risk of extinction due to environmental changes (Morris and Doak 2002, p122).

Density dependence is generally represented in population models by specifying one or more of its vital rates (or growth rate in case of count-based models) as functions of population density[14]. Although the functions used in early models assume linear relationships (Hutchinson 1978), there is a trend of recognising the non-linear nature of this relationship with more and more vital rates (Ragen and Fowler 1992, Williams et al. 2002). The functions in use represent either a positive or negative relationship, and cover a range of shapes such as exponential, hyperbolic, power, and logarithmic (Runge and Johnson 2002). Polynomial functions which cover both positive as well as negative relationships at different levels of density are also in use, albeit rarely (Ragen and Fowler 1992, Murray 1994).

Since it is impossible to know what mathematical function best describes the form of the density dependence response operative in nature (DeMaster 1981), selection of a function is either based on guess work or, at best, based on a statistical fit to the

[14] Therefore, unlike count-based models, demographic models have the flexibility of modelling different vital rates using separate functions.

observed data. Chapter six of this thesis deals with the problems associated with this selection. It also develops an objective criterion to evaluate the suitability of a given function. That criterion is used in chapter seven to evaluate a spectrum of commonly used functions. In chapter eight I investigate the possible effects on population predictions by the subjective selection of a function. There I propose a relatively function-insensitive method of modelling density dependence that can be used with some forms of data.

3.4.3 Other characteristics

The later chapters of this thesis mainly focus on incorporating environmental stochasticity and density dependence into population models. These two characteristics are prominent in virtually all populations, and are required to represent in most models. In addition, there is a long list of other characteristics of populations that are worth having in models. The relative usefulness of each of these characteristics is population-specific. Here I will have a brief look at those characteristics that are generally believed to be more important in population models.

3.4.3.1 Human activities

Human activities on animal populations take various direct and indirect forms such as harvest of species of commercial interest, incidental bycatch, and unintentional degradation of the environment. It is rare to see modelling of unquantifiable activities such as environmental degradation included directly in population models, even though assessments of such activities on populations are not uncommon (e.g., Lusseau and Higham 2004, Neumann and Orams 2005). Animal harvest models have a long history; in fact most management models used today were originally developed as simpler harvest models. Models intended for conservation of animals explicitly include bycatch into the dynamic process as an additional form of mortality (e.g., Breen et al. 2003).

3.4.3.2 Catastrophic events

Although catastrophic events such as severe disease outbreaks and earthquakes can be considered as extreme environmental variations, the methods used to model environmental variation are not generally adequate enough to account for variations of this nature. One way of modelling them is to model catastrophic events and environmental stochasticity separately, and join both together using a probability of catastrophic years (e.g., Hilborn and Wade 1999, Breen et al. 2003). Even though catastrophic events are generally considered as causes for population decline they can sometimes enhance their performance. For example, plant populations have been observed to decline with frequent fire, but their performance can be enhanced by infrequent catastrophic fire (e.g., Manders 1987, Hoffmann 1999). This is because of the differences in growth, survival, and reproduction of the smaller size classes produced by fire (Silva et al. 1991).

3.4.3.3 Heterogeneity in population

Individuals in populations are not homogeneous. All animal populations, including mammals and invertebrates, pass through a series of stages in their life (newborns, juveniles, breeders, postbreeders etc). In addition, individuals are dissimilar to each other even within the same stage. The individual based models (section 3.3.2) treat this heterogeneity in an excellent way by accounting each individual separately and tracking through all events (birth, growth, reproduction, and death etc). Structured models are population-based alternatives that are intended to handle population heterogeneity. Most structured models are capable of capturing this individual dissimilarity to an acceptable level, although some limitations are given in sections 3.3.5 and 3.4.3.4.

3.4.3.4 Senescence

Senescence, the impaired contribution to population viability at old age, is a prominent characteristic in almost all species[15]. The importance of senescence in population models was recognised half a century ago by Medawar (1952). In principle, structured models are designed to recognize the heterogeneity of the population including accounting for senescence. However in practice, senescence cannot be adequately represented by the population structures used in structured models. For example in age structured models, when vital rates estimates are not available for each age group separately, it is a common practice to use the same set of vital rates for all animals beyond a certain age. This results in a single group for all those ages, the *plus-group,* (hence the model structure turns from a pure age-structure to a combination of age and stage structures)[16]. Surviving animals in the plus group are recycling in the same group. There are at least two problems with this practice.

- Within-group heterogeneity: Contrary to what the model assumes, older animals in the plus group are less likely to contribute to future generations (to population existence) by way of survival and reproduction than the younger individuals in the same group.
- Retaining unrealistically old animals: The model does not impose a maximum age; it allows animals in plus group to live to unrealistically older ages. The proportion of animals in the group who are beyond a realistic maximum age becomes considerably high if the estimated survival rate for that group is high. For example, if 50% of a cohort enters the adult stage and the survival rate for adults (after accounting for within-group heterogeneity) is 0.85, then 1.6% of the cohort remain in the stage after another 20 years. This percentage increases to 5.6% if the adult survival rate is 0.9. The model assumes they are surviving and reproducing at the same rate as others in the group.

Both of these problems are common to stage structured models too; none of them is directly accounted for by the model structure.

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[15] The opposite phenomenon is possible in some situations, e.g., the number of eggs produced by some fish species is positively related to their body size. Since fishes are continuously growing, older individuals are larger, so they produce more eggs than younger adults (Pitcher and Hart 1982). However, since this is an unlikely situation in populations of large mammals I ignore that situation from this thesis.

[16] Such simplified models are common in marine mammal literature (Brault and Caswell 1993). They are good approximations for fully age-structured models for many long lived species if the senescence is not strong (Levin et al. 1996, Heppell et al. 2000).

(A) Within-group heterogeneity in structured models

How can age-based heterogeneity be addressed when information about individuals' age is not retained? For animals in the plus group (or in the final stage in stage structured models), the survival and reproduction rates can be assumed to be decreasing functions of age. These functions need to be estimated if one wants to represent the under-performance of older animals. Such estimation is impossible when no information about the animals' age is retained, and the available information is only an estimate of the vital rate for the whole group. A simple approximation is to down-weight the vital rate estimates for the whole group to represent the poor performance of some of these individuals. There is no consensus on how to down-weight the vital rate estimates; some examples are given by Chivers (1999) and Heppell et al. (2000).

Is such a discount always warranted? This depends on, among other factors, how vital rates were estimated for that group. Suppose, for example, if the survival was estimated using data from younger animals in the group (where the survival rate is generally high), then it makes sense to down-weight the estimate to reflect senescence. For example, in an ongoing study of NZSL population, animals that were marked at birth during the last several years are now being observed as breeders. Those data are being used to estimate the survival rates and reproductive rates for adults. Since it is unlikely that any of them has reached the age of senescence, the estimated vital rates could be considered as overestimations, and so the discounting of these estimates could be justified. On the other hand, if the vital rates were estimated using data from a random sample of adults covering all ages, then the effect of senescence is already reflected in the estimates. In that case another discounting cannot be justified. Data from established mark-recapture studies usually show another characteristic. Because marked animals in such samples tend to be older than the animals in general (Manly 1970, p17), the survival and reproduction estimates from those studies are likely to be underestimated, and so the senescence is likely be represented (or over-represented) in these estimates. This could be the situation with survival estimates obtained from recapturing breeding NZSL females that were branded as breeders years ago. Therefore, in general discounting adjustments are not appropriate for all vital rate estimates in general; the appropriateness for a given vital

rate estimate is dependent on the direction and magnitude of possible bias in the estimate.

(B) Retaining unrealistically older individuals in the model

The above discounting of the vital rates accounts only for the variation of vital rates within the ages of the group, in producing an 'average estimate' for the entire group. It does not account for erroneously retaining unrealistically old animals recycling in the last stage (or in plus group). Because the survival rate of the plus group is age-independent, cohorts persist indefinitely. If the survival rate is high, it can be many years before a cohort becomes so small that it has no influence on population growth.

To partially compensate for retaining unrealistically old animals in the model, the above discounted survival rate can be adjusted again to account for the maximum life span, by multiplying it by $(1-\gamma)$ where γ is the proportion of animals that should leave the group within a year due to reaching the maximum age. In other words, γ is the proportion of animals that progress to the next group if another (hypothetical) group existed in the model covering ages from maximum age to infinity. This hypothetical group is the same as the 'death as a stage' group used in (Caswell and Fujiwara 2004), and γ can be considered as the transition rate to that group (Heppell et al. 2000). The estimation of transition rates is dependent on whether the model is stage-structured, or age-structured with a plus group. Caswell(2001) gave several formulas for estimating transition rates under various conditions that can be used here. With some minor modifications, I use one of those formulas in this thesis to estimate transition rates between stages (formula 2.3 and 5.2).

Unlike the adjustment suggested earlier to account for reduced performance of older animals in the group that are below the maximum age, this adjustment is not required for any vital rate other than survival rate because the aim of the adjustment is only to eliminate retaining unrealistically old animals in the model.

3.4.3.5 Correlation

In this context, correlation means the non-independence of different vital rates (cross-correlation) or the same vital rate between times (auto-correlation). Both of these can exist with varying levels of time lags. None of them imply a causal or directional relationship. Hypothetically, both positive and negative correlations could be created by environmental factors, but in reality positive correlations are more common (Halley 1996, Morris and Doak 2002).

The variations observed in vital rates are composed of two components; process variation (i.e., true variation) and sampling variation. Observed variation (measured as variance, covariance, or correlation) is generally used as an estimator of process variation. Even in the absence of a process correlation it can appear to be present in estimated vital rates as a result of sampling correlation. Suppose if the same raw data were used to estimate different vital rates, those estimates might be cross-correlated. Similarly, if longitudinal data were collected about a vital rate using a single cohort of individuals, the resultant estimates could be auto-correlated.
Inclusion of correlation makes models more realistic. Despite theoretical developments already in place to capture the complex nature of correlation structures (section 4.1.4), it is rare to find examples of such models in practice. Limited data availability often forces modellers to use identically and independently distributed (iid) vital rates even if they are aware of its shortfalls (e.g., Breen et al. 2003). Morris and Doak (2002, p138) suggested a criterion to decide whether iid representation is preferable for a given situation.

The effect of autocorrelation on model predictions is influenced by the nature of the density dependence. When (positive) autocorrelation is present, the effect of environmental variation will be larger, hence density independent models predict a higher extinction probability and a shorter time to reach the extinction. In density dependent models, the extinction risk can either increase or decrease depending on the values of other parameters in the model, and the range over which the strength of autocorrelation is varied. The key determinant for density dependent models is whether the slope of the curve plotting N_{t+1} against N_t at the equilibrium point is

positive or negative (Ripa and Heino 1999). If the slope is positive then positive autocorrelation will increase the extinction risk. If the slope is negative then increasing positive autocorrelation can decrease extinction risk over some range of values (Morris and Doak 2002, p135). Therefore, whether the temporal autocorrelation will increase or decrease the extinction risk depends on whether or not density dependence is operating, and if so, on the 'shape' of the density dependence near the population equilibrium. This shows the importance of representing density dependence with an appropriately shaped function (i.e., linear, sigmoid, etc). The appropriateness of the shapes of various density dependence functions is discussed in chapter six.

For a model with a given form of density dependence, the extent to which model outcomes are influenced by the presence of correlation can be investigated using the principles of uncertainty analysis (section 3.5.2). For example, by comparing outcomes obtained from the model with vital rates that are perfectly correlated and those that are iid, (e.g., Slooten et al. 2000). Such investigations are beyond the scope of this thesis.

3.4.4 Remark on representing population characteristics

So far in this chapter I briefly mentioned some of the population characteristics that are important in building population models. This is in no way an exhaustive list of possible characteristics. For example, I have not discussed the ways of modelling interspecies interactions known to exist in ecosystems. Direct interactions of predator and prey species, and direct or indirect competition between species for resources such as food and space etc are well known to exist between animal species, and can be modelled under the multispecies models (section 3.3.3). I mentioned metapopulation and multisite models as ways of modelling animal movements (section 3.3.6), but details on modelling such movements are excluded from this thesis. I deliberately excluded some population characteristics that are not known to exist in mammal populations even though they are prominent characteristics in some other species (e.g., cannibalism in insects and fish species). Because the importance of

a certain characteristic for the dynamic of a given population is population-specific, the characteristics that are not discussed in this thesis could be more important in modelling some other given population.

The modelling does not intend to represent all population characteristics in the model. The process of modelling involves abstraction and simplification, and thus entails losing detailed information and retaining only the essential characteristics. The whole modelling process can be viewed as a filter, in which the full complement of information passes through the filter and only the system attributes or characteristics that are essential to the modelling objectives are retained (Williams et al. 2002).

Consequently selecting appropriate population characteristics and deciding on suitable forms to represent them in models are not straightforward tasks. Density dependence and environmental stochasticity are commonly included characteristics in population models. Their importance and relevance in all areas of population modelling have been well documented for decades (e.g., Boyce 1977, Sauer 1983). Predictions from density independent deterministic models are remarkably different from predictions from density dependent stochastic models. The former are believed to be so unrealistic that present day models are almost entirely density dependent and stochastic. The importance of these two characteristics evident from that the concept of population regulation being formed around density dependence (Royama 1977), while stochasticity embeds the variable environments into the models. The following chapters of this thesis will focus on representing these two characteristics in models.

Any population model, irrespective of how carefully selected its structural features to represent population characteristics, is always an approximate depiction of the nature. Therefore before using any newly built model for making inferences about the population, it should be subjected to additional scrutiny to ensure it can represent the dynamics of the process adequately and appropriately. In the next section of this chapter we look at how to assess the adequacy of a newly built model in the presence of uncertainty in regard to its structure and inputs.

3.5 The model assessment and interpretation

The initial construction of a model is only the beginning of any modelling process. The subsequent stages in the process involve the assessment of model performance as well as biological interpretation of outputs. Model assessment is required to ascertain whether the model outcomes in fact elucidate the underlying process.

In the majority of cases for which predictions are required, it is not possible to model every element that can be comprehensively justified on the basis of a proven mechanism. Rather, the process of model construction is an attempt to arrive at a parsimonious and robust characterisation of the system under study. The elements of this characterisation will necessarily be phenomenological; they are bits of mathematical machinery that behave in accordance with what is known about the system, with or without constituting an explanation of that behaviour. Model predictions may display extreme sensitivity to apparently minor changes in model specifications, and/or great sensitivity to imprecision in input parameters (Wood and Thomas 1999). Model assessment focuses on these issues.

For any population model, a fundamental concern is how much confidence can be placed in its results or predictions. It is unwise to place a great confidence on its outputs until the model has been tested to determine its accuracy and ability to make reliable predictions (Bart 1995). Generally, two sets of questions have been used to assess this confidence. The first is whether it predictions are consistent with historical observations. In analytical studies, consistency is often judged by the empirical fit (Eberhardt 1981). In numerical studies, historical consistency has become associated with the terms verification and validation. For example, if the population growth predicted by the model is inconsistent with the field observations (or with what could be expected biologically) it implies the model is not a 'valid' reflection of the observations. The second question is, how robust the model's predictions are when alternative forms of the model are used or in the presence of uncertainty in its inputs. This question is generally referred to as uncertainty analysis.

3.5.1 Model verification and validation

The procedures to 'check out' the model, to verify that the model's mathematical structure is what was intended, and its performance reflects the data used in its development, are collectively known as 'model verification' (Law and Kelton 2000). Important components of verification involve cross-checking mathematical formulas and computer codes for accuracy, and input values for appropriateness. This step is often overlooked or downplayed, although it can make a crucial difference in validity and efficiency of all subsequent work (Williams et al. 2002). The structure of a verified model conforms to the investigator's intentions, and its behaviour accords with the data on which it is based, but in no way refers to a correspondence between the model and reality.

The verification ensures consistency of the model with the data used to build it, but not with other available data. Validation is different from verification in that it attempt to ensure consistency between predictions and data other than those used in model development. A true validation of a model would require tracking replicate populations that experienced similar conditions and comparing them with model predictions (Beissinger 2002). This is something far better suited to a laboratory than to the field. In practical terms, validation involves refinements to the model structure in order to strengthen its consistency with historical information, and fitting additional data to the model to test the consistency (Ragen and Fowler 1992). The process of verification and validation consists of an iterative sequence of model testing and refinement, because a verified model may be found to be invalid and as a consequence will need to be refined and verified again, until the investigator is satisfied that the model is properly validated over the pertinent range of operating conditions.

Validation can be seen as a check against 'prediction bias' that arises in statistical regression when models are 'overfitted' to data and therefore produce misleading results when used with independent data (Williams et al. 2002). Unlike the model selection procedure used in regression analysis, where the comparison is done between models, the model validation procedure is used to test the fit of data to a given model. In that sense, model validation can be recognized as an application of

the hypothesis testing, in that the model in question is used as a testable hypothesis wherein model behaviour is compared with predictions other than the reference behaviours it was designed to produce (Williams et al. 2002). Different hypotheses (i.e., different models) with quite different predictions can fit to the same data well (Pascual et al. 1997). Therefore a validated model no way implies a 'correct' model (Morris and Doak 2002, ch 12).

The notion that models should be subjected to additional assessments above and beyond their development is strongly emphasized, precisely because in practice it has often not been recognized (Williams et al. 2002). Given that all models are approximations, Anderson and Burnham (2001) on the other hand have opined that the "concept of validation is of relatively little worth in the empirical sciences"[17]. Population models, especially those used in conservation biology, are rarely tested against independently gathered field data (Beissinger 2002, Belovsky et al. 2002), Armbruster and Lande (1993) is a notable exception to this. These models instead compare model results with those from similar populations or from similar species. This can be seen as an implicit validation. For example, Breen (2003) predicted a maximum possible growth rate of 3% for the NZSL population from their verified model, whereas it is typically about 8-12% for pinniped species. In the absence of a plausible ecological explanation this result could be treated as a sign of an 'invalid' model.

Model verification and validation are insufficient to assess the confidence warranted by a particular model with its specific mathematical structure, assumptions, input parameters, and historical observations (Ragen and Fowler 1992). Given that a model's predictions agree with historical observations, the possibility that the model truly reflects the natural dynamics of the population is a question of conditional probability. That is because an 'incorrect' model may also generate 'valid' results within the range of data (Pascual et al. 1997). The important point is that, confidence

[17] This opinion is based on the way validation is used in empirical sciences, not on its concept. The above description essentially describes a single-hypothesis approach where inferences are tied to an a priori hypothesis (to a single model) which investigation leading to a decision either to reject or provisionally retain the hypothesis. On the other hand, a multiple-hypothesis approach is conditional on a set of a priori hypotheses with associated prior probabilities that sum to one over the hypothesis set. The changes in hypothesis probabilities (learning) are entirely conditional on the members of that set. The single-hypothesis approaches are worth little as their values depend heavily on the nature of the alternative hypothesis.

cannot be determined simply by model verification and validation, but is best determined relative to other valid models.

3.5.2 Uncertainty analysis

The second set of questions used in assessing the reliability of a model is, whether the model represents the dynamics in nature adequately, or in other words, are the predictions merely a consequence of selecting one valid model against other possible valid models? A major limitation of using a model to infer population outcomes without testing the model beyond validation is that there is no formal way of representing uncertainty about the conclusions. Uncertainty arises from two sources. First, input parameter values are rarely known precisely. Second, the model structure is rarely based on a proven mechanism; it only represents an attempt to construct a parsimonious characterisation of the system under study. Because both historical observations and model parameters are often characterized by considerable uncertainty, other models may also pass through the validation process (Morris 1990). When many valid models exist, the confidence that can be placed in a model cannot be determined simply by model validation; the substantial model misidentification that this permits can lead to serious errors in predictions (Pascual et al. 1997).

One of the fundamental problems conservation biologists face when they provide advice to managers is how to characterise and account for the uncertainties which result from measurement errors and model mis-specification. The uncertainty analyses go a step beyond validation. They focus on the responsiveness of model predictions to imprecise knowledge about the model structure (i.e., model uncertainty) and the inputs (i.e., parameter uncertainty), and transform the imprecision to uncertainty in model outputs[18]. This assessment is especially critical when apparently realistic models vary in their management implications (Ragen and Fowler 1992). For example, the risk of population decline has been found to be very sensitive to the distribution of individuals among age-classes (Burgman et al. 1995), groupings of

[18] Although the methodology of uncertainty analysis is very similar to that of sensitivity analysis (section 3.5.3) where the measures like sensitivity and elasticity are used to infer the possible changes of model outputs to changes in inputs, uncertainty analysis is conceptually different.

data (Eberhardt 1981), and to the precision of vital rates estimates (McCarthy et al. 1995). Failure to adequately account for this uncertainty can have drastic consequences (e.g., Ludwig et al. 1993, Wood and Thomas 1999). Recognition of this is apparent from the literature of all areas of modelling in last decade (Harwood 2000). More attention has been given in the literature to parameter uncertainty than to model uncertainty, although Sainsbury (1988) and Bartholow (1996) are some exceptions.

Even though uncertainty affects the results of population viability analyses, it does not invalidate the use of an uncertain model or uncertain inputs. The correct representation of uncertainty, literally as a frequency of outcomes, is exactly what is needed to inform management (Goodman 2002). In demographic studies uncertainty is treated basically in two phases.

(1) When estimating vital rates and other model parameters. This treatment of parameter uncertainty could be performed during separate prior studies outside the model building process, or as part of fitting the model to data to estimate parameters from the model.

(2) When making predictions about the fate of the population where uncertainty about model structure and input information are transformed to uncertainties in inferences.

These two phases may not be distinguishable when both are taking place as a single process (e.g., Bayesian PVAs).

(A) Treating uncertainty when estimating vital rates

The methods used to estimate vital rates and other model parameters by fitting models to data can be grouped under frequentist methods and Bayesian methods. The interpretations of precision estimates obtainable from those two methods are quite different. The concept of all frequentist methods lies with 'long-run' frequency of statistics under repeated sampling where estimates are obtained as confidence intervals. Such a confidence interval is interpreted as (e.g., the 95% confidence interval) the interval that will contain the true value of vital rate in 95% of a series of repeated experiments. In contrast, posterior distributions produced by Bayesian methods can be used to make probability statements about the parameter, which is

regarded as a random variable. For example, given the model assumptions, a 95% credible interval has the properties that there is a 95% probability that the true value of the vital rate is in the interval, and that values of the vital rate in the interval have the same or higher probability density than any value outside the interval (Wade 2002). Therefore, unlike confidence intervals in frequentist methods, estimates from Bayesian methods have straightforward biological interpretations about the parameter uncertainty. Bayesian methods also allow information from other populations, other species, or from expert judgments to be included in the analysis (Hoyle and Maunder 2004).

Until recent past, the frequentist methods were the most common areas of statistics among population biologists when dealing with uncertainty in parameter estimation (Givens 1999). Although fairly new to population biology (Wade 2002), there is an increasing trend towards Bayesian methods (Ellison 1996, Punt and Hilborn 1997).

(B) Treating uncertainty when making predictions

When making predictions, uncertainty analysis focuses on the population forecasts made by the model in a methodically similar but conceptually different way to the sensitivity analysis (section 3.5.3). Here the structure, assumptions, or parameters of the valid model are varied to reflect the uncertainty in knowledge and to transform that uncertainty to model predictions. The uncertainty associated with a model outcome is a combined effect of all of the uncertainties associated with each vital rate as well as in the model structure. Almost all vital rates in the model are associated with some form of uncertainty, and they follow varying (and unknown) probability distributions. Therefore it is almost impossible to use analytical methods to draw statements about the uncertainty associated with model outcomes, thus simulation methods are employed in almost all situations.

With regards to model uncertainty, the simulations could involve generating inferences using a range of possible variations of the model. This may mean using expert judgments in selecting possible forms of the model and their components. For example, this could include generating population predictions from models with a

various number of possible stages (in stage structured models), with and without certain model components such as stochasticity, and different forms for the same component, such as differently shaped density dependent functions.

Similarly, with regards to parameter uncertainty, various possible combinations of parameter values are used in predicting the probable ranges for population outcomes. For example, Ragen (1995) used numerical simulations to generate frequency distributions of estimates for the model outputs by systematically varying the input parameters and validating simulation results in comparison with historical observations. If the demographic rate estimates are available in the form of confidence intervals (as is the case in frequentist methods), one intuitive approach is to select several fixed points for each vital rate covering their individual confidence intervals, and use all possible combinations of them in the population projection (e.g., Manly and Walshe 1999). One may opt to exclude any identifiable non-realistic parameter combinations from this analysis. If the estimates of input parameters (vital rates) are available in the form of probability distributions (this is the case in Bayesian estimation), then values for input parameters can be sampled from those distributions (e.g., Caswell et al. 1998, Slooten et al. 2000). This resampling from probability distributions is analogous to resampling data from parametric distributions in parametric bootstrapping (Davison and Hinkley 1997), even though what is sampled here are the parameters of the model rather than data. In this way the population outcome is estimated as a probability distribution rather than as a confidence interval.

(C) Integrated modelling

The input parameters and vital rates used in population models are often piecemeal, they come from separate studies and analyses, and sometimes without an attempt to check their compatibility. The aim of recent studies has paid attention to integrated analysis of different types of census and demographic data, paving the way for integrated population models which combine all information (and uncertainties) into a single model (e.g., Besbeas et al. 2002, Besbeas et al. 2003, Breen et al. 2003, Besbeas et al. 2005).

On this basis, Thomas et al. (2005) described a unified framework for embedding uncertain stochastic dynamics where uncertainties in inputs, including both model uncertainty and parameter uncertainty, translate into precision estimates on outputs, to generate model outputs as statistical inferences. When a number of models are specified a priori, and these are given prior weights, the joint likelihood is used to estimate posterior probability of different models given data. Such integrated models are becoming common in various fields, e.g., Elliott and Little (2005) in human demography, Breen et al. (2003) and Hoyle and Maunder (2004) in marine mammals conservation, Freeman and Crick (2003) in conservation of birds.

Bayesian analysis based integrated models attempt to include all relevant data for a population into one analysis by combining analyses, sharing parameters, and simultaneously estimating all parameters, using a combined objective function. It ensures that model assumptions and parameter estimates are consistent throughout the analysis, that uncertainty is propagated through the analysis, and that the correlations among parameters are preserved (Hoyle and Maunder 2004). Perhaps the most important aspect of Bayesian integrated models is the way it considers the system as a whole, so that inconsistencies can be observed and resolved, and all forms of uncertainties are accounted for in a unified framework (Punt and Hilborn 1997).

3.5.3 Sensitivity analysis

Sensitivity analysis is usually used to investigate the responsiveness of the outputs of a model to variations in its inputs. It is so common in population biology that it is now rare to see a published report on model development on population growth without sensitivity analysis (Horvitz et al. 1997). This analysis can be extended to investigate the responsiveness of model outputs to both model structure and model inputs. Structure, assumptions and/or input parameters of a valid model are investigated to determine which elements most strongly influence the predictions. However, this analysis is frequently limited to a small number of alternative structures in which only a single element of the original model is adjusted at a time, thereby failing to account for the interactions among those elements. Hence its usefulness as a measure of

sensitivity to model structure is somewhat limited (Smith and Polacheck 1984). This is in contrast to its widespread use in examining the responsiveness of outputs to input parameters.

In its most common use (i.e., when related to sensitivity to the parameters) this analysis uses quantitative measures such as *sensitivity* and *elasticity* (Caswell 2001) to quantify the responsiveness of the model predictions to changes in model parameters. Deterministic transition matrix based population models provide a simple, eigen-based analytical way to assess the sensitivity of a population to perturbations (Caswell 2001). In analytical calculations, sensitivity and elasticity are treated as derivatives; sensitivity gives the local slope of output (say, growth rate λ) as a function of a vital rate, while elasticity gives the local slope of $\log(\lambda)$ as a function of log of a vital rate. Therefore, none of them may predict the result of large perturbations, depending on the non-linearity in the relationship between λ and vital rates (which is often far from linear). Though the second derivative might improve the prediction, a numerical perturbation analysis approach is more straightforward (Horvitz et al. 1997). With more realistic and complex models, simulation based numerical approaches are commonly used.

Even though the eigenvalue based sensitivity analysis is common for examining the growth rate of deterministic matrix models, the extension of these analytical techniques to population viability is not appropriate, because obtaining analytical solutions for viability models is often complex, if not impossible (Burgman et al. 1993). This is reflected in the literature in that sensitivity analysis of the risk of population decline has not been conducted in the majority of studies published before mid 1990's (e.g., Burgman and Lamont 1992). A logistic regression based numerical sensitivity analysis method is recommended for population viability studies (McCarthy et al. 1995).

Among other things, sensitivity analysis can be used for following purposes.

- To make predictions about the relative effect of various management alternatives before field manipulations or conservation strategies are implemented.

- To make quantitative measures of contributions from different input parameters to output, or to estimate the relative importance of input parameters.
- Suppose uncertainty is known to exist in more than one input parameter, sensitivity analysis clarifies where efforts should be focused in improving estimates of vital rates in order to improve accuracy/precision of the output estimate. All other things being equal, the biggest payoff comes from improving the estimates of vital rates to which the outcome is most sensitive.
- To quantify the effect of environmental perturbations. The responses of different vital parameters to environmental factors are diverse. The growth rate (asymptotic growth rate λ in the case of structured models) is an integrative result of these responses. Therefore λ is frequently used as a measure of the population-level consequences of the effect of the environment on the vital parameters. Here the effect of a treatment factor (environmental variation) on λ is decomposed into contributions from each input parameter of the model.
- Suppose if some environmental differences have produced differences in vital rates, and hence in growth rates among two or more populations. Sensitivity analysis provides measures on how much each of the vital rates contributes to these observed differences between growth rates. Here sensitivity analysis is used as a retrospective analysis even though it is more usual to perform as a prospective form of analysis.
- To evaluate alternative management strategies. Any management strategy alters one or more model parameters in order to achieve its goal. The relative effectiveness of these can be evaluated by their impact on the outcome.
- Genetic variation produces individuals whose vital rates are perturbed from the overall population values. Out of these, natural selection processes choose animals whose perturbations are most favourable (e.g., more capable of defending against predators). Sensitivity analysis tells us which vital rates are mostly subjected to this selective pressure.

Verification and validation aim to refine the model in order to ensure model predictions are consistent with the data used for its construction as well as with other

historical data. Uncertainty analyses quantify the effects of uncertainty in model structure and of input parameters on model predictions. Sensitivity analysis helps in measuring responsiveness of model outcome to model structure and to input parameters, and hence in analysing for various management options. However, neither model verification/validation nor the uncertainty/sensitivity analysis implies searching for the 'best' model for the system.

In this chapter we briefly looked at various aspects of population modelling. The focus of the rest of the thesis is to review how some of the population characteristics discussed in this chapter, specifically the environmental stochasticity and density dependence, can be incorporated into model building. As an overview of model building, this chapter included some sections (sections 3.4.3 onwards) that are not directly relevant to the main focus of the thesis. However, this general overview provides background information as to where the above two characteristics fit into the modelling framework. Although this thesis does not consider in great detail how other characteristics can be incorporated in to the modelling process, it in no way implies that their importance is secondary.

In the next two chapters we look at modelling environmental stochasticity in population models, followed by three chapters on density dependence. Specific reviews with respect to the modelling of these two characteristics are given in chapters four and six respectively. Different approaches to modelling them are discussed and compared, and some possible improvements are suggested.

4 Environmental Stochasticity

4.1 Introduction

Environmental stochasticity is the temporal variability in the dynamics process. No population behaves exactly the same way throughout; they all are subjected to temporal variations either caused by internal factors, or as responses to external factors such as environmental fluctuations. This chapter deals with the representation of environmental variability in population dynamics models. In particular, I address the question of how different assumptions about the probability distribution of the stochastic variation can be built into population models.

4.1.1 Importance of environmental stochasticity in population models

Although deterministic population models have the advantage that they are simple and in most cases can be handled analytically (McCallum 2000), they are poor in representing realism. The outputs of population viability analysis (PVA), such as probability of extinction, can be highly sensitive to the presence of stochasticity, even in cases where stochastic variation is small (Shaffer 1987, Engen et al. 2001). Ignoring environmental stochasticity may overestimate the precision of population predictions (Engen et al. 2001), and population persistence (Soulé 1987, Caswell 2001), while underestimating the risk of extinction (Burgman et al. 1993), even if demographic variation and uncertainty about the demographic rates have been taken

into account. The purpose of this chapter is to investigate ways that environmental stochasticity can be represented in population models.

This chapter has three main sections. The first section is an introduction about representing environmental stochasticity in population models. It also presents some important characteristics of populations (and their limitations) that are represented within the framework of environmental stochasticity. The second section introduces statistical distributions that are in use, or can be used, for this purpose. The last section concentrates on the properties that can be used to gauge the suitability of those distributions. In this way, I present a biologically acceptable, practical, and objective way of selecting one statistical distribution over others to represent environmental stochasticity in models.

4.1.2 Stochasticity as distinction from uncertainty

Before any discussions of environmental stochasticity can take place it is necessary to understand what exactly is meant by that term. Environmental stochasticity refers to actual variations in environmental conditions that are represented as variations in vital rates (birth rate, death rate etc). In addition, ecological data and the parameter estimations derived from them are usually subjected to a quite different form of variation, termed observation error or uncertainty. We need to distinguish environmental stochasticity from uncertainty in vital rates. Uncertainty is the ignorance or imprecision of the knowledge, and does not represent actual variations that occur on those rates over time (section 3.5.2). For this reason, unlike stochasticity, uncertainty in parameter values affects only the confidence that can be placed in population predictions, but not the actual fate of the population (Engen et al. 2001)[1]. The purposes of representing stochasticity (process error) and uncertainty (measurement or observation error) in population models are quite different, and

[1] As an example, interval estimates produced by most estimation procedures are based on the sampling distribution of the parameter, and so they are a measures of precision by which the parameter is known (uncertainty), rather than a description of how the value of the parameter varies between individuals (demographic variation), or through time and space (environmental variation), (Lebreton and Clobert 1993). Inclusion of uncertainty in a population model transforms the uncertainty in input parameters to uncertainty in population outcome, but it no way represents the possible variation in population outcome.

hence need to be dealt with separately (McCallum 2000). The uncertainty was discussed in section 3.5. I intend to focus only on stochasticity in this chapter.

Environmental stochasticity is generally represented in population models as temporal changes in vital rates (Nakaoka 1997). The observed temporal variation of a parameter (e.g., standard deviation of survival rate estimates between years) is commonly used as a measure of environmental stochasticity. Because this variability includes environmental stochasticity as well as measurement error, those measures tend to overestimate the true variation due to environmental factors. Gaston and McArdle (1994) described several methods of analysing variability in the context of abundance data. Several approaches have been proposed to separate uncertainty from stochasticity in order to account them separately in models (e.g., Burnham et al. 1987, Gould and Nichols 1998, Wade 1999, Akcakaya 2002, Barker et al. 2002)[2]. Discussion on such analyses is beyond the scope of this chapter. For the rest of the chapter I assume an estimate for the environmental variability (e.g., mean and standard deviation of the true variability of the vital rate after removing the sampling variability) is available, and the focus will be on how it can be expressed best in population models. Here I will review the distributions commonly in use in representing environmental stochasticity in vital rates and derive conclusions on their suitability. I basically focus on survival rate, a vital rate always on the unit range, although the ideas can easily be used for vital rates over other ranges.

Unpredictable catastrophic events such as severe disease outbreaks can be considered as extreme environmental fluctuations. In modelling the NZSL population these events have been dealt within the framework of modelling environmental stochasticity (e.g., Hilborn and Wade 1999, Breen et al. 2003). This requires introduction of an additional parameter, the probability of catastrophic years, which cannot be estimated from available NZSL data. In this chapter I will distinguish these catastrophic events from the variations due to 'ordinary' environmental factors.

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[2] Burnham et al. (1987) described a variance-components approach to the separation of sampling variance from total variance. Gould and Nichols (1998) demonstrated the ease and importance of this approach with case studies. Barker et al. (2002) used errors-in-variables regression to separate measurement error. Akcakaya (2002) proposed a method to isolate environmental stochasticity in vital rates from observed variability by estimating and subtracting demographic variability. Engen et al. (1998) proposed a method to estimate the separate contributions from demographic and environmental stochasticity to variance in the population growth rate.

4.1.3 Importance of modelling stochasticity beyond mean and variance

A commonly used analytical formula for estimating approximate stochastic growth rate (Tuljapurkar 1997) contains the mean and variance of stochastic variation, but not the actual distribution of the variation. This implies that, depending on how good this approximation is, the mean and variance of the stochastic variation are important in predicting the fate of a population, but that the distribution itself is less important or not important. However, the same mean and variance can be achieved for a vital rate by having quite differently shaped distributions such as unimodal and J shaped. If the vital rate varies as a unimodal distribution it has a low probability of realising values near its upper boundary, while if the distribution is J shaped this probability is very high. Therefore it is unlikely for a prediction from a population model or subsequent PVA to be independent of the distribution chosen. Most modellers seem to choose a distribution without justifying the choice. Therefore it is worthwhile to see the effect of choice of distribution on the population outcomes, and develop an objective criterion to select a distribution. This is the objective of this chapter.

4.1.4 Correlated and iid stochastic vital rates

It is widely accepted that vital rates are not independently and identically distributed (iid) in most populations, and hence models with iid variates do not represent the stochasticity realistically. In fact one can argue the representation of cross-correlation (i.e., correlation between different vital rates with or without time lag) and/or auto-correlation (i.e., correlation within the same vital rate between time steps) should be the norm, rather than the exception, in any 'realistic' population model. One of the approaches to incorporate autocorrelation into stochastic modelling is to obtain mean of the vital rate from an autocorrelation modelling, and use that mean in modelling stochastic variability. However, Considering practical limitations, Morris and Doak (2002, p138) and Burgman et al. (1993, p151) presented two different criteria to decide whether iid representation is preferable for a given population. Models such as discrete-state markov chain (DMC) and autoregressive moving average (ARMA) have

been developed to capture the complex natures of the correlation (Caswell 2001, p378). Supporting tools for those models such as methods of generating correlated vital rates are also available in the literature (e.g., Todd and Ng 2001). However, it is rare to find examples of using such models and methods in practice. The reason may be that in real situations, data are likely to be a significant obstacle - i.e, information about correlation structure is lacking. For example, a review of plant recruitment showed that at least a decade's worth of data would be needed before reliable estimates of the mean and variance of recruitment parameters can be made (Clark et al. 1999), and similar information for survival is also required to estimate correlation parameters between the two vital rates. Given these issues, it is not surprising that reliable information about the correlation between vital rates is not available. For this reason, most population modellers are forced to use iid vital rates even though they are aware of the shortfalls (e.g., Breen et al. 2003, in modelling NZSL population). Although uncorrelated vital rates tend to make the model somewhat unrealistic, the magnitude of bias in population predictions due to ignoring correlation relative to other sources of bias such as uncertainty/unreliability present in data or in model structure is unknown. The extend to which a population outcomes influenced by correlation structure can be investigated by comparing population predictions obtained from the model with perfect correlation and without correlation (e.g., Slooten et al. 2000). This thesis does not focus on such investigations.

In this chapter I mainly focus on the statistical distributions that can be used to represent iid variates in population models. It has been shown that, after removing seasonal cycles, most terrestrial localities in continents show little autocorrelation in weather patterns over times longer than one year (Namias 1978, Nicholls 1980, Lande et al. 2003, p16). Therefore, although the usefulness of an iid investigation is unarguably somewhat restricted, it can play an important role in some situations. Before looking at distributions in use for this purpose let us consider some characteristics we expect in such distributions.

4.1.5 Properties to consider when selecting a distribution to represent environmental variation

Inclusion of environmental variation in a population model requires simulating population dynamics under stochastic conditions, which involves regenerating environmental states from a distribution that represents the temporal variability of vital rate(s) as realistically as possible. Often we do not have data to estimate such a distribution, and therefore are compelled to use a distribution that we think the underlying variation will look like. At this stage it is important to see what properties a distribution should have in order to be considered as the distribution to use to represent the stochastic variability.

(A) Simplicity in applicability

Any distribution is defined by its parameters[3], and hence those parameters should be able to be specified (i.e., to be determined) from available information about the stochastic variation of the vital rate. The most commonly available information about the stochastic variation is a series of estimates for the vital rate over a period, or sometimes simply the estimates for the mean and variance of the temporal variation only. In this chapter I consider how this stochasticity can be represented in models assuming that these estimates represent the true stochasticity. It is true that these estimates, especially the estimates of stochastic variance, usually include measurement error. However, the discussion in this chapter is about the ability of various distributions in modelling the mean and variance as intended. If we can show a certain distribution can achieve the intended mean and variance then unarguably that distribution is better than any distribution that cannot, irrespective of whether we have precise estimates or not.

In order to represent the stochastic variability through a statistical distribution, parameters for that distribution need to be specified from the known information (i.e., in terms of the mean and variance of that distribution). For practical reasons, this

[3] Parameters that are used to define the statistical distribution should not be confused with the vital rates that can also be named as parameters of the population dynamics model.

specification and subsequent regeneration of environmental states from the distribution needs to be simple.

Ideally, it should be possible to simulate environmental states (i.e., generate variates) from the selected distribution for all realistic combinations of mean (m) and standard deviation (s), in both iid and correlated forms if desired. There are many algorithms useful in simulating such iid variates from selected distributions detailed in literature, (e.g., Fishman 1973, Kennedy and Gentle 1980, Ripley 1987, Swartzman and Kaluzny 1987, Burgman et al. 1993). At least two methods have been used in the literature to generate correlated stochastic variates (Doak et al. 1994, Gross et al. 1998), but they both have some weaknesses. Todd and Ng (2001) developed a method to eliminate those weaknesses in generating correlated variates from a probitnormal distribution (section 4.2.5). In fact, for most other distributions it is still not known how correlated variates can be generated. In sections 5.6.4 and 5.6.8 I discuss this difficulty in relevance to distributions used later in this chapter.

(B) Statistical validity

The selected distribution should be able to simulate the variation of the vital rate within its required bounds (e.g., between zero and one in the case of survival rate, and non-negative in the case of reproduction). For example, it is impossible to generate such values from some commonly used distributions (e.g., from normal or lognormal distributions) without imposing a truncation.

The selected distribution should also have the ability to simulate the effect of environmental variation by generating data according to the shape of the field data, or to the shape what we think the underlying variation look like (i.e., its shape should be flexible). Here the 'flexible shape' does not refer simply to the possibility of having various shapes, rather it refers to the ability of the distribution to change its shape to suit the variation represented by data. In fact, having a shape that is undesirable in representing stochastic variation is an unacceptable property. The intended shape can be unimodal in most cases. In addition to unimodal shape, multimodal shapes, J shape, and U shape can also be desirable if the information that is available about the

variability suggests so. Distribution can be J or U shaped when the variability is large, or bimodally shaped when good and bad years are involved. Some of these shapes are shown in Figure 4.1. (Note, in this chapter I use the term "J shape" to imply both "J shape" and "inverted J shape"). However, none of those shapes are desirable if they are created unintentionally as a consequence of selecting a distribution. There are some distributions with realised shapes that are very different to those expected by the modeller (see later in this chapter).

The values of vital rates generated from the distribution should have the intended mean and variability. It is easy to imagine that statistical properties of data generated from truncated distributions are not the same as intended. In addition, as we see later, values generated from some untruncated distributions do not comply with intended variability for some combinations of mean (m) and standard deviation (s). Of course, once the possible range of data is specified, the combinations of m and s that could exist within that range under any distribution are automatically restricted. Therefore, before we look at specific distributions, it is reasonable to see which combinations of m and s can exist in a given range.

4.1.6 Maximum possible variability for a given range

The maximum possible variability of a finite random variable is a function of its mean (m) and standard deviation (s), and so the range of the variable limits the set of sensible combinations of m and s that are possible over the range. For any finite random variable, there exists a maximum limit that the variability can never exceed irrespective of what distribution it follows, because the finite range creates an upper limit to how far the variates can spread. Therefore it is impossible, and does not make sense trying to represent a variability that is larger than this maximum possible level. However, the maximum level of variability that can be represented by a given distribution can be smaller than the above possible maximum. In addition, the largest variability of a distribution depends on the shape of the distribution. For example, in cases where there are reasons to believe the variability follows a unimodally shaped distribution then, it does not make sense to represent a variability that is larger than

that of a unimodal distribution in that range. For this reason it is useful to know the largest level of variability for differently shaped finite distributions.

4.1.6.1 Maximum possible variability under any finite distribution

The greatest variance of a random variable occurs, irrespective of the distribution it follows, when all realizations are at the lowest and highest points of its range, i.e., probability distribution takes a U shape. Here, 'lowest and highest' implies the variable has a finite range, and it behaves like a discrete variable at this extreme situation, irrespective of whether the variable itself is discrete or continuous in general. The fractions of the realizations that lie at the lowest point (a) and the highest point (b) are dependent on the relative distances to a and b from the mean (m). It can be shown (see appendix 1) that for this situation m, a, b and standard deviation (s) are related by

$$s \leq \sqrt{(b-m)(m-a)} \qquad \textbf{(4.1)}$$

This gives the maximum possible standard deviation of a finite variable as a function of its mean and range. This function has a maximum when $m = \frac{1}{2}(a+b)$. In that situation, the above formula is reduced to

$$s \leq \frac{b-a}{2} \qquad \textbf{(4.2)}$$

giving the upper limit for the possible standard deviations within a given range. In the case of a vital rate on the unit interval, (i.e., survival rate), $a = 0$, and $b = 1$, and hence the above two formulas reduce to

$$s \leq \sqrt{(1-m)m} \qquad \text{and} \qquad s \leq \frac{1}{2} \qquad \textbf{(4.3)}$$

respectively. Since these formulas specify the maximum level of standard deviation that can exist for any given mean survival rate under any distribution, it does not make sense to use any combination of m and s with any distribution that does not confirm to the above requirement.

4.1.6.2 Maximum possible variability under any finite unimodal distribution

It is possible for vital rates to have stochastic distributions of various shapes. For example, the distribution can take a bimodal shape when the environmental conditions fluctuate between favourable and unfavourable levels forcing vital rate to respond accordingly. It can take a J shape if the vital rate remains close to its possible maximum or minimum level most years with occasional departures. The unimodal shape, where vital rate remains closer to a certain value (mode) in most years and with a lesser possibility of having values away from the mode, is the most common. I will discuss the desirability of certain shapes of distributions later in section 4.3.7. In any unimodal finite distribution, the shape of the distribution becomes flatter with increasing variability, and approaches the uniform distribution at the level of variability that unimodality is lost. Therefore the maximum standard deviation of any finite unimodal distribution should be smaller than that of uniform distribution over the same range, which is $\frac{b-a}{\sqrt{12}}$. Since the mean of the distribution lies exactly at the middle of its range at the largest variability, the s can be as large as $\frac{b-a}{\sqrt{12}}$ only for symmetric unimodal distributions; for skewed unimodal distributions s is smaller. Johnson and Rogers (1951) established that, 'for a random variable, there exists a unimodal distribution if and only if $\left|\frac{m-M}{\sqrt{3}}\right| \leq s$', where m is the mean and M is the mode[4]. Combining these two results, the possible range of s for an unmodal variate over a finite range can be given as

$$\left|\frac{m-M}{\sqrt{3}}\right| \leq s < \frac{b-a}{\sqrt{12}}.$$

This upper limit should be smaller for skewed unimodal distributions. This implies that, in the case of a vital rate over the unit interval such as survival rate, the standard deviation that can exist with a unimodal distribution is smaller than $1/\sqrt{12}$.

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[4] This should not be interpreted as 'a given distribution is unimodal if and only if $\left|\frac{m-M}{s}\right| \leq \sqrt{3}$'. Correct interpretation is 'if this condition is correct for a variable then there will be some unimodal distribution to represent the variable, while if this condition is incorrect then variable cannot be represented by any unimodal distribution'. Correctness or otherwise of this condition is not a test for the unimodality of a given distribution. Therefore, for a given distribution, this is a necessary condition for unimodality, but not a sufficient condition.

In summary, there are certain theoretical limitations to combinations of m and s that can be represented by a specific shape under any distribution. Therefore moving the mean-standard deviation combinations beyond those limits implies a change in shape.

4.2 Statistical distributions in use in representing environmental variation

In recent years, population modellers have turned their attention more to stochastic models that take due care to represent temporal variability by considering a range of values for input vital rates, rather than those that use average values to obtain possible population outcomes. One way of representing observed variation of vital rate in a stochastic population model is resampling vital rate parameters at each time step from a list of observed values. This is analogous to resampling data with a nonparametric bootstrap (Davison and Hinkley 1997). This has the advantage of not relying on any statistical distribution, and it guarantees that all sampled values are possible values. However this requires a long list of estimates for the vital rate to sample from, something that is not available in most cases (we will discuss this later in section 5.6.1). In most models a simple statistical distribution is used to represent the variation in these rates, where parameters for the distribution are estimated (or specified) from observed variation. Then the vital rate for each time step of the population projection is sampled from this distribution as a random variate in analogues to parametric bootstrapping[5]. However, the choice of the distribution is not justified in the majority of studies. It is perhaps based on the modeler's convenience, expert opinion, or previous practice (e.g., Rosenberg and Brault 1993, Hilborn and Wade 1999, Engen et al. 2001). In the case of latest NZSL population model (Breen et al. 2003), the distribution used is entirely new, and no reason or justification is given for that choice. I will consider that distribution separately in section 4.2.6. Although occasionally statistical fit is taken into the consideration when choosing a distribution

[5] Although vital rates are parameters in population dynamics models, in simulation studies they are included as random variables to represent the environmental variation. This random variable is represented by the chosen statistical distribution where parameters for the distribution are specified in terms of observed variation (i.e., in terms of mean and variance of the stochastic variation). Therefore it should not be confused with the parameters of population dynamics model (i.e., vital rates) and the parameters of the probability distribution that describe the variation of the vital rate.

(e.g., Taillie et al. 1995, Nakaoka 1997), this is seldom practical because it requires data over a long period (Clark et al. 1999). For this reason, often there is no explicit or implicit check to see whether the variation introduced by the chosen distribution really represents the variation intended. The aim of this chapter is to see whether the presently used distributions actually represent the intended stochastic variation. I first discuss the properties of some distributions that are commonly used for this purpose, and then explore their suitability.

4.2.1 Normal distribution

One of the most commonly used distributions to represent stochasticity in vital rates is the normal distribution (e.g., Ferriere et al. 1996 for reproduction, Pascual et al. 1997 for growth rate, Wade 1998 for bycatch rate, Manly and Schmutz 2001 for survival). There are at least three advantages of using this distribution. First, it is simple enough to implement in almost any software. Analytical solutions are easy to obtain since the normal distribution theory is well developed. For example, if the reproduction by each stage is normally distributed then the total reproduction is also normally distributed. Second, available information (i.e. mean and standard deviation of stochastic variation) can directly be used as parameters for the distribution. Third, random variates generation is easy and accurate, so it is easy to ensure that the variability of generated variates are the same as intended. Fourth, it is possible to simulate correlated vital rates (e.g., Burgman et al. 1993, p280).

However, this distribution has several severe drawbacks. Most importantly, there is no flexibility on the range of the vital rate. Generated vital rates can go well beyond the acceptable range, for example, beyond zero-to-one for survival rate, or below zero for reproduction. Due to this reason, in most population models, the normal distribution is replaced by a truncated normal distribution (e.g., Manly and Schmutz 2001, Runge and Johnson 2002). Fishman (1973, p228) outlined a general method for generating variates from any continuous truncated distribution. However, it is practically problematic, and to my knowledge no one has used this '*direct*' method to generate truncated normal survival rates. Instead the common way of sampling from this distribution is sampling from an untruncated normal distribution, and discarding all

the values those are below the lower bound and above the upper bound (e.g., Nakaoka 1997). I refer to this implicit way as '*truncating*'. For the sake of practical simplicity, the truncated normal distribution is often approximated by sampling from a normal distribution and replacing values that are below the lower bound by the lower bound, and similarly those are above the upper bound by the upper bound (e.g., Manly and Walshe 1999) for environmental variation in reproduction and in adult survival, (Soulé 1987, p26) for reproduction. I refer to this approximation as '*limiting*'. The appropriateness of truncating and limiting is doubtful.

These restricted distributions (irrespective of the way the vital rates are generated from them – whether directly or by truncation or limiting) add new problems. The mean and variance of the restricted distribution will no longer be the same as intended. Due to the restriction, the variance will always be smaller than intended, while the mean may be higher or lower than intended depending on the symmetry of the truncation (Todd and Ng 2001). The magnitude of the effect of this truncation cannot be considered 'small' unless the mean of the vital rate is close to the middle of its range, and the variance is small relative to the range. For example, in the case of large, long-lived, slow growing mammals (including NZSL), adult survival rate is generally large and away from 0.5, hence the effect of truncation can be assumed to be large. Caswell (2001, p412) gives an example where mean and variance of data generated from truncated distributions differ from intended mean and variance by 25% and 35% respectively. Additionally, the limiting approximation for truncation creates peaks at each boundary. This can alter the shape of distribution remarkably, especially if the degree of truncation is large. Depending on the degree of truncation, the resultant distribution bears little resemblance to the intended distribution. Truncation, even without the limiting approximation, creates knife-edge boundaries for the distribution of the vital rate, the realism of which is hard to imagine. At the cost of the above drawbacks, the truncated normal distributions offer flexibility on the lower and upper boundaries, and hence add skewness to the distribution. However, they do not offer any flexibility to the shape of the distribution within those two boundaries. To my knowledge there has been no attempt taken to correct these problems.

Irrespective of these drawbacks the truncated normal distribution is still in use (Nakaoka 1997). Manly and Schmutz (2001) used truncated normal (with limiting) for survival rate. Raftery et al. (1995) and Givens (1999) used a normal distribution (with truncating) for survival. Runge and Johnson (2002) used truncated normal distribution for both survival rate and reproductive rate.

One might suggest handling some of these problems by starting with a different mean and standard deviation for the original normal distribution (say, μ and σ) so that the truncated distribution will have the required mean and standard deviation (m and s). This method requires specifying μ and σ in terms of required m and s, which is not easy. Cohen (1949, 1950) showed a method of estimating parameters for the truncated normal distribution, and Cohen (1951) generalised it to a series of other distributions. However this method needs a set of sample data (i.e., observations) from that distribution (knowing m and s is insufficient), and the method is not straightforward.

The ultimate problem in using the normal distribution or the truncated normal distribution is the inability to represent the variability as intended, and the lack of flexibility. Once flexibility in range is gained by truncation, it loses the ability to have the required mean and variance. Suppose even if vital rates can be generated from truncated normal distribution with required mean and variance, it still does not have the flexibility in shape of the distribution. Although observed stochastic distributions for many vital rates are often skewed in varying degree, this distribution offers only little flexibility of having such shapes. In addition, there are instances that J shaped or U shaped distributions are more suitable in describing the observed stochasticity. This can happen when observed variability is large compared to the range, or when the mean is very close to a boundary. Such shapes cannot be represented by a truncated normal distribution. For these reasons I will not consider this distribution any further.

4.2.2 Lognormal distribution

The lognormal distribution is commonly used to represent positively skewed stochastic variations such as in reproductive rate and population size (Nakaoka 1997, Higgins et al. 2000, Hilborn et al. 2000, Caswell 2001). Hilborn and Walters (1992,

p264) argue in favor of using a lognormal distribution for random variation in recruitment for situations where animals do not enter the population immediately after birth (e.g., recruiting juvenile fish to the fishable population) even if the distribution of birth itself is not skewed to right. Even though this distribution (after truncation) can be used to represent the stochasticity in survival rate if the survival rate is distributed with a small mean and a long right tail towards one, it is not generally used for survival.

In addition to representing the environmental variability, this distribution is used to represent the uncertainly in parameter values. In case of NZSL population, PBR formula is used to decide the level of MALFiRM (section 2.3.1). This formula uses lognormal distribution to represent the uncertainty in one of the input parameters, the population size estimate.

If $\ln(S-\theta) \sim N(\mu,\sigma^2)$ or equivalently if $S=\theta+e^{\mu+x}$, where $x \sim N(0,\sigma^2)$ then variable S is said to follow the lognormal distribution. Since θ is the lower boundary that variable S can take, it is common to select θ to be zero in order to represent non-negative variables. Therefore, the resultant two-parameter distribution can be given as $S=e^{\mu}e^{x}$, where e^{x} is a lognormal multiplicative error term. Environmental process error is generally added to reproduction in this way (Hilborn et al. 2000). Standard algorithms to generate variates from lognormal distribution are available (e.g., Burgman et al. 1993, p279, Wade 1998, Wade and DeMaster 1999). These algorithms require the mean and variance of the log of the vital rate (μ and σ^2) to be specified in terms of the mean and standard deviation of the vital rate (m and s), which, unlike in some other distributions, can be done analytically.

It is common to use this distribution when a positively skewed distribution is sensible for the vital rate on hand (e.g., fecundity). This distribution has a range from a specified lower limit θ (usually the zero) to infinity, hence generated vital rates can range beyond the acceptable limits. For this reason modellers tend to use a truncated lognormal distribution (e.g., Burgman and Lamont 1992). As stated under the truncated normal distribution, random number generation directly from the truncated lognormal distribution is complicated. Therefore vital rates generation is usually done

by first generating values from the untruncated lognormal distribution, and then discarding any values beyond two bounds (i.e., truncating), or replacing values beyond the bounds by the closest bound (i.e., limiting). In either way, resultant values will not have the intended mean and variance. Also, this distribution does not have the flexibility of having various shapes. Therefore problems associated with the normal distribution are common to this distribution too. Due to these drawbacks, I will not explore this distribution any further.

4.2.3 Beta distribution

The beta distribution is becoming a popular choice in finding solutions to most of the problems encountered in normal and lognormal distributions (e.g., Higgins et al. 2000). Johnson et al. (1994a) offers a large list of beta distribution based modelling applications in a diverse range of fields beyond population modelling. To my knowledge this distribution has not been used for the NZSL population. The distribution has the probability density function (pdf) of

$$f_S(S) = \frac{(S-a)^{\upsilon-1}(b-S)^{\omega-1}}{B(\upsilon,\omega)(b-a)^{\upsilon+\omega-1}}, \qquad \nu,\omega > 0 \qquad \textbf{(4.4)}$$

where, a and b are the lower and upper boundary for the vital rate S, ν and ω are the shape parameters of the distribution, and $B(\upsilon,\omega)$ is the beta function (Johnson et al. 1994a). In the case of survival rate the above pdf is reduced to

$$f_S(S) = \frac{(S)^{\upsilon-1}(1-S)^{\omega-1}}{B(\upsilon,\omega)} \qquad \textbf{(4.5)}$$

which is known as the standard beta distribution (appendix 2). As shown in appendix 3, the parameters for this distribution can be specified in terms of intended mean (m) and standard deviation (s) of the vital rate in the following way.[6]

$$\upsilon = \left(\frac{m-a}{b-a}\right)\left(\frac{(b-m)(\text{m-a})}{s^2} - 1\right) \qquad \textbf{(4.6)}$$

$$\omega = \left(\frac{b-m}{b-a}\right)\left(\frac{(b-m)(\text{m-a})}{s^2} - 1\right) \qquad \textbf{(4.7).}$$

[6] Throughout this chapter I consider the possibility of specifying parameters for various distributions from mean and standard deviation of vital rates. Since mean and standard deviation are usually estimated from raw data in practice, this is an indirect way of seeing the possibility of estimating parameters from field data.

For the standard beta distribution these equations are simplified to

$$v = m\left(\frac{(1-m)m}{s^2} - 1\right) \quad \text{and} \quad \omega = (1-m)\left(\frac{(1-m)m}{s^2} - 1\right).$$

It should be noted here, shape parameters for this distribution can be obtained exactly for any given sensible[7] combination of m and s, so the S values generated using these shape parameters should have the required mean and standard deviation.

This distribution allows the flexibility of fixing bounds at any range by selecting values for a and b parameters. Also, it can take a wide range of shapes including unimodal, J-shaped and U-shaped depending on the values of the shape parameters, which in turn depend on the mean and standard deviation (see Figure 4.1). This distribution is undefined if any of the shape parameters are non-positive. If both shape parameters are greater than one the distribution is unimodal, if both are smaller than one it is U shaped, and if one is greater than one and other is less than one the distribution is J shaped. If the two shape parameters are the same the distribution is symmetrical. The further away that the ratio of the two parameters is from one, the distribution become more asymmetric. Since the v and ω parameters are simple functions of m and s, it is easy to know the shape of the distribution for any given combination of mean and standard deviation.

The shape changes gradually from one form to another with the gradual change in m and s. This offers the flexibility of representing a wide variety of shapes. One should not see this positive feature as a potential problem of instability in the shapes of the distribution, because in those situations a J or U shape is warranted by the variability in data, and is not merely an accident. As stated in section 4.1.6, there are certain theoretical limitations to combinations of m and s that can be represented by a specific shape under any distribution. Therefore moving the combinations of m and s beyond those limits implies a change in shape.

[7] Appendix 1 defines the combinations of m and s of a variable which can be represented by a distribution when the range of the variable is finite. Any combination that cannot be represented by such a distribution is not 'sensible'.

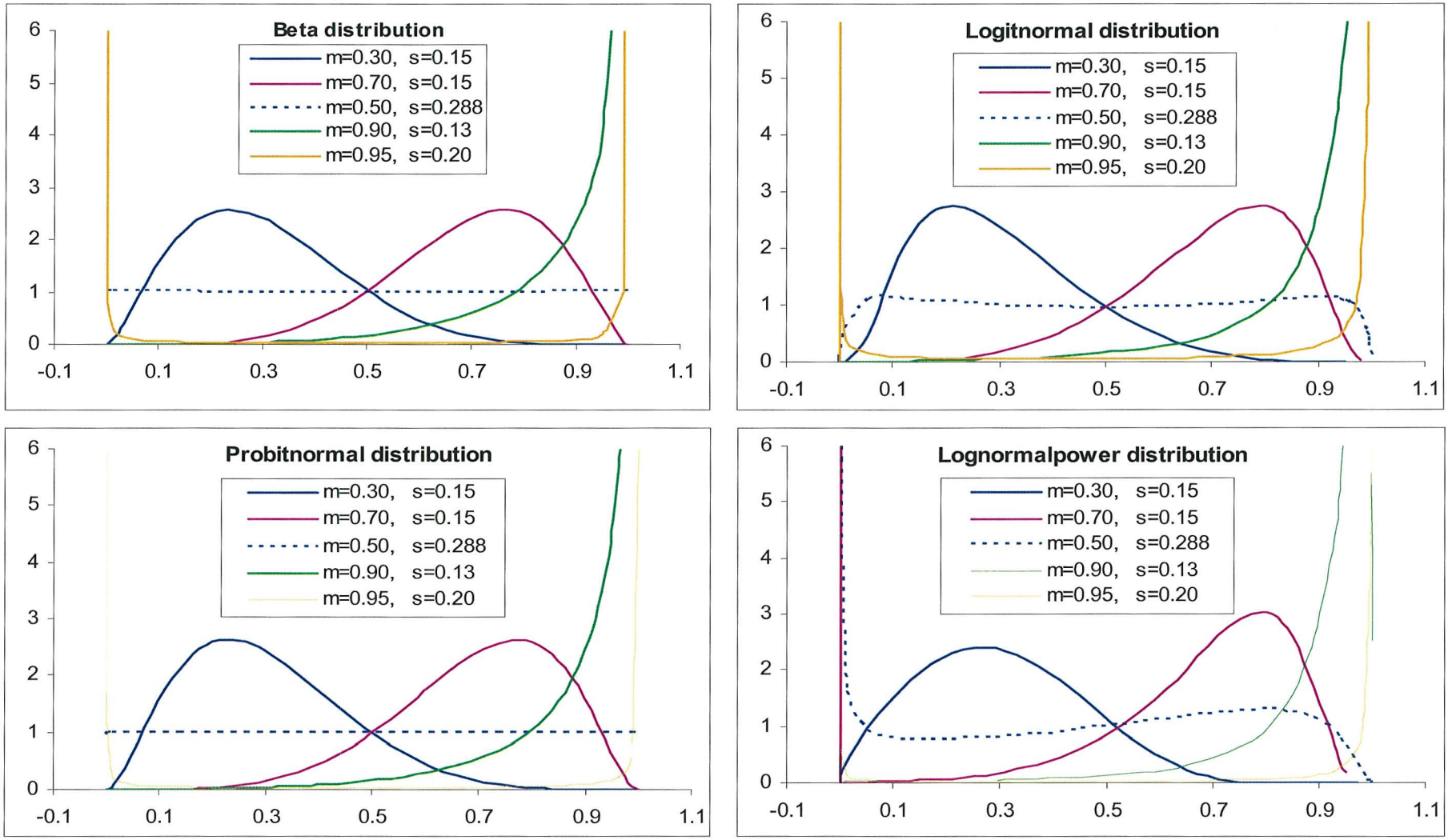

Figure 4.1: Probability density functions of distributions showing possibility of modelling a wide variety of shapes. Mean (m) and standard deviation (s) combinations shown were selected so that when $m = 0.30$ and $s = 0.15$ the beta distribution is skewed to right, when $m = 0.70$ and $s = 0.15$ it is skewed to left, when $m = 0.50$ and $s = 0.288$ it is uniform, when $m = 0.90$ and $s = 0.13$ it is J shaped, and when $m = 0.95$ and $s = 0.2$ it is U shaped. For easy comparison, the same combinations of m and s were used for other distributions.

There are standard methods and algorithms of generating random numbers from this distribution. Some comments about the reliability of some of those generators are given in section 5.6.4.

4.2.4 Logitnormal distribution

The major problems observed with the use of normal and the lognormal distributions in representing environmental stochasticity in survival were that:

- their range is not restricted to the unit interval,
- values generated from these distributions do not have the required statistical properties if truncation is used to restrict the range,
- distributions do not have flexible shapes.

One of the obvious ways of getting around the first two of these problems is to transform normal variates by the inverse logit transformation (instead of truncation). This leads to the logitnormal distribution.

The stochasticity of vital rate S is said to follow a logitnormal distribution if the logit(S) follows a normal distribution, i.e.,

if $\ln\left(\frac{S}{1-S}\right) \sim N(\mu,\sigma^2)$ then S is logitnormally distributed.

Aitchison and Shen (1980) discussed the properties of this distribution, and Frederic and Lad (2005) gave some technical details. Once appropriate values for parameters μ and σ^2 are obtained (or specified), S values can be generated as $S = \frac{e^x}{1+e^x}$, where $X \sim N(\mu,\sigma^2)$. Truncation or limiting is not required here as the S values generated in this way are automatically restricted to the unit interval. It is possible to show (see appendix 2) that the probability density function of S is given by

$$f_S(s) = \left|\frac{1}{s(1-s)}\right| \frac{1}{\sigma\sqrt{2\pi}} \exp\left(\frac{-[\log it(s)-\mu]^2}{2\sigma^2}\right), \qquad \text{where } 0 < s < 1. \qquad \textbf{(4.8)}$$

Similar to the beta distribution, this distribution can take a wide range of shapes depending on the mean and standard deviation used (see Figure 4.1).

(A) Specification of logitnormal parameters (μ and σ) from mean and standard deviation of vital rate

Values for parameters μ and σ need to be specified in terms of the required mean (m) and standard deviation (s) of variate S in order to ensure that the resultant S values will have the required mean and standard deviation. Since there is no analytical way of specifying this (Aitchison and Shen 1980, Frederic and Lad 2005), one of the ways that this can be done is by using the delta method approximation (Stuart and Ord 1994) as

$$\mu \approx \ln\left(\frac{m}{1-m}\right) \qquad \text{and} \qquad \sigma \approx \frac{s}{m(1-m)}.$$

This approximation can be improved by using numerical integration (see section 4.3.6 and appendix 3). However, as observed in this study, depending on the algorithms used, if the s is very close to the maximum possible standard deviation then numerical procedures are likely to fail. As seen from formula 4.1, even a small s can be very close to the maximum possible standard deviation if the mean is close to a boundary.

4.2.5 Probitnormal distribution

This distribution offers another option in modelling vital rates without using truncation, and Todd and Ng (2001) recommended it for generating survival rates[8]. Similar to the logitnormal distribution, the basic concept based on this distribution is to generate random variates from a well known distribution and transform them into the required range (i.e., zero-to-one range in case of survival) using a suitable non-linear transformation. In the logitnormal distribution, random normal variates and the inverse-logit transformation were used. For the probitnormal distribution we use normal variates and the *cumulative distribution function of the standard normal* as the suitable transformation.

A variate S is said to be probitnormally distributed if

[8] The Todd and Ng (2001) recommendation is based on the ability of this distribution to generate correlated vital rates, which is not the subject of this chapter.

$$S \sim \Phi(x),\ X \sim N(\mu,\sigma^2) \text{ or equivalently } \Phi^{-1}(S) \sim N(\mu,\sigma^2),$$

where Φ refers to the cumulative distribution function of the standard normal. It can be shown that the probability density function of S is given by

$$f_S(s) = \phi\left(\frac{\Phi^{-1}(s)-\mu}{\sigma}\right)\frac{1}{\sigma.\phi(\Phi^{-1}(s))}, \qquad 0 \le s \le 1 \qquad \textbf{(4.9)}$$

where ϕ is the density function of the standard normal (see appendix 2).

An equivalent version of this formula is $f_S(s) = f_X(x)\sqrt{2\pi}\exp(x^2/2)$, where $X \sim N(\mu,\sigma^2)$, $S = \Phi(x)$, and $f_X(x)$ is the probability density function of x (see appendix 2). This version is easier to evaluate in Matlab®, and used in producing Figure 4.1. Like the pdf of the beta distribution, the probitnormal pdf can take a wide range of shapes, depending on the values of μ and σ (i.e.; depending on the mean and variance), see Figure 4.1.

It is necessary to specify parameters for the underlying normal distribution (μ and σ) so that when $X \sim N(\mu,\sigma^2)$, $S = \Phi(x)$ has the required mean (m) and standard deviation (s). A numerical method must be used here as μ and σ cannot be specified in terms of m and s using analytical solutions (appendix 3). However, if s is very close to its possible maximum then this numerical procedure is likely to fail (depending on algorithm and software used). Standard subroutines are available in modelling software that can be used in evaluating the $S = \Phi(x)$ transformation at the stage of generating random values from this distribution (e.g.; *normsdist* function in Excel, *erf* function in Matlab®).

4.2.6 Lognormalpower distribution

The lognormalpower distribution is new in representing environmental stochasticity. Hilborn and Wade (1999) proposed this distribution to represent the environmental variability of the survival rate of the New Zealand Sea Lion population (the motivating population introduced in chapter 2). Although this particular population model was not used for managing NZSL, Breen (2003) used the same distribution for

the same population. To my knowledge this is the only population model that has used this distribution. In fact this use was the entire motivation for the investigation in the present chapter (i.e., investigating the suitability of various distributions to represent environmental stochasticity). Therefore, in addition to the NZSL serving as a motivating example population, lognormalpower distribution also serves as a motivating example distribution for this study.

In the lognormal distribution (section 4.2.2), environmental process error is added through a lognormal multiplicative error term. Although the lognormal distribution is commonly used in modelling reproduction, it poses problems when dealing with the survival rate, as the multiplicative term can often push the survival rate beyond the zero-to-one range. Hilborn et al. (1997) attempted to correct this situation by using a lognormalpower term (instead of the multiplicative error term). The motivation for this approach comes from the concept based on logitnormal and probitnormal distributions where random normal variates are transformed to unit range using a certain transformation. Hilborn et al.(1997) modeled the vital rate S as

$$S = A^{\exp(x)}, \text{ where } X \sim N(0,\sigma^2) \text{ and } 0 < \mathrm{A} < 1.$$

Here σ signifies the amount of stochastic variation, but it is not the standard deviation of the stochastic variation, which is smaller than σ. I refer to this distribution as the lognormalpower distribution. That is, S is Lognormalpower distributed if

$$\ln\left(\frac{\ln(S)}{\ln(A)}\right) \sim N\left(0,\sigma^2\right) \qquad \text{where } 0 < \mathrm{A} < 1.$$

It is possible to show (appendix 2) that the pdf of S is given by

$$f_S(s) = \left|\frac{1}{s\ln(s)}\right| \frac{1}{\sigma\sqrt{2\pi}} \exp\left(-\left\{\ln\left[\frac{\ln(s)}{\ln(A)}\right]\right\}^2 \Big/ 2\sigma^2\right), \quad \text{where } 0 < s < 1. \quad \textbf{(4.10)}$$

It may be noted that like the logitnormal distribution, even though $f_S(s)$ is undefined when S = 0 or S = 1, it will not be an obstacle with a continuous variable like S. Similar to the other distributions discussed above, this distribution can take a wide variety of shapes (see Figure 4.1). The distribution is unimodal when s is very small (i.e., when σ is very small).

(A) Specification of lognormalpower parameters (A and σ) from mean and variance of vital rate

Values for parameters A and σ need to be specified in terms of the required mean (m) and required standard deviation (s) of variate S in order to ensure that the resultant vital rates will have the required mean and standard deviation. Using the delta method they can be approximated as

$$\sigma = \sqrt{\frac{s^2}{[m\ln(m)]^2}} \qquad \text{and} \qquad A = m.$$

It was observed that this approximation is not suitable for some combinations of m and s, as the variates generated by using this approximation will have a mean and variance that are quite different form those intended, especially if the intended s is large (see section 4.3.6). It is not easy to improve this estimation procedure by using more terms from the Taylor series expansion because the involved formulas are difficult to solve for A and σ. Using numerical integration we can improve the estimation (see appendix 3). However, similar to the logitnormal and probitnormal distributions, if s is very close to its possible maximum then this numerical procedure is likely to fail (depending on the algorithms and software used).

4.3 Comparing suitability of distributions

The properties of distributions worth considering in representing the environmental variation were stated in section 4.1.5. These properties include,

- the simplicity in obtaining parameter values of the distributions, and in generating random values from the distribution (including the ability to use analytical methods and simple software),
- the ability to use all realistic combinations of mean and variance,
- the compatibility of vital rates generated from the distribution with intended statistical properties (i.e., with required mean and variance),
- having a shape that is desirable, flexible, and intended[9].

[9] In addition to the properties mentioned here, there are more properties that are important such as the ability to achieve the required range and required correlation structure. I will ignore them until section 5.6.

In this section I compare the last four distributions on these properties, excluding normal and lognormal distributions for reasons given earlier.

4.3.1 Ability to specify parameters using available information

Whatever distribution is used to represent the stochasticity, stochastic vital rates generated from that distribution should be in consistent with the available knowledge about the variation of vital rate. Therefore, it should be able to estimate (i.e., specify) parameters for the distribution from the available information about the stochastic variation. Information about the environmental variability is available in most cases either in the form of a list of observations, or as a mean and standard deviation obtained from analyses such as mark-recapture. Therefore, we limit our discussion to the case where the estimates for mean and standard deviation of the stochastic variation are available. It should be remembered here that the uncertainty present in these estimates needs separate attention as stated in sections 3.5 and 4.1.2.

Parameter values obtained using the methods in appendix 3 for different combination of mean and standard deviations are presented in appendix 4A to 4D respectively for four distributions. (See the first 5 columns. The third column shows the variability in another measure that we discuss in section 5.6.9). Mean values used in these tables were selected to be spaced between 0.5 and 1.0, which is the most likely range for large mammal's survival rate. Standard deviations were selected to cover most of the realistic range with each mean value.

Table 4.1(a) summarises the comparison of the methods for obtaining the parameter values for these four distributions, given the mean and standard deviation. It should be noted that, as seen in section 4.2, the beta distribution is the only distribution (out of the distributions considered here) where analytical methods can be used, and parameters can be specified exactly. For other distributions, numerical methods are needed to be employed to obtain parameter values with required accuracy. The disadvantages of numerical methods are that their convergence can be software dependent, and that they are unable to specify the parameters with exact accuracy.

Table 4.1: Specifying parameters for distributions from mean and standard deviation of stochastic variation, and generating data from those distributions

(a) Obtaining parameter values

Distribution	Parameters	Method of specifying	Approximation methods	Comments
Beta	ν and ω	Analytic	Not applicable	Exact accuracy obtainable.
LogitNormal	μ and σ	Numeric	Delta method. Not good when s close to its possible maximum.	Accuracy is not exact. Numerical procedure likely to fail when s close to its possible maximum. Not easily implemented in spreadsheets. Slow to implement.
Probitnormal	μ and σ	Numeric	None known	Accuracy is not exact. Numerical procedure likely to fail when s close to its possible maximum. Not easily implemented in spreadsheets. Slow to implement.
Lognormalpower	A and σ	Numeric	Delta method. Not good when s close to its possible maximum.	Accuracy is not exact. Numerical procedure likely to fail when s close to its possible maximum. Not easily implemented in spreadsheets. Slow to implement.

(b) Generating random variates

Distribution	Method
Beta	Use ν and ω parameters to generate beta variates over the unit interval using available algorithms (e.g., Johnk 1964 and Ahrens & Dieter 1974, *betaval* function in (Morris and Doak 2002)), or using standard software (e.g.; Matlab, Poptools)[10].
Logitnormal	Generate normal variates with estimated μ and σ parameters using standard methods and use inverselogit transformation.
ProbitNormal	Generate normal variates (x) with estimated μ and σ using standard procedures. Then transform them to unit interval by taking $S = \Phi(x)$ transformation.
Lognormalpower	Generate normal variates with estimated A and σ parameters and use lognormalpower transformation.

[10] Reliability of these beta generators will be discussed in section 5.6.4.

Numerical integration is not easy to implement in simple software like spreadsheets, and demands purpose written codes. Compared to analytical methods, numerical integration methods require significantly larger computing time to run population dynamics models. The delta method can be used as an analytical approximation to the numerical methods. The reliability of this approximation is discussed later in section 4.3.6.

4.3.2 Generating vital rates from the distributions

Methods for generating random variates from these four distributions are summarized in Table 4.1.(b). They involve using beta generators (for the beta distribution) and normal generators (for other distributions) with the use of transformations. Reliable random number generators to produce normal variates and beta variates are readily available[11]. Two of the transformations (inverse logit and lognormalpower) are very simple and can be implemented easily. There are standard functions available in software to use for the probit transformation, for example the *error* function in Matlab® as stated in section 4.2.

4.3.3 Ability to use all sensible combinations of mean and standard deviation

As shown earlier (section 4.1.6), if the combinations of mean (m) and standard deviation (s) do not satisfy the inequality $(b-m)(m-a) \geq s^2$ then those combinations cannot exist in the finite interval a-to-b with any distribution. Therefore it is not sensible to try and to fit a combination of m and s that does not comply with this relationship. (If one does have this sort of data, the reliability of the data needs to be investigated before attempting to use them in a population model). Note that this formula also implies s values as large as 0.5 can be represented for survival rate. Since this is a very large variability when considering environmental stochasticity, one

[11] I compare the reliability of some commonly available beta generators in section 5.6.4.

might argue that it is unnecessary to look at the possibility of using a variability that is close to its possible maximum. However, the above formula also says that when the mean becomes closer to a bound, the maximum possible value of s gradually approaches zero. For this reason, even very small s values can be very close to the maximum possible. Therefore it is necessary to look at the possibility of using variability close to its possible maximum especially when the mean is close to a boundary.

It will be seen later (in appendix 5) that the relationship between the m and the maximum possible s for the beta distribution is the same as the relationship given in the preceding paragraph. This implies that if a distribution exists with a given combination of m and s over a finite interval, then that combination can be represented by the beta distribution over the same interval. Therefore the whole sensible range of m and s combinations can be modelled by the beta distribution.

Unlike the beta distribution, not all the combinations of m and s can be modelled with the other three distributions, even though they are all defined over the entire range of m and s combinations that the beta distribution is defined for (except that density function of logitnormal and lognormalpower are undefined at s = 0 and s = 1). Appendices 4B to 4D appear to suggest that all these distributions can be used (i.e., parameter values can be obtained and the variates can be generated) over almost the entire range of m and s combinations. However, it is observed that the numerical procedures involved in parameter specification can fail or be inaccurate when s is very close (closer than what are shown in appendix 4B to 4D) to its possible maximum. It may be that such '*very large*' s do not occur often (even if theoretically possible), and hence the above difficulty with numerical procedures poses no problem in practice[12]. (That may explain why this problem is not apparent in Figure 4.2, which is introduced in section 4.3.5). This difficulty with numerical procedure may not be a problem if and when better ways of estimating parameters are developed.

[12] Mean survival rate will often be close to upper bound, and the standard deviation as estimated from field data often large for large mammals. However, those mean-variability combinations are still confined to the $(b-m)(m-a) \geq s^2$ relationship. Here 'very large' implies s is almost at its possible maximum level.

4.3.4 Reliability of parameter specification

Since it was observed in section 4.3.1 that the numerical integration procedure might not produce distribution parameters accurately when s is close to its possible maximum, it is interesting to check the accuracy of parameters produced by these methods. For this purpose I used numerical integration again to find the mean and standard deviation of the distributions defined by the parameter values obtained by numerical methods (columns six and seven in appendix 4B to 4D). If the numerical methods produce parameter values accurately then the mean and standard deviation found in this way should be same as those intended (given in columns one and two). It can be seen that, on the basis of achieving the required mean and standard deviation, the parameter specification is good for all distributions when s is small. However, when s is large the variability represented by estimated parameters is slightly biased. Also, it should be noted that although the s levels covered in those tables are 'large' enough to represent environmental stochasticity, they are not 'very close' to the possible maximums (with the given means) to the extent that numerical procedures do not work.

4.3.5 Ability to simulate variability as intended

One of the properties we expect from any distribution is the ability to generate vital rates with the required mean and variability. This may not be always the case due to possible biases in parameter specification, or errors in random number generation caused by limitations in the software or algorithms used. In the interest of exploring this possibility I generated random variates[13] for each combination of intended m and s (used in section 4.3.1) for each distribution. The mean and standard deviation of these variates were given in columns 8 and 9 in appendices 4A to 4D. They are summarized in Figure 4.2 for easy comparison with those intended. It seems all distributions perform fairly well in terms of parameter specification and variates generation, even though their performance is not exact when s is large.

[13] Here I generated 2000 data values for each combination of intended mean and s. I calculated the standard errors of the mean of generated data, and found they all are within 1% even at the highest level of s used (i.e.; s = 0.45), and therefore it was assumed 2000 data values is sufficient to control sampling/simulation error.

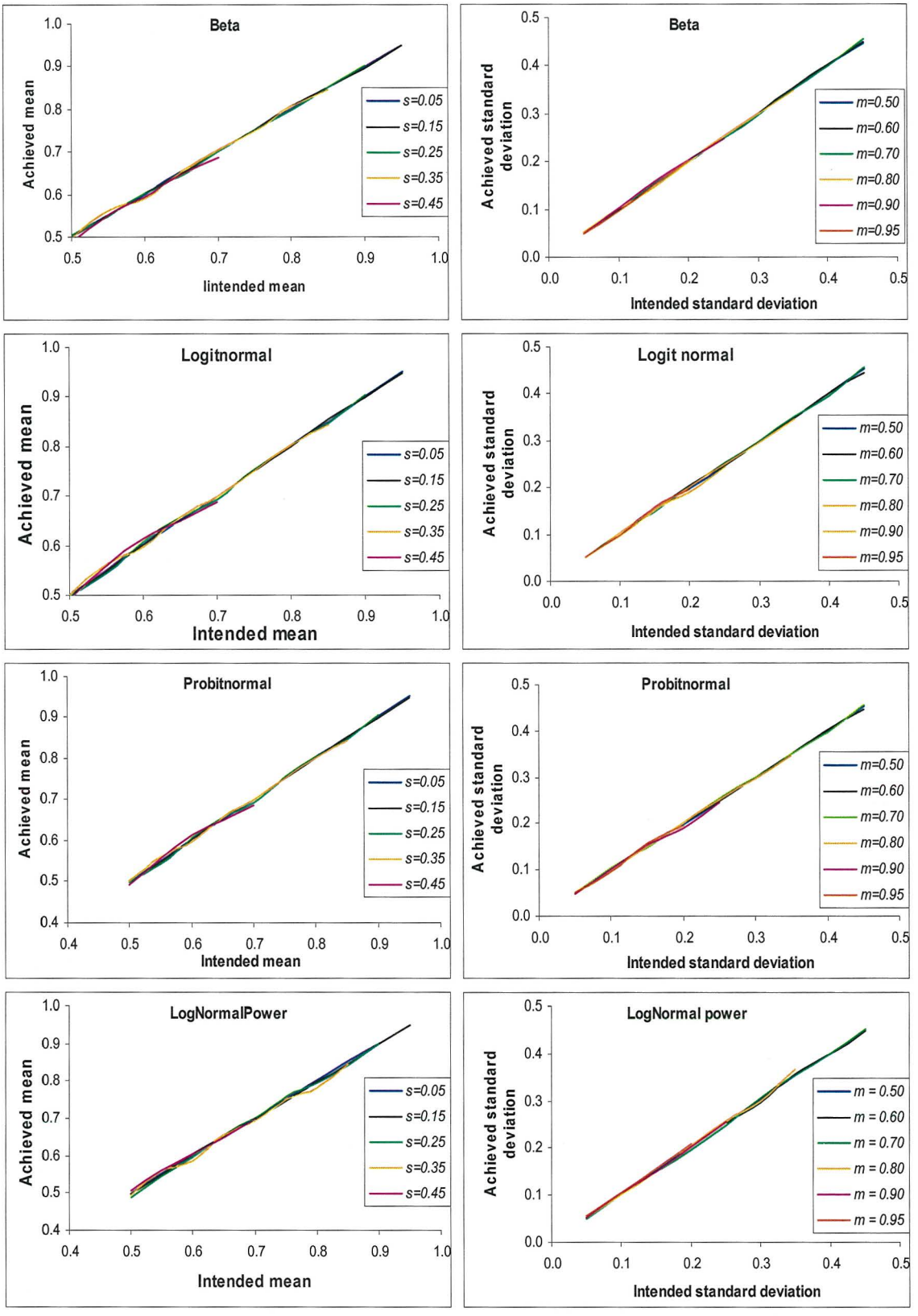

Figure 4.2: Comparison of mean and variability of intended vital rates with those achieved from various distributions (when parameters were obtained analytically for beta distribution and numerically for other distributions).

4.3.6 Suitability of the Delta method approximation in specifying parameters

The commonest way of avoiding difficulties in numerical integration and the most likely approach in practice, is the use of the delta method approximation whenever it simplifies the calculation. Parameters approximated in this way do not represent the intended distribution exactly. For example, as Figure 4.3 indicates, probability density functions (of distributions) obtained using the delta method and numerical integration are different. Therefore it is worthwhile to see to what extent the generated vital rates are affected by this bias, or whether this bias is small enough that we do not need to use numerical integration methods.

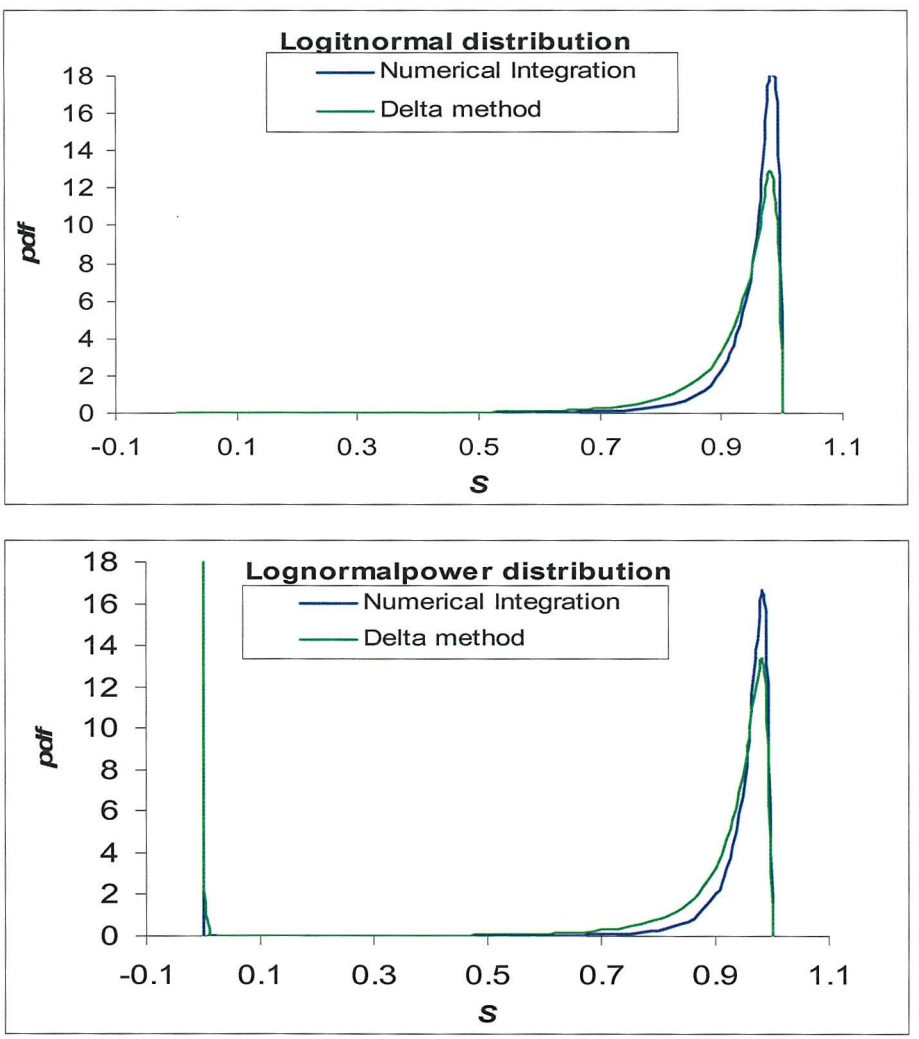

Figure 4.3: Shapes of distributions when estimated using delta method and numerical integration. The intended mean is 0.95 and the intended standard deviation is 0.05.

To investigate this, I repeated the specification of parameters and generation of vital rates (as in section 4.3.5) for the logitnormal and lognormalpower distributions using the delta method. Figure 4.4 compares the mean and standard deviation of the generated vital rates with those intended. It is clear from Figures 4.2 and 4.4 that the delta method approximation can be 'good', if at all, only when the mean is away from boundaries and the standard deviation is small (i.e.; only when *s* is small compared to the maximum possible standard deviation for that mean). For this reason, I did not use the delta method approximation for the remainder of this thesis.

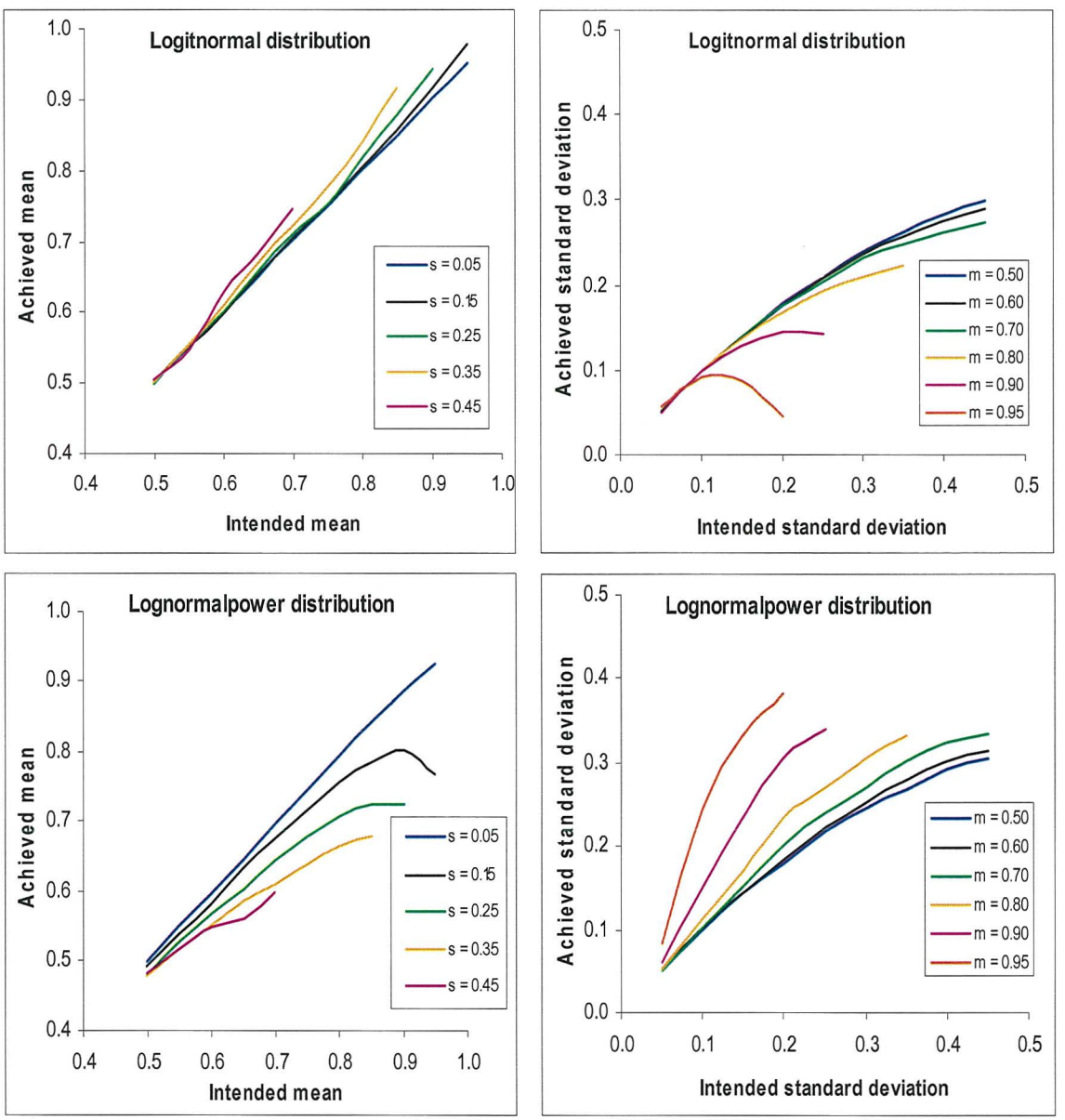

Figure 4.4: Reliability of the delta method approximation for specifying parameters in terms of mean and variability.

4.3.7 Shape of the distribution

It was observed in section 4.2 that each distribution can take a wide variety of shapes to represent various combinations of mean and standard deviation. This flexibility in shape is important as it reflects the ability of the distribution to change its shape to suit different forms of stochastic variations. Here the 'flexibility in shape' does not simply refer to the possibility of having various shapes. In fact the ability to have a shape that is not suitable for stochastic variation is an undesirable property. Therefore it is necessary to see whether the shapes of these distributions are suitable to represent the intended environmental variability. It may be possible that for some distributions the required combination of mean and standard deviation can exist in the given range (equation 4.1), but that the variability represented by the shape of the distribution is not the one intended. As an example, if a vital rate remains close to a certain value for most years with stochastic variation around that value, then a unimodal shape may be desirable in representing it. However, if it remains very close to one of the boundaries for most years with occasional deviations, then desirable shape may be J shaped. If the variability is so large that the vital rate fluctuates between its boundaries then a U shape can represents it better. If there are good and bad years then a multimodal shape may be appropriate to represent that variability. Although it may be possible to select several distributions to represent the required combination of mean and standard deviation, the shape of those distributions could be different from the one intended. Therefore, it is important to consider the suitability of the shape of the distribution to represent the stochastic variability. In order to investigate the possibility of having undesirable shapes I produced a series of probability density curves for all four distributions for different combinations of m and s (Figure 4.5). Here m and s combinations were selected to represent a wide range of possibilities that are common in marine mammal modelling. Observations on the shapes of these distributions are summarised in Table 4.2.

Although all four distributions take somewhat similar shapes at smaller levels of s (with minor differences), lognormalpower distribution and probitnormal distribution tend to have a peak near zero at larger levels of s (Figure 4.5). Of these the peak of probitnormal distribution is smaller and could sometimes be unnoticed as Todd and Ng (2001) report that their numerical investigations resulted in probitnormal

distributions that are very close to beta distribution (beta distribution does not have such a peak). The peak in the lognormalpower distribution is very prominent, and becomes more obvious when s is moderate or large. It is questionable whether such a shape is suitable in representing stochastic variation.

As seen from Figure 4.5, all four distributions can take a wide variety of shapes. However, of all the distributions, beta is the only distribution where the shape can be determined analytically for any given combination of m and s (see Appendix 5 for derivations). Figure 4.6, which is reproduced from appendix 5, summarises the combinations of m and s that correspond to various shapes. The figure shows five regions- two of them correspond to J shaped distributions, and one each corresponds to unimodal and U shaped distributions. The region labelled as undefined indicates those combinations that cannot exist in the given range. All theoretically possible combinations of m and s (as defined by equation 4.1) can be represented by a beta distribution with a specific shape.

Unlike the beta distribution, the shape of any other distribution that corresponds to a given combination of m and s cannot be determined until their parameter values are obtained numerically. Therefore the beta is the only distribution that allows checking m and s combinations against intended shapes. The shape of the beta distribution changes gradually from unimodal to J shape and to U shape with increasing s (given the mean remains unchanged). Therefore, the beta distribution offers more flexibility in having different shapes to suit differently shaped temporal variability.

Table 4.2: Summary of shapes of distributions

Distribution	Shape	Comments
Beta	1. Wide range of shapes possible including uniform, unimodal, symmetric, positive or negative skew, J shaped or U shaped. 2. Shape can be determined by *m* and *s* analytically (see appendix 5).	1. Do not have shapes that are unsuitable in representing environmental stochasticity. 2. The shape can be determined in advance (i.e., it can be determined in advance whether intended variability is actually represented or not).
Logitnormal	1. Wide range of shapes possible including unimodal, symmetric, Positive or negative skew, J shaped or U shaped, but not Uniform. 2. Shape cannot be determined from *m* and *s* analytically.	1. Shape is similar to beta distribution for almost all combinations of mean and s. 2. Not known which shape is used (i.e., shape cannot be determined in advance). However, it does not have shapes that are unsuitable in representing environmental variability.
Probitnormal	1. Shape can be unimodal, symmetric, positive or negative skew, J shaped, or U shaped. 2. Small peak near zero when *s* is large. 3. Shape cannot be determined from *m* and s.	1. Shape is similar to beta distribution in most combinations of *m* and *s*. 2. Not known which shape is used (i.e., shape cannot be determined in advance). However, it does not have shapes that are unsuitable in representing environmental variability.
Lognormalpower	1. Unimodal when *s* is small. 2. Peak near zero when *s* is moderate or large. This peak becomes narrower and closer to zero at larger *s*, so shape appears similar to U shape. 3. Shape cannot be determined from *m* and *s* analytically.	1. Shape is similar to beta distribution only when *s* is very small. 2. Peak near zero makes the shape undesirable to represent environmental variability. 3. Not known which shape is used (i.e., shape cannot be determined in advance). It can even take undesirable shapes).

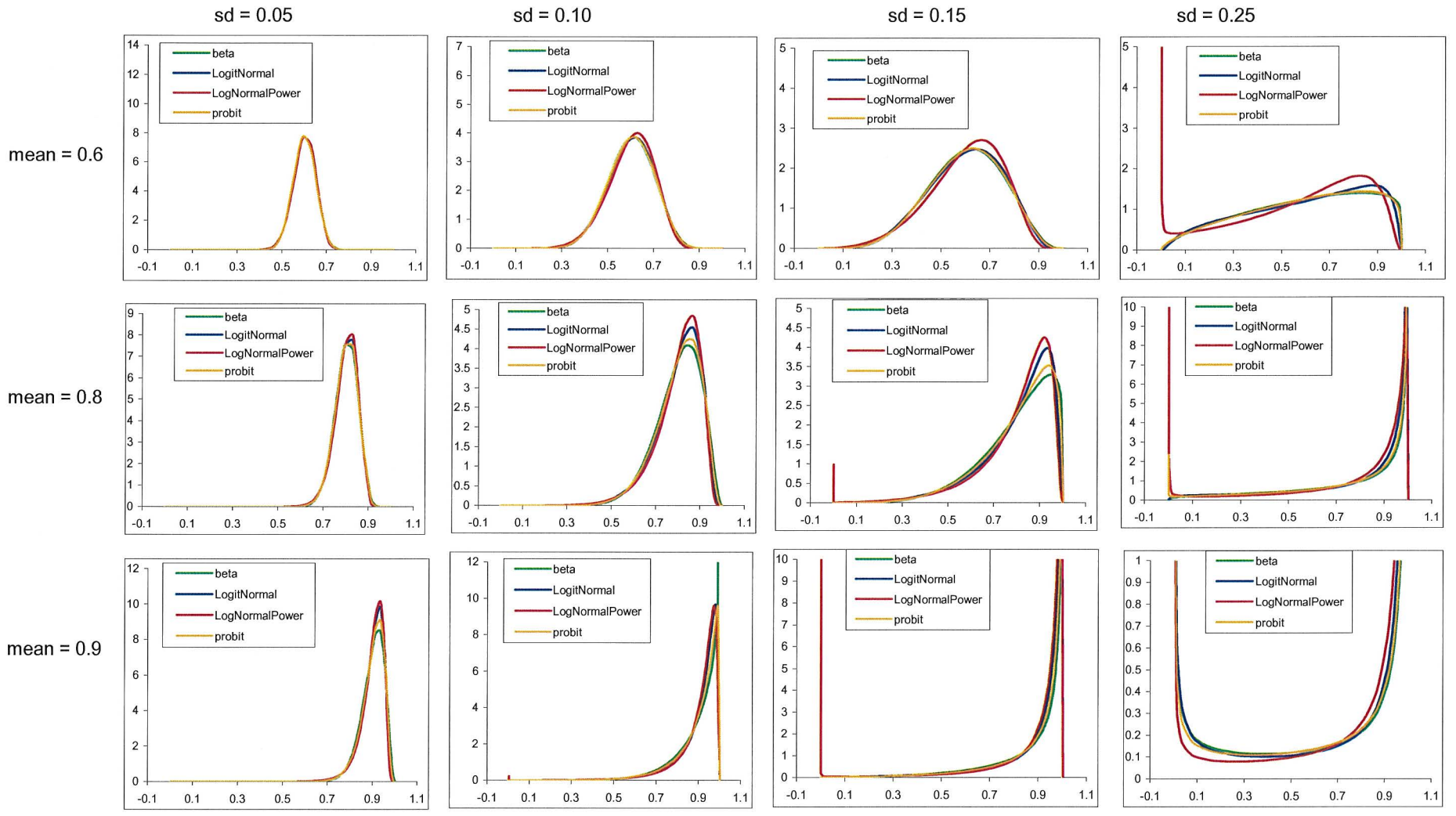

Figure 4.5: Shapes of distributions with different means (m) and standard deviations (s). Shapes under all four distributions are unimodal and similar when s is smaller. They generally become J shaped and finally U shaped when s is increasing. The lognormalpower distribution has a different shape with a peak near zero when s is moderate or large.

Table 4.3: Summary of characteristics of statistical distributions that are relevant to representing environmental variation

Distribution	Shape	Range	Parameter specification form mean and standard deviation	vital rate generation	Possibility of using any sensible combination of *m* and *s*	Ability to represent required variability exactly
Beta	Flexible & can be checked against intended.	Flexible	Exact and analytic.	Possible	Possible	Possible
Logitnormal	Flexible, cannot be checked against intended.	Flexible	Numerical & approximate.	Possible	Difficult to use when *s* is close to its possible maximum.	not exact
Lognormalpower	Dominant peak at zero, cannot be checked against intended.	Flexible	Numerical & approximate.	Possible	Difficult to use when *s* is close to its possible maximum.	not exact
Probit	Flexible, cannot be checked against intended.	Flexible	Numerical & approximate.	Possible	Difficult to use when *s* is close to its possible maximum.	not exact

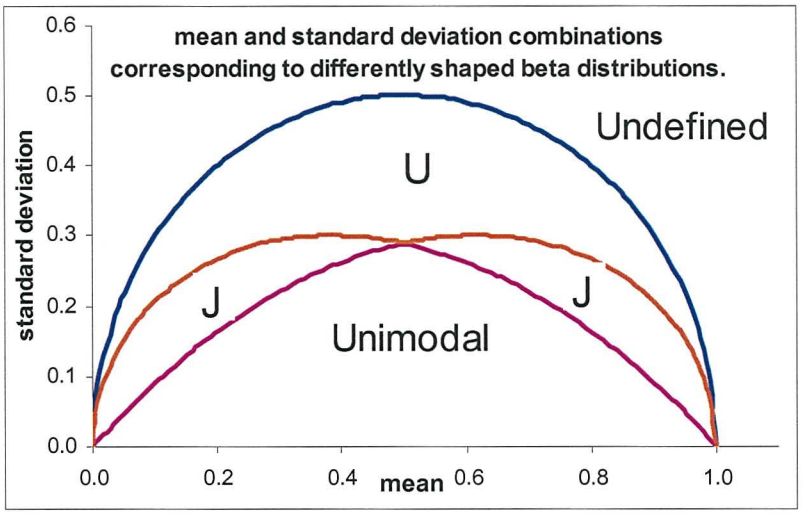

Figure 4.6: Relationship between mean and standard deviation for differently shaped standard beta distributions. The beta distribution is not defined for any combination of m and s that falls outside the half-circle area. The four sub areas in the half-circle area show the combinations of m and s correspond to unimodal, J shaped and U shaped beta distributions as marked, with J shape having two distinct areas. Any combination of m and s defined by equation 4.1 can be represented by one of the shapes.

4.4 Summary

In this chapter I first discussed various approaches in representing environmental stochasticity in population models. I then focused on the approach where environmental stochasticity is represented in population models through demographic rates that vary independently and identically as random variates of statistical distributions. I discussed the strengths and weaknesses of presently used statistical distributions as well as some prospective distributions. I compared them on the basis of properties that are indicative of their ability to represent the desired form of environmental stochasticity in a population model. These properties are summarised in Table 4.3. For several reasons the beta distribution appeared to be better than the other distributions examined. In next chapter I will see how the differences between these distributions translate to population predictions. I delayed the discussion on the results of this chapter until the end of next chapter because it is easier to discuss them both together.

5 Effect of Stochastic Distribution on Population Predictions

5.1 Introduction

In the previous chapter we discussed the characteristics of some commonly used statistical distributions, and some other candidate ones, in relation to their ability to represent environmental stochasticity in population models. We observed that some distributions do not represent the natural variation in exactly the way intended. Therefore it is worthwhile to see to what extent population predictions are sensitive to these shortcomings. It is natural to raise the question of whether the outcome of a population dynamics model will be significantly affected by these dissimilarities, or whether the effect is so insignificant that these differences have no practical importance and any of those distributions can be used in a population model irrespective of the above differences[1] In the present chapter I try to answer this question by using these distributions in the same population model to simulate population outcome.

In assessing the effect of the distribution used to represent the environmental stochasticity on the population outcome one can use either a simulation based approach or an analytic approach such as calculation of sensitivities (Caswell 2001). The simulation based approaches are straightforward, because we can directly see the effect of a distribution. Sensitivity analysis based on eigen value based analyses do not directly show the effect of distribution, but instead shows the effect on the stochastic growth rate caused by shifting the mean vital rate. If there is a bias in representing vital rates by using a certain distribution (as we saw for some

[1] Similar questions have been examined with regard to the demographic stochasticity. It has been observed that population outcomes can be very sensitive to the distribution used e.g., between normal and gamma distributions (Caswell 2001,p492), and between normal and lognormal distributions (Nakaoka 1997).

distributions) then in that case sensitivity indirectly measures the effect of using that distribution[2]. Further, analytical solutions have not been developed for most population performance indicators of interest in conservation biology. For example, even though the sensitivity of stochastic growth rate to changes in vital rates can be calculated analytically (Caswell 2005), no such methods are available for indicators such as probability of extinction, time to extinction, or time to recover. In this chapter I therefore use a simulation based approach.

5.2 Population dynamics model and the Monte Carlo simulation

For the simulation I use the basic population dynamics model introduced in chapter 2 for NZSL population, with the exception of ignoring density dependence and bycatch. However these simulations were not intended to investigate the fate of the NZSL population, but were rather designed to compare possible consequences of using different stochastic distributions. The population model and the corresponding dynamics can be summarised by Figure 5.1 and Equation 5.1 respectively. This model was selected to be simple enough to interpret results easily but general enough to contain the features common to most populations of large mammals.

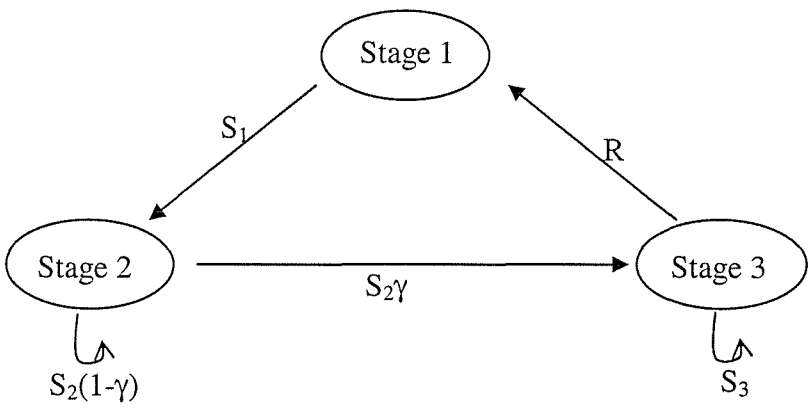

Figure 5.1: Life cycle diagram for the stage-structured population model used in the case study. S_1 to S_3 are stage-specific survival rates, R is the reproductive rate, γ is the proportion of individuals that move from Stage2 to Stage3 at the end of the year

[2] Here the 'sensitivity' is the sensitivity of the growth rate to changes in vital rates. The word 'change' is used to denote shifts in mean value of the vital rate as a consequence of using a particular distribution, not to denote the stochastic variation. In sections 4.3.6 we observed that it is easier to use some distributions together with the delta method approximation. This produces a slight bias in the specified parameters of those distributions. Therefore a sensitivity approach can also be used to assess the acceptability of such approximations.

$$\begin{bmatrix} 0 & 0 & S_3R \\ S_1 & S_2(1-\gamma) & 0 \\ 0 & S_2\gamma & S_3 \end{bmatrix} \begin{bmatrix} N_{1,t} \\ N_{2,t} \\ N_{3,t} \end{bmatrix} = \begin{bmatrix} N_{1,t+1} \\ N_{2,t+1} \\ N_{3,t+1} \end{bmatrix} \qquad \textbf{(5.1)}$$

In this equation $N_{i,t}$ is the population size of stage i at time t, and parameter γ is the transition rate from stage2 to stage3. Under the assumption of within-stage stable age structure, this parameter was estimated using equation 5.2 (see section 2.5 for the notation and explanation).

$$\gamma = \frac{1}{\bar{t}} \exp\left\{ -\ln\left(\lambda / s_2 \right) \left(\frac{\bar{t}}{2} - \frac{V(t)}{2\bar{t}} \right) \right\} \qquad \textbf{(5.2)}$$

All elements in the population projection matrix can be calculated once γ is estimated. I estimated γ assuming stationary population (i.e., $\lambda = 1$) as done by Crouse et al.(1987). This assumption is considered appropriate in this context as the intention of this exercise is not to determine the fate of the population but rather to investigate the effect of different stochastic distributions on the outcome of the population.

In this exercise I simulated environmental stochasticity in the adult survival parameter S_3 with each of four distributions, using one distribution at a time. In the interest of interpretational simplicity, stochasticity in any other vital rate was not simulated. Although this makes the model unrealistic, it does serve the purpose of the investigation. A range of values was used for all other parameters of the model in order to cover a wide range of possibilities. At each time step of the population projection, I replaced the value of the adult survival with a random value drawn from the chosen distribution. In order to ensure all distributions reflect the same level of stochasticity, parameters for each distribution were first specified in terms of the mean and the variance of the intended stochastic variance. This ensures that the mean of the distribution is same as the deterministic value of the adult survival, and the variance reflects the required level of stochasticity. Once a random value is drawn from the distribution, it is used as the survival rate for all adults in that year. A series of standard deviations were used to cover a wide range of stochasticity levels in adult survival. This simulation implies that the same level of stochasticity is used with each distribution, making results from different distributions comparable.

It is necessary to compare the effect of the above four distributions while covering a wide range of values for other vital rates. For that reason, one has to start with different levels of stage-specific survival rates and different degrees of variability, and use combinations of them in each of the distributions. This could end up with an unmanageable number of combinations, justifying the selection of a manageable, but still representative subset of combinations. For this reason I introduce a new parameter (C) and used that parameter to vary all stage-specific survival rates in the manner as described below.

In order to cover a range of stage specific survival rates one can vary all of them by adding a common constant (positive or negative) as $S_i^* = S_i + C$ for i = 1, 2, 3, where S_i and S_i^* are the survival rate in stage i before and after adding C, and the constant C determines the direction and magnitude of change in survival rates from S_i. This allows the changing of the mean survival rates of all stages by varying a single quantity C. However, this can sometimes shift S_i^* beyond the acceptable range of zero-to-one. To avoid this situation I multiplied C by $S_i(1-S_i)$, which is the product of two distances from lower bound of zero to S_i and upper bound of one to S_i. The resultant equation becomes

$$S_i^* = S_i + S_i(1-S_i)C \quad , \qquad -1 < C < +1. \qquad \textbf{(5.3)}$$

For the same values of C, this equation allows a larger shift when S_i is closer to the middle of its range than when it is closer to a boundary, and therefore avoids moving S_i^* out of the acceptable range. For a given S_i value the corresponding S_i^* values will be proportional to C (Figure 5.2). This does not allow the flexibility of moving survival rate of one stage upwards while for another stage downwards. However, in the interest of simplicity, I choose not to have that flexibility, even though this is a likely situation for some populations under certain circumstances.

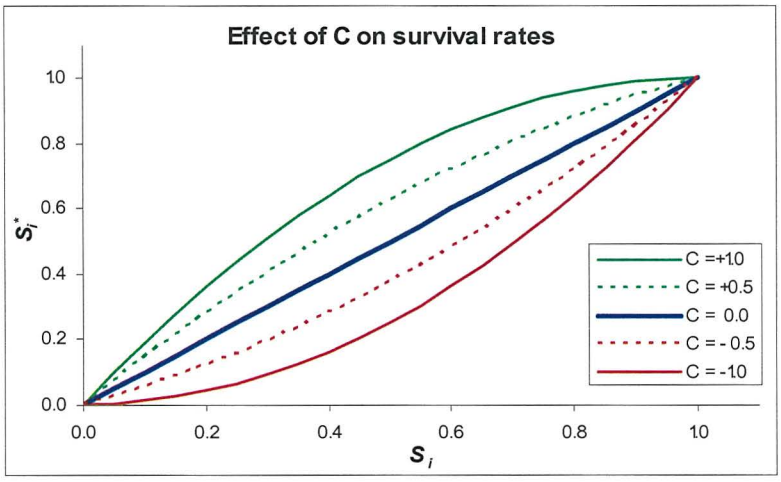

Figure 5.2: This figure shows the way that input survival rate in stage i (S_i) is transformed by C to effective survival rates (S_i^*).

Since C is an indicator of how big the effective survival rate of each stage (S_i^*) is compared to their input values (S_i), we can change the single parameter C to change S_i^* of all stages, instead of changing S_i of each stage individually. Therefore, combinations of different values of C and standard deviation (s) can be used to compare the effect of different distributions under a variety of benign and challenging 'population statuses' or 'survival statuses'. For example, three levels of survival rates (say high, medium, and low) can be used in the population model by simply choosing three different levels of C without changing any of the S_i inputs; a larger C corresponds to larger survival rates in all stages.

The dynamics of the population described in this population dynamics model was programmed using Matlab® software. Input values used for the survival rate were S_i = 0.80, 0.85, and 0.90 respectively for three stages. I included three levels of C (i.e., C = - 0.5, 0, and + 0.5). The corresponding stage-specific survival rates effective in the model as produced by equation 5.3 are given in Table 5.1. These values are roughly similar to the survival rates of the NZSL population.

A wide range of standard deviations (i.e., $0 \leq s \leq 0.2$) were used in the simulation. The upper limit of 0.2 was selected because standard deviation of 0.2 can be considered large enough when representing variation in survival rate due to environmental changes.

Table 5.1: Stage specific mean survival rates effective in the simulation (S_i^*) at three levels of C .

	S_1 = 0.80	S_2 = 0.85	S_3 = 0.90
C = - 0.5	0.720	0.786	0.855
C = 0.0	0.800	0.850	0.900
C = + 0.5	0.880	0.914	0.945

The initial year stage-specific population distribution was based on the stable stage distribution with the chosen input values for the model parameters, scaled by the number of stage1 animals in the initial year[3]. A wide range of levels were used for the input parameters (R from 0.3 to 0.45, $\bar{t}$ from 1 to 3, and V(t) from 1 to 2). Although the choice of these values is arbitrary, it is assumed to be close to the values for large mammal populations, particularly to the NZSL population. Under each combination of C and s, and under each distribution, the population was projected for a period of about five generations (i.e., 75 years). The population growth rate and the probability of quasiextinction were then estimated. For this purpose, I defined quasiextinction as the total population (excluding newborns) falling below a certain level, at least once over the projection period. Use of 'at least once' instead of 'at the end of the simulation period' is arbitrary, but it may be more conservative and sensible for situations where the population level falling below a certain level is critical. It is equally possible to use other definitions for quasiextinction. The use of 'total population size' in assessing quasiextinction risk is also arbitrary. Therefore I re-estimated the quasiextinction risk under two new definitions (i.e., reproduction falling below a certain level at least once over the projection period, and adult population falling below a certain level over the projection period). Since risks estimated under all three definitions were very similar I do not present results obtained for these two new definitions. For each combination of mean and standard deviation under each distribution, I also calculated the elasticity of stochastic growth rate to the changes in adult survival rate using the method of Caswell (2005).

[3] The number of stage1 animals in the initial year was chosen to be 1400 which is close to the present female pup production of the New Zealand Sea Lion population. Although the initial population size has an effect on the predicted fate of a population, it is unlikely to affect the differences in fates predicted by various distributions, and hence is unlikely to influence the comparison of distributions.

5.3 Effect of distribution on stochastic growth rate

As expected, the stochastic population growth rate[4] increases when the mean survival rate (as measured by C) increases. There is no difference between predictions from different distributions at small levels of variability. Predictions appear to differ at higher variability (Figure 5.3A). These differences do not depend on the level of the mean survival rate. The only exception observed is the lognormalpower distribution that predicts a larger growth rate than predicted by other distributions when mean and standard deviation are large. As seen in Figure 5.3B, the growth rate becomes smaller when stochasticity intensifies. This observation is consistent with the Tuljapurkar (1997) formula for stochastic growth rate, and with most previous results. All four distributions predict similar results, with the exception of the lognormalpower distribution which shows different behaviour when mean and standard deviation are large.

5.4 Effect of distribution on extinction risk

The probabilities of quasiextinction under different distributions (on the basis of the total population size) are compared in Figure 5.4 for different levels of C and standard deviations. As shown there, the population does not become extinct under any distribution when C is large (i.e., mean survival rate is large) and standard deviation is small. Extinction risk curves appear to be similar under all distributions for most combinations of mean and standard deviations, except when the mean and standard deviation are both large. In particular, the lognormalpower distribution, the distribution that is under consideration by the management to use with NZSL population, predicts a lower risk of extinction in these combinations.

[4] Here the population growth rate is the rate at which the size of the total population (excluding new born animals) changes from one time step to the next. In addition to this, I investigated how the adult growth rate and reproductive growth rate (i.e., rates at which adult population level and reproduction changes with time) respond to the changes in C and *s*. Since the observed patterns were not different from the ones above they are not shown here.

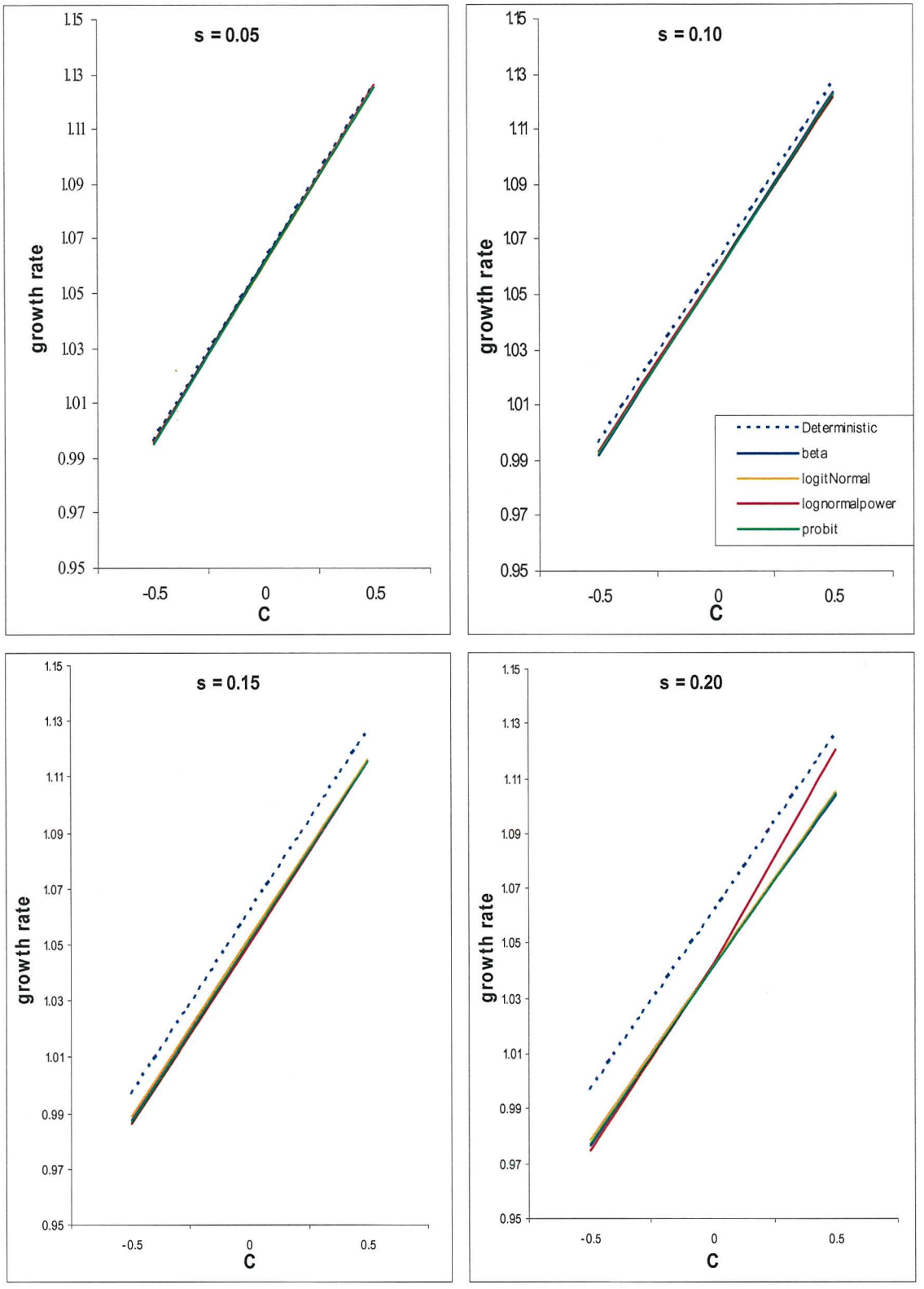

Figure 5.3A: Stochastic population growth rates predicted by different distributions for different combinations of mean and standard deviation. This shows the effect of the mean of the stochastic distribution at different levels of stochasticity. C is a measure of the mean adult survival rate, and *s* is the standard deviation of stochastic variation around the mean.

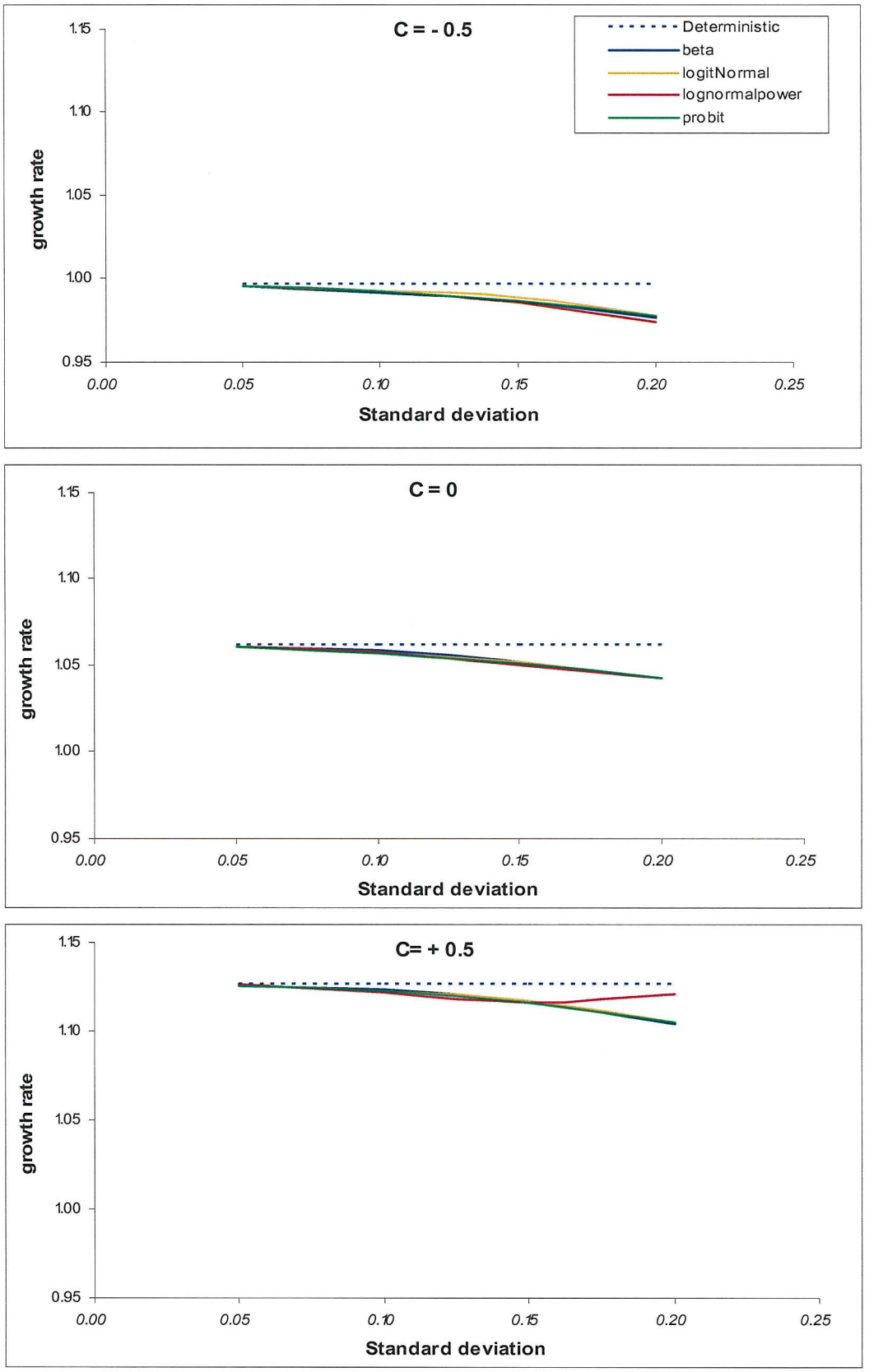

Figure 5.3B: Stochastic population growth rates predicted by different distributions at different combinations of mean and standard deviations. This shows the effect of level of stochasticity at different levels of mean. C is a measure of mean adult survival rate, and *s* is the standard deviation of the stochastic variation around the mean.

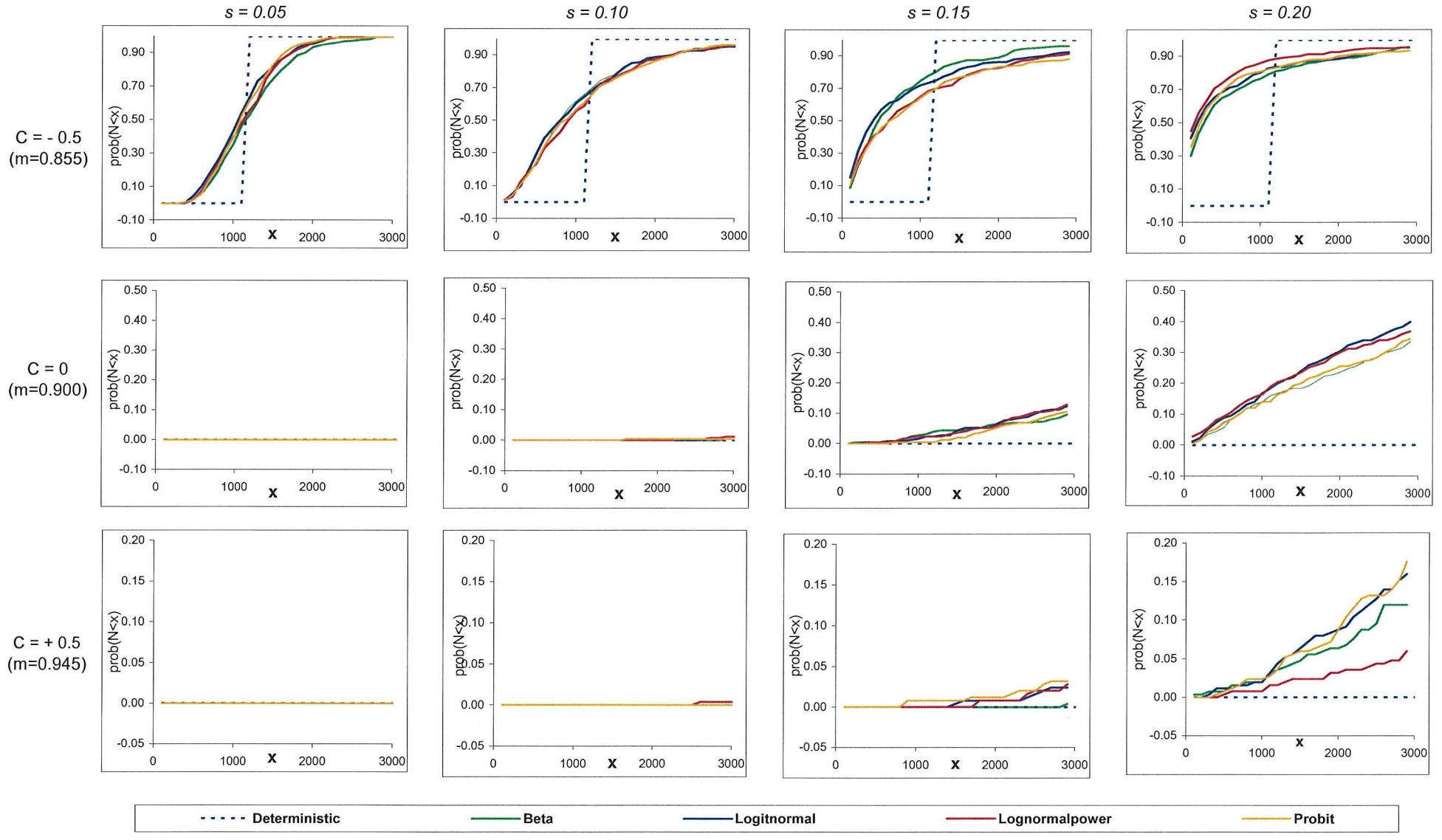

Figure 5.4: Comparison of probabilities of quasiextinction under different distributions for different levels of C and *s*. (C is an index for the mean *m*, and *s* is the standard deviation). Each panel compares the probability of total population falling below a given level (x) for a specific combination of C and *s*. Note different scales used at each level of C. At lower levels of *s* the extinction curves are almost indistinguishable between distributions. Lognormalpower distribution predicts a lower risk when C and *s* are large (see section 5.2 for the level of possible simulation error).

5.5 Effect of distribution on the elasticity of stochastic growth rate

Elasticity measures the responsiveness of growth rate to changes in the (mean level of the) vital rate, on a proportional scale. It does not measure the effect of the distribution on the fate of the population predicted by the model directly. However here I use the elasticity as an indirect measure of that effect. If a certain distribution causes a bias in representing intended variability then the effect of that bias on the population growth rate (as measured by elasticity) can be seen as the effect of the distribution.

Such a bias may be caused by more than one reason. First, in this case study we used comparable levels of mean-variance combinations with all distributions. However, we had to use numerical procedures to obtain parameter values for distributions (except for the beta distribution). In chapter 4 we observed that the simulated survival rate may not have exactly the required mean under some distributions. This could be due to inaccurate parameter values resulting from numerical procedures and/or due to limitations in the random number generators. Second, a likely simpler alternative to numerical procedures is the delta method approximation that also leads to bias in simulated variability (section 4.3.6). These biases possibly yield different results in population outcome which can be treated as indirect measures of the effect of distribution.

If the effect of this bias on the population outcome is not great then we may not have to worry too much about the bias of not achieving the required mean. This effect can best be viewed as the elasticity of stochastic growth rate to changes in the (mean adult) survival rate (i.e., the percentage bias in the estimated stochastic growth rate caused by 1% bias in mean of adult survival rate). Intuitively, this bias in growth rate should depend on the level of survival (i.e., on C), and on the level of stochastic variation (i.e., on s). I estimated this elasticity for different combinations of C and s for each distributions using the method of Caswell (2005). As seen from Figure 5.5, elasticity is dependent on the distribution used to represent the variation. This is

consistent with Caswell (2005, eq 15) because the year-to-year growth rate, which depends on the chosen distribution, is a component of this formula[5].

The elasticity ranges from about 0.69 to 0.76 (averaging at about 0.73), meaning a 1% bias in adult survival rate translates to about a 0.73% bias in stochastic growth rate (Figure 5.5). This is a large bias if compared with the range of possible growth rates for marine mammals, for example, Breen (2003) predicted 3% maximum possible growth rate for the NZSL. This high elasticity is consistent with the results of Lebreton and Clobert (1993) for long-lived species. The higher the C the lower the elasticity at a given level of stochasticity (at a given level of *s*), implying that a change in mean survival has less effect when the mean survival is large. Elasticity is found to be larger when the standard deviation is larger. This is not surprising because a stronger variability has a larger effect on growth rate when the mean change is the same. Elasticity does not depend on the distribution when the level of stochasticity is small. It differs between distributions at large levels of standard deviations, with the lognormalpower distribution having the largest elasticity. Therefore unbiased representation of survival rate is more important for this distribution, and so approximation procedures such as the delta method are unsuitable, particularly for this distribution.

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[5] It is common to use analytical formulas in assessing sensitivity of deterministic growth rate to changes of vital rates (Caswell 2001), but not in assessing the sensitivity of stochastic growth rate, because the theory was developed only recently. According to Caswell (2005), the elasticity of stochastic growth rate λ_s to a vital rate θ is given by

$$\frac{\partial \log \lambda_s}{\partial \log \theta} = \lim_{T \to \infty} \frac{1}{T} \sum_{t=0}^{T-1} \frac{\theta_t V_{t+1}' \frac{\partial A_t}{\partial \theta} w_t}{R_t V_{t+1}' w_{t+1}}$$

, where T is the number of time steps in the projection period, A_t is the population projection matrix at time t, θ_t is the value of vital rate θ at time step t, W_t and V_t are the right and left eigenvectors of A_t (or equivalently, stable age distribution and reproductive value corresponds to matrix A_t), V_{t+1}' is the transpose of V_{t+1}, and R_t is the growth rate from time step t to step (t+1).

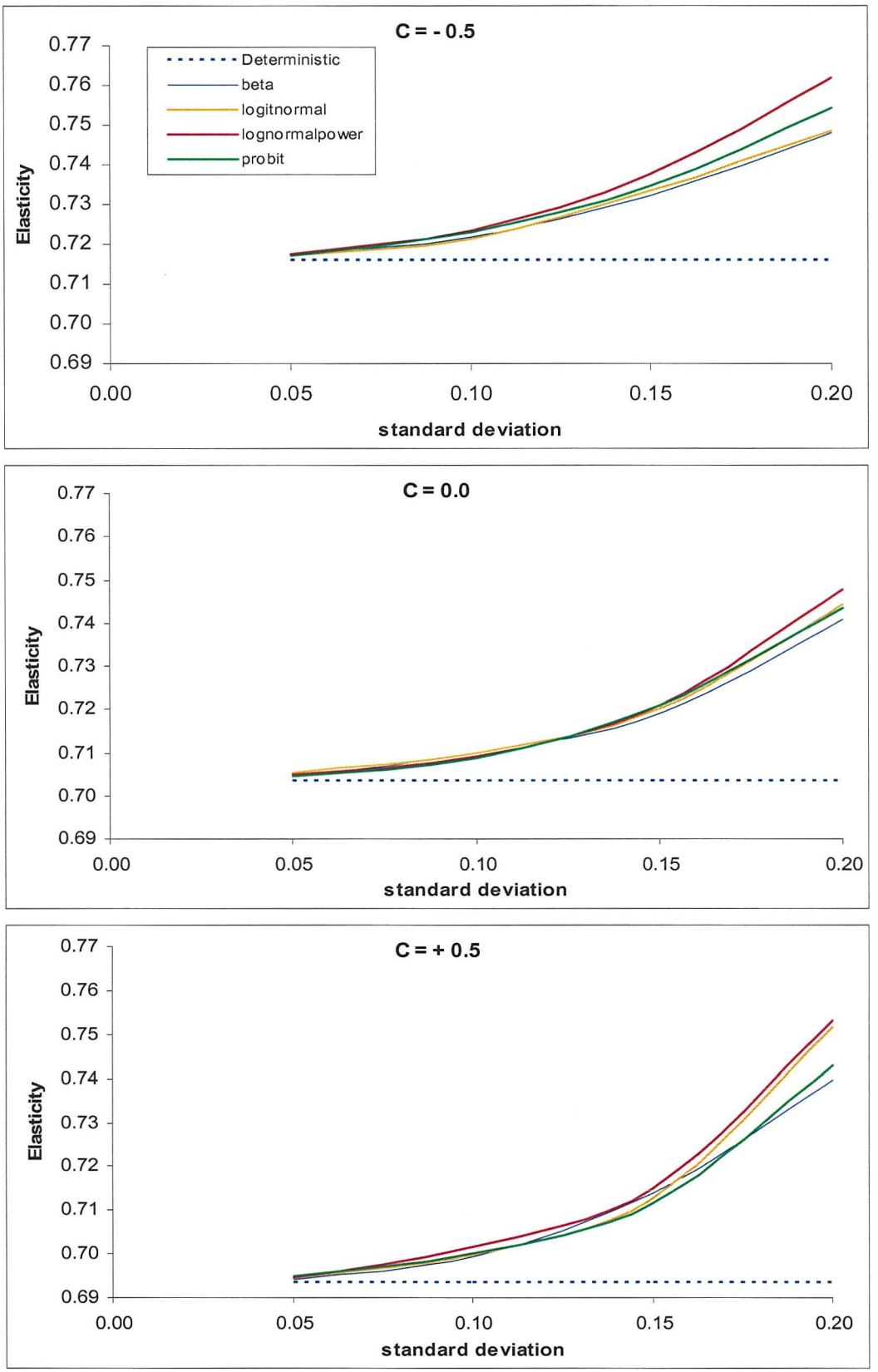

Figure 5.5: Comparison of elasticity of stochastic growth rate with respect to changes in mean adult survival rate when the stochasticity in adult survival rate is represented by different distributions. C is a measure of the mean adult survival rate, and standard deviation measures the temporal variability around the mean.

5.6 Discussion

5.6.1 Other possible approaches in modelling stochasticity

The purpose of modelling environmental stochastic variation is to reflect the effect of environmental changes on population predictions, by realistically representing them in the population model. In the approach we discussed earlier in this chapter, variation of the vital rate is assumed to follow a specific statistical distribution, parameters for that distribution are specified, and a value of the vital rate is sampled from that distribution at each time step of the population projection. This is analogous to the parametric bootstrap procedure where data values are resampled from a distribution that is estimated from observed data.

A possible alternative approach can be suggested that is analogous to the nonparametric bootstrap procedure, where a value of the vital rate for each year is randomly selected from a list of observations. This method seems justifiable since the natural variation does not need to follow a specific distribution as assumed in our approach. In addition, this approach will guarantee that no improbable values are used, and more commonly observed values are more frequently used in the projection. Another advantage is that any cross-correlation observed in data is automatically included in the analysis when representing stochasticity in more than one vital rate at once. This procedure avoids the difficulty of selecting a suitable statistical distribution and specifying its parameters. However, there are several drawbacks in this approach.

- It is highly unlikely that a long list of observations is available for the vital rate. Even if such a list exists it is quite possible that the entire range of possible values is not covered by the observation list. It becomes restrictive because this method does not allow for the selection of any unobserved values even if those values are highly probable.
- The variability represented by a list of observations consists of both process error and observation error. This approach automatically includes both these forms of variations in the projection. When using the approach I described earlier, it is possible to remove the observation error from the total variation

contained in a list of observations (e.g., Gould and Nichols 1998), enabling me to use only the process error in the projection as required.

Bayesian methods provide another approach, where although it is necessary to specify the distribution that follows the stochastic variation, the difficulty of explicitly estimating (or specifying) parameters for that distribution does not arise. Although there is controversy about the use of Bayesian approach which I will not go into, it is sometimes argued that Bayesian methods are preferable for modelling in conservation biology over other methods for several reasons (Wade 1999, Wade 2000, Wade 2002).

5.6.2 Choice of distribution

The choice of the distribution to represent the environmental variation seems subjective in most population models. It is often based on the experience with other species, convenience, or previous practice, and it is not justified in the majority of cases. Modelers rarely check whether the variation introduced by the chosen distribution represents the intended variation. For example, it can be seen from the literature that truncated distributions are still used even in situations where the mean is not far from one of the boundaries (i.e., when the truncation is considerably asymmetric, causing the resultant mean to be different from intended). The disadvantages of these truncated distributions were discussed in section 4.2. For example, the reduction in standard deviation due to truncation will translate to an underestimation of the possible variation in population outcomes, leading to an underestimation of extinction risk in population viability analyses. Although adjustments such as truncation are likely to produce spurious results they are rarely documented.

It is not clear whether the inflexibility in the shape of the distributions, and the inability of achieving the required mean and standard deviation, are known or not, because it is difficult to find any discussion on this issue, let alone on how to deal with them. For this reason, it is unclear whether the models that use these distributions are able to predict the fate of the population reasonably. The main focus in this

chapter was to see whether the distributions that are in use could actually represent the stochastic variation as intended.

5.6.3 Parameter estimation and speed of implementing population model

Here the term 'estimation' is used in the sense of obtaining numerical values for parameters using known or specified information (i.e., from mean and standard deviation) about the stochastic variation, rather than estimating parameters from raw data. Out of the four distributions discussed in this chapter the beta distribution is the only distribution where the parameters can be estimated analytically and exactly (appendix 3). Although parameter values can be obtained with 'a specified accuracy', using numerical methods, those values are not 'exact'. In addition, numerical implementations are slow, and often require specialised software or skills. In most situations this difficulty can be overcome by using the delta method approximation. However, this approximation yields biased estimates especially when the standard deviation is close to its possible maximum (see section 4.3.6).

If the parameter values are inaccurate, simulated variation will be different from that which is intended. As a consequence the model can give misleading results without any warning on its reliability. Better numerical procedures are yet to be developed to overcome this difficulty. Even if such problems were avoided with better numerical procedures, most numerical procedures cannot easily be implemented in simple software. There is no such problem with the beta distribution where analytic solutions are available.

Theoretically all four distributions are defined over the entire range of sensible combinations of mean and standard deviation[6]. However, according to my experience, numerical procedures can fail to perform, or can be inaccurate when the standard deviation is very close to its possible maximum for a given mean. This is partially dependent on the software used. It is unlikely that this may pose a concern in practice,

[6] A mean (m) and standard deviation (s) combination is not sensible for the finite interval between a and b if they do not satisfy the requirement of $(b-m)(m-a) \geq s^2$. See section 4.1.6.

because a standard deviation near its maximum may be beyond most practical situations. A more serious problem is the undesirable shapes of distributions even at the moderate levels of standard deviations, as observed for the lognormalpower distribution (see sections 4.3.7 and 5.6.5).

It was observed that the implementation time is heavily dependent on the distribution used. For a comparison of times taken by these distributions, the following list shows the time taken to produce the same number of trajectories by the population model used in the case study[7]. Clearly the distribution used affects the time-efficiency of the projection, with the beta distribution being the most efficient.

Deterministic	98 seconds
Beta distribution	170 seconds
LogitNormal distribution	270 seconds
Probitnormal distribution	964 seconds
LogNormalPower distribution	538 seconds

The density independent model used in this case study only requires the parameter values to be calculated once for each combination of input values, irrespective of the number of trajectories and length of trajectories. In contrast, in density dependent models parameters has to be calculated at each time step. Therefore, in general, the time differences shown above will be larger for density dependent models.

5.6.4 Reliability of beta random number generators

At each time step of the prediction, a random value needs to be sampled from the corresponding distribution. This requires a beta generator for the beta distribution, and a normal generator for other three distributions. Reliable random number generators are readily available for the normal distribution in most standard modelling software. As seen in section 4.3.5, variates generated using beta and normal generators (i.e., variates from beta, logitnormal, probitnormal and lognormalpower distributions) seem close to what were intended for most combinations of mean and standard deviations (Figure 4.2). The exception is when the standard deviation is very close to its possible maximum (Appendices 4B to 4D).

[7] These are the time taken by the Matlab programme used in the case study to produce 250 trajectories of 75 years long, with the identical input values, using a 233mHz Pentium computer.

However, according to my experience, some care is necessary in selecting a beta generator. As example, Figure 5.6 compares properties of intended beta variates to those generated by four beta generators. The four generators are, *Betadat* function (written by the author based on algorithms of Johnk (1964) and Ahrens & Dieter (1974) in Kennedy and Gentle (1980, p216), *Betaval* function given in Morris and Doak (2002, p277), the *csbetarnd* function in the computational statistics toolbox for Matlab® (which can be downloaded free from http://lib.stat.cmu.edu), and *Betarv* function given by Morris and Doak (2002, p80). Clearly the *csbetarnd* generator performs poorly for some combinations of mean and standard deviations, notably when the intended standard deviation is close it its possible maximum. In addition, data generated from *csbetarnd* generator will be significantly different from what was intended if the shape parameters are large (i.e., larger than about 200) even if the intended standard deviation is small compared to its possible maximum (e.g., when $m = 0.5$, $s = 0.05$). For all analyses in this thesis *Betadat* function was used. I have not examined the reliability of other available beta generators such as *betarnd* function in the Matlab statistics toolbox, *dBetaDev* function in PopTools®, and the algorithm given by Fishman (1973, p208). Although there are beta generators in statistical software such as minitab® they are not easily portable into modelling software.

5.6.5 Flexibility and suitability of the shape

As seen from Figure 4.1, all of the distributions can take a wide variety of shapes, signaling the flexibility in their shapes to suit the intended shape. However, when values for the mean (m) and standard deviation (s) are specified, then the shape is determined by them, hence there is no flexibility in choosing all three of them under any distribution. Therefore, if the shape determined by the m and s combination is not the one intended, it implies either that an incompatible mean and standard deviation combination has been used or that the selected distribution is unsuitable. Hence, the ability to compare the shape determined by the chosen combination of m and s against the intended shape is valuable. As detailed in section 4.3.7, the beta distribution is the only distribution (out of the distributions examined) where this comparison can be done analytically and easily.

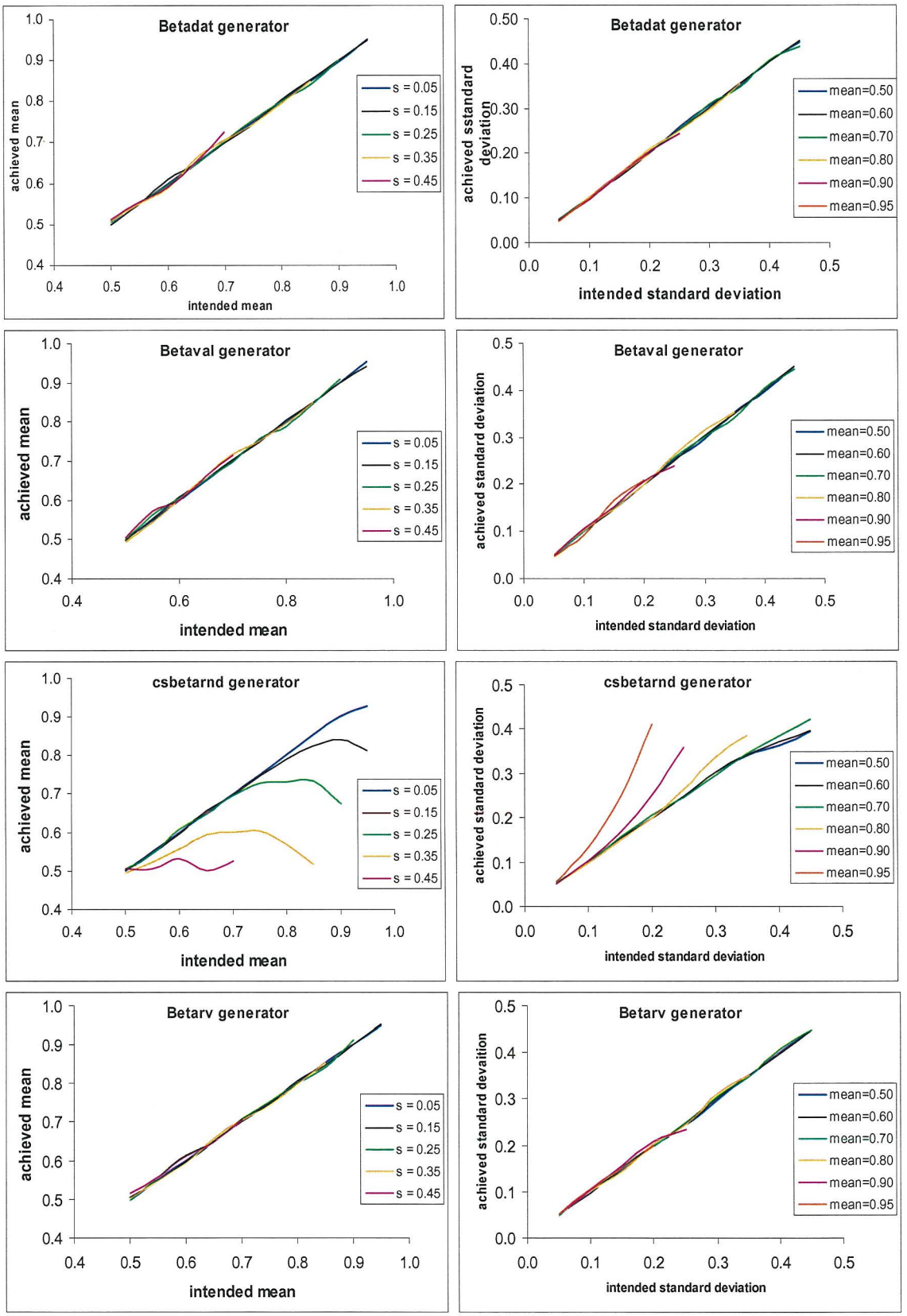

Figure 5.6: Compares the mean and standard deviation of generated beta variates with the intended mean and standard deviation. Four beta generators (*Betadat, Betaval, csbetarnd,* and *Betarv*) were used. The achieved mean and standard deviation are based on 2000 variates.

It is important to note that the shapes observed in the beta, logitnormal and probitnormal distributions may be used to represent some forms of environmental stochasticity adequately (see Figure 4.5). Further, as seen by visual comparisons, they are very similar for most combinations of m and s tried. The probitnormal distribution has a peak near zero when s is 'large', this peak is not as prominent as the one observed in lognormalpower distribution. Although the shape of the lognormalpower distribution is similar to other distributions when s is small, it has a prominent peak near zero at moderate to large s. This is observable for some combinations of m and s even when the m is far from the boundaries. For example, when m = 0.5 and $s = \sqrt{1/12}$ the lognormalpower distribution has a peak near zero while the other three distributions take a shape that is very similar to the uniform distribution. (Note that $s = \sqrt{1/12}$ is the standard deviation of a uniform distribution with a unit range.) Similarly, when m = 0.5 and s = 0.25 other distributions take unimodal shapes, while the lognormalpower distribution is bimodal with one mode near zero (the usual peak) and the other near 0.7. The peak near zero becomes more prominent when s is larger. It is highly unlikely that a distribution of this shape may adequately represent the environmental variability of a vital rate. It may be important to remind readers at this stage that the lognormalpower distribution is the one used in the proposed NZSL management model, and its distinctive shape, which is described here, was one of the reasons that I was motivated to examine the suitability of various distributions for this purpose in this part of the thesis.

5.6.6 Possibility of explaining simulation results from shapes of distributions

One of the results seen in the case study is that the growth rate increases with an increasing mean survival rate under all distributions (Figure 5.3A). This is a result that can be expected intuitively in density independent models. There is no difference in growth rate between distributions at lower levels of stochasticity. When s is large, the growth rate generally decreases under all distributions (Figure 5.3B). This is in agreement with the commonly accepted result (Tuljapurkar 1997) that the growth rate becomes smaller in stochastic environments than in deterministic environments. There

are arguments in favour of and against this standard result (e.g., Higgins et al. 2000, Efford 2001, Higgins et al. 2001). An exception observed is that for higher means and standard deviations, the growth rate predicted by the lognormalpower distribution is larger than what is predicted by other distributions (i.e., very close to what is predicted by the deterministic model). In order to see the possibility of interpreting the results of the case study by the properties of distributions, I produced probability density functions for each distribution for the same mean (measured by C) and standard deviation combinations that were used in the case study (see Figure 5.7). As seen there, for a higher mean and higher standard deviation (e.g., at C = + 0.5 and s = 0.20), all distributions are U shaped, but the left peak of the lognormalpower distribution is much narrower than those of other distributions. Generally, this makes the survival rates generated from this distribution become larger and closer to the upper boundary than those generated from other distributions. This is in agreement with the observation of the larger growth rate at this combination of m and s under the lognormalpower distribution. This distribution is used in the newest model proposed for NZSL population. Since large m is appropriate for the adult NZSL which represent a large proportion of the population, it is likely that this model tends to overestimate the growth rate, and consequently tends to underestimate the extinction risk in highly changing environments.

Another result observed in the case study is that the quasiextinction risk increases with increasing stochasticity, with no difference between the risks predicted by distributions at lower levels of m and s (Figure 5.4). For higher means and higher standard deviations the lognormalpower distribution predicts a lower risk. This result is also in agreement with the different shape of the lognormalpower distribution stated above.

It is observed in the case study that the elasticity of growth rate reduces with the increasing mean survival rate (i.e., with increasing C in Figure 5.5), meaning the (proportional) change in survival rate has a lower (proportional) effect if the survival rate is already higher. Elasticity increases with increasing stochastic variability (i.e., with increasing s) under all distributions, implying that more intense variability has a larger effect on growth rate than slight variability for any given level of mean survival rate. These observations are easy to understand. Elasticity seems larger when the

variability follows the lognormalpower distribution than when it follows any other distribution (including deterministic). This is most marked at lower levels of mean survival rate (i.e., when C = - 0.5). This is in agreement with Figure 5.7 (see top row of panels) as the shape of the lognormalpower distribution is more sensitive to changes in *s*. This is less clear at higher levels of mean survival (see the C = 0 and C = +0.5 panels in Figure 5.5). This result is also in agreement with the shape of the lognormalpower distribution as explained when interpreting growth rate.

One of the distinguishing results that we noticed in the behaviour of the lognormalpower distribution is that it is much closer to the deterministic model than the other distributions in terms of the predicted growth rate and extinction risk when both the mean and standard deviations are large (Figures 5.3A, 5.3B, and 5.4). One might argue that this result does not have much practical value, because a very large standard deviation such as 0.2 is probably unlikely to exist in combination with a large mean (such as 0.945 as used in this demonstration). However, in the pdf of the lognormalpower distribution, we can see the peak near the lower boundary even at a smaller levels of *s* such as 0.1 (see Figure 5.7). Therefore we should be cautious when using a distribution of such a shape to represent environmental variation.

The results of all four distributions are very similar, except that the lognormalpower distribution sometimes has slightly different behaviour. In particular, the lognormalpower distribution has a larger growth rate, lower extinction risk, and larger elasticity at higher mean and higher standard deviation. The interrelationships of these results are compatible, and consistent with the shapes of the corresponding pdf curves.

In this case study I used a range of values for all parameters of the model in order to cover a wide range of possibilities. One might argue that this may include some impossible combinations of parameters, and hence predictions obtained by this simulation may not reflect the true fate of the population. However, this does not invalidate the findings, as the objective of this study was to compare the effect of using different distributions, and not to predict the fate of a given population.

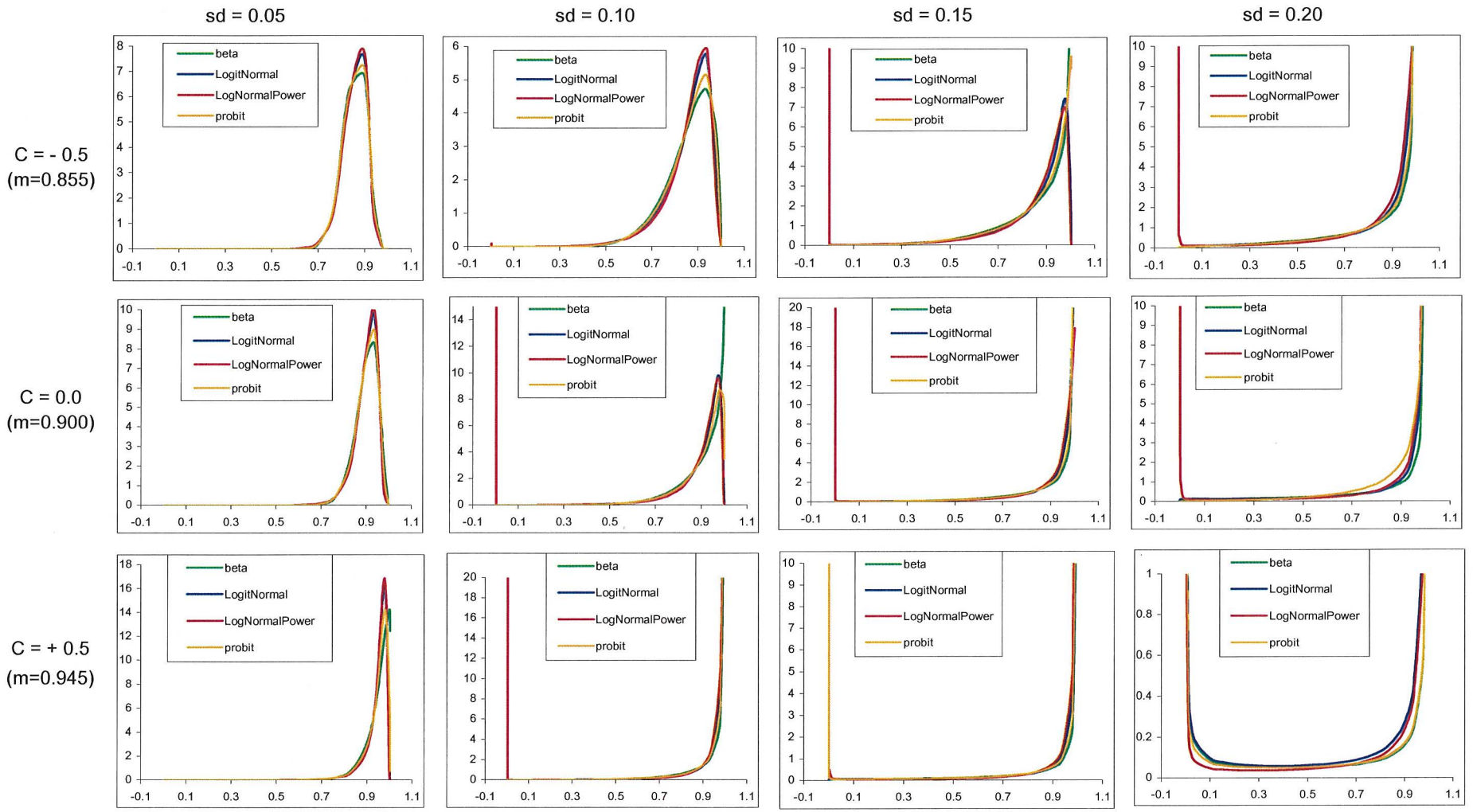

Figure 5.7: Probability density functions of the four distributions for the C and *s* combinations used in the case study. C is a measure of mean. Since the input adult survival rate used in case study was 0.90, C value of -0.5, 0 and +0.5 correspond to means of 0.855, 0.900, and 0.945 respectively. (This figure differs from Figure 4.5 only in the values of *m* and *s* used).

5.6.7 Modelling vital rates over other intervals

In above sections we concentrated on the survival rate, a vital rate on the unit interval. It is relevant to see whether these distributions can be used for other vital rates that are not necessarily on the unit interval. One might suggest that this can be done by defining distributions over a general interval instead of the unit interval. For example, if the logitnormal distribution is defined as

$$\ln\left(\frac{S-L}{U-S}\right) \sim N(\mu_1, \sigma_1^2)$$

then, once parameters μ_1 and σ_1 are specified in terms of mean and standard deviation, random variates can be generated directly over the range L-to-U as

$$S = \frac{L + U e^x}{1 + e^x}, \text{ where } X \sim N(\mu_1, \sigma_1^2).$$

A problem with this approach is that it is not easy to specify the parameters μ_1 and σ_1 from mean and standard deviation. Moreover, this approach is not easy to use with some distributions (e.g., the lognormalpower and probitnormal distributions)[8]. In addition, most available random number generators are designed to generate variates over the unit interval and then transform them to the required range (using a linear transformation), rather than generating data directly over the required range as expected in the above approach.

The most straightforward approach for any distribution will be to generate data over the unit interval and transform them to the desired interval using a linear transformation. If this approach is to work, it is necessary to transform the required mean and standard deviation to the unit scale (using the inverse of the above linear transformation), use those transformed mean and standard deviation to specify parameters for the distribution, then generate data over the unit scale, and transform

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[8] One might suggest, by defining lognormalpower distribution as $S = A^{\exp(x)}$, where $x \sim N(0, \sigma^2)$, and $0 < A$, we can directly generate non-negative lognormalpower variates, without restricting to unit interval. However we experience some problems in specifying A and s parameters here. Also, this does not offer the flexibility of fixing lower and upper boundaries at specified levels. This approach is not easy for the probitnormal distribution.

them back to the required range[9]. This back transformation to the required range will take the form of

$S = L + (U - L)y$, for beta distribution,

$S = L + (U - L)\frac{e^x}{1+e^x}$, for logitnormal distribution,

and $S = L + (U - L)\Phi(x)$ for probitnormal distribution,

where y is the beta variate over the unit interval, $X \sim N(\mu, \sigma^2)$, and $\Phi(x)$ is the standard normal cumulative function of x. For the lognormalpower distribution, this will take the form of

$S = L + (U - L)A^{\exp(x)}$, where $x \sim N(0, \sigma^2)$, $0 < A < 1$.

5.6.8 Possibility of generating correlated vital rates

Although the presence of cross-correlation and auto-correlation is considered to be realistic in population models, correlated vital rates are rarely used in representing environmental stochasticity due to practical constraints (section 4.1.4). For this reason, this thesis did not pay much attention to the subject of modelling environmental stochasticity and autocorrelation together. Since iid distributions limit the usefulness of it to some extend in situations where weather patterns are autocorrelated, I shall investigate the possibility of generating correlated vital rates from the distributions studied in this chapter.

There are standard techniques to generate correlated vital rates from the normal distribution (e.g., Burgman et al. 1993). These techniques can be adapted to generate correlated vital rates from distributions that are based on the normal distribution. Todd and Ng (2001) detailed how cross correlated survival rates can be generated from the probitnormal distribution using these techniques. This method requires additional information: parameters for the distribution that describes the second vital rate, and correlation parameters between the two vital rates. The logitnormal and

[9] In the case of the beta distribution, unlike in other distributions, it is not necessary to transform the required mean and standard deviation to the unit scale before specifying parameters. Parameters specified using the required mean and standard deviation can directly be used to generate variates over the unit scale, and transform them to the required range. This is because linear transformations do not alter the shape parameters of beta distributions (Evans et al. 1993).

lognormalpower distributions are based on the normal distribution, and they follow the same general steps as for the probitnormal distribution with the major difference being that they use different transformations to the standard normal cumulative transformation. For this reason, it should be possible to adapt the method of Todd and Ng (2001) to generate cross-correlated data from these distributions, provided the required additional information is available.

However, it is not known how to generate correlated random variates from the beta distribution. Doak et al. (1994) avoided this difficulty by assuming the variations in all vital rates were driven by a common environmental factor, and simplifying the correlation structure among vital rates to individual correlations with that environmental factor. Gross et al. (1998) gave another method to generate correlated beta variates, in which they first generated standard normal variates with the required correlations, and then transformed them to beta variates with the required mean and standard deviation. However, this transformation is likely to alter the correlation coefficients severely for some combinations of means, variances and correlations. To my knowledge, no method to overcome these shortcomings has been developed yet. Therefore, it is impossible to use the beta distribution (or any other distribution based on the beta distribution) in generating correlated vital rates.

5.6.9 Suitability of standard deviation in representing variability of vital rates in population model

One of the problems of using standard deviation as a measure of the variability of survival rate is that the maximum possible standard deviation depends on the mean survival rate (m). Therefore, as seen from equation 4.3, even if a given standard deviation (s) is only a small fraction of the maximum possible standard deviation (s^*) when the mean is close to 0.5, the same standard deviation can be a very large fraction (or above the possible maximum) when the mean survival rate is close to zero or one. For example, equation 4.3 implies that if m = 0.80 then s = 0.35 is possible but if m = 0.95 then s = 0.35 is impossible. A measure of variability without this difficulty will be the variability as a fraction of its possible maximum. This is equivalent to using

variance on an odds scale. Link (1999) used this idea by defining a parameter which he called the concentration parameter (θ) to represent the variation. θ is a measure of compactness (rather than a measure of variability), and is defined as $\theta = \frac{s^* - s^2}{s^2}$.

When the interval is unity, which is the case for survival, $\theta = \frac{m(1-m) - s^2}{s^2}$. The greater the concentration parameter, the smaller the standard deviation, and so the realised values of the vital rate are more closely clustered about the mean. θ is considered to be a better measure of variation than s when comparing finite distributions with different means, because, unlike s, the maximum possible or minimum possible θ does not depend on the mean. Therefore, as seen in Figure 5.8, θ is comparable between mean levels, and hence pdfs with the same values of θ will have much more similar shapes for different levels of the mean.

We can see from the formula for θ that θ does not change when survival is replaced by mortality (i.e., when mean is replaced by 1-mean), therefore it can be used regardless of whether survival rate or mortality rate is represented in a population model. It could also be argued that standard deviation in fact does not reflect the 'true measure' of variability unless the distribution is normal, because the concept of standard deviation was developed in the field of statistics in parallel with the concept of normally distributed data.

In this work I used the standard deviation as the measure of stochastic variation in vital rate because it is the most commonly used measure to represent the variation in ecological studies. It measures the variation from the mean, regardless of the level of the mean (or level of maximum possible variance). Representing variability by the standard deviation in this study does not invalidate the findings of this chapter, because we did not intend to compare the effect of variation at different mean levels; rather we focused on the effect of the distribution that is used to represent the variability.

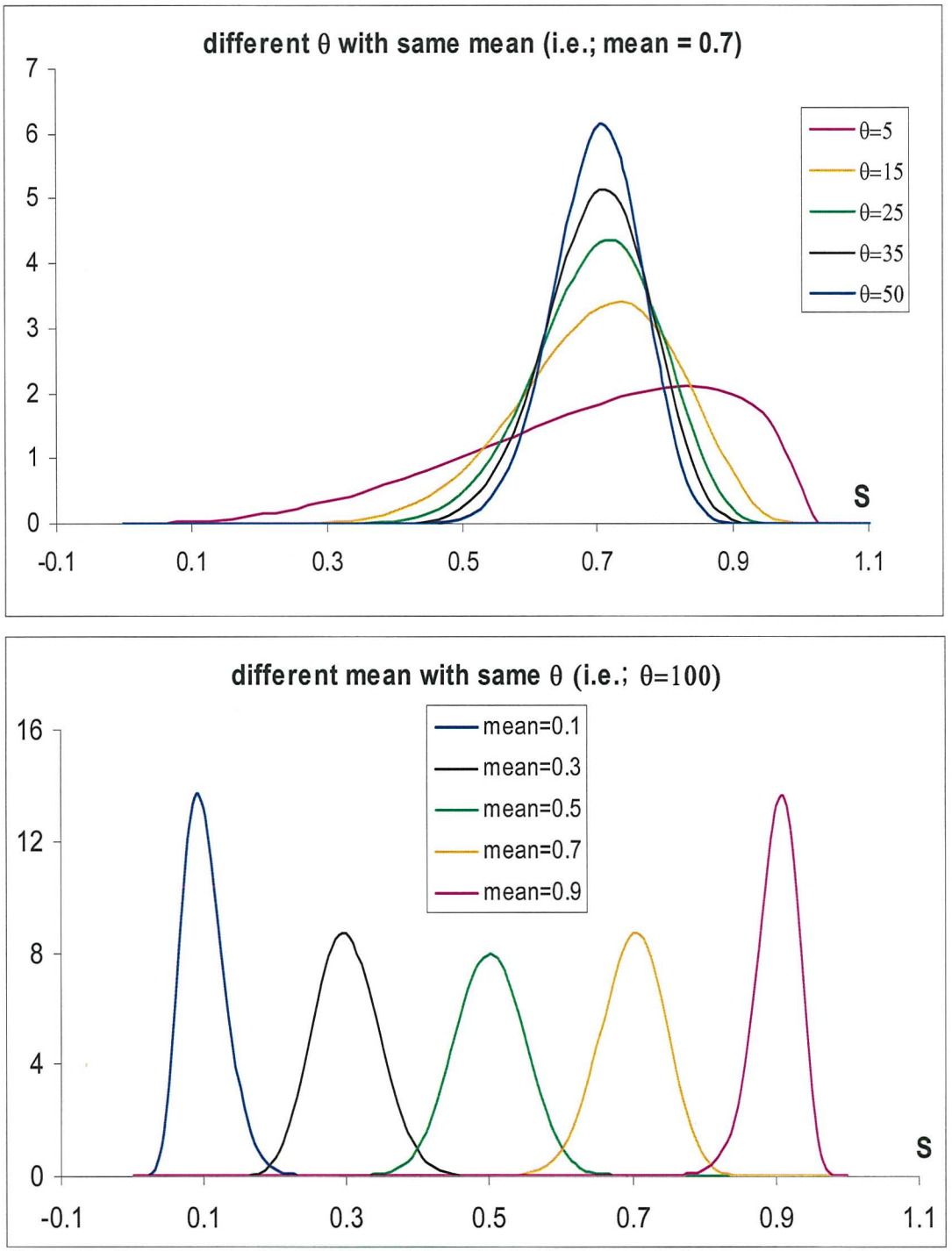

Figure: 5.8: Probability density functions of the beta distribution to illustrate the convenience of representing variability by θ. The top panel shows the pdfs of distributions with the same mean but different θ, while the bottom panel shows the pdfs for different means but the same θ.

5.6.10 The relationship between U shaped distributions and risk of extinction

As seen in this chapter, when the mean survival rate becomes larger (while s remains the same) most distributions turn from unimodal to J shape and then to U shape. It is easy to see that random variates generated from J shaped distributions are concentrated near the upper boundary (assuming the mean is closer to upper boundary than to lower boundary), while data from U shaped distributions are bimodal. For this reason, one can suspect that U shaped distributions might predict higher quasiextinction risks than J shaped distributions. If this happens, it is contradictory to the widely accepted result of "if the level of stochasticity remains the same, higher mean survival lowers the quasiextinction risk". This was examined by estimating quasiextinction risk for a series of increasing mean levels where pdfs change from J shape to U shape (keeping s the same). Results in Figure 5.9 show that a higher mean produces a lower risk even if the pdf turns from a J shape to a U shape. This is in agreement with the generally accepted result.

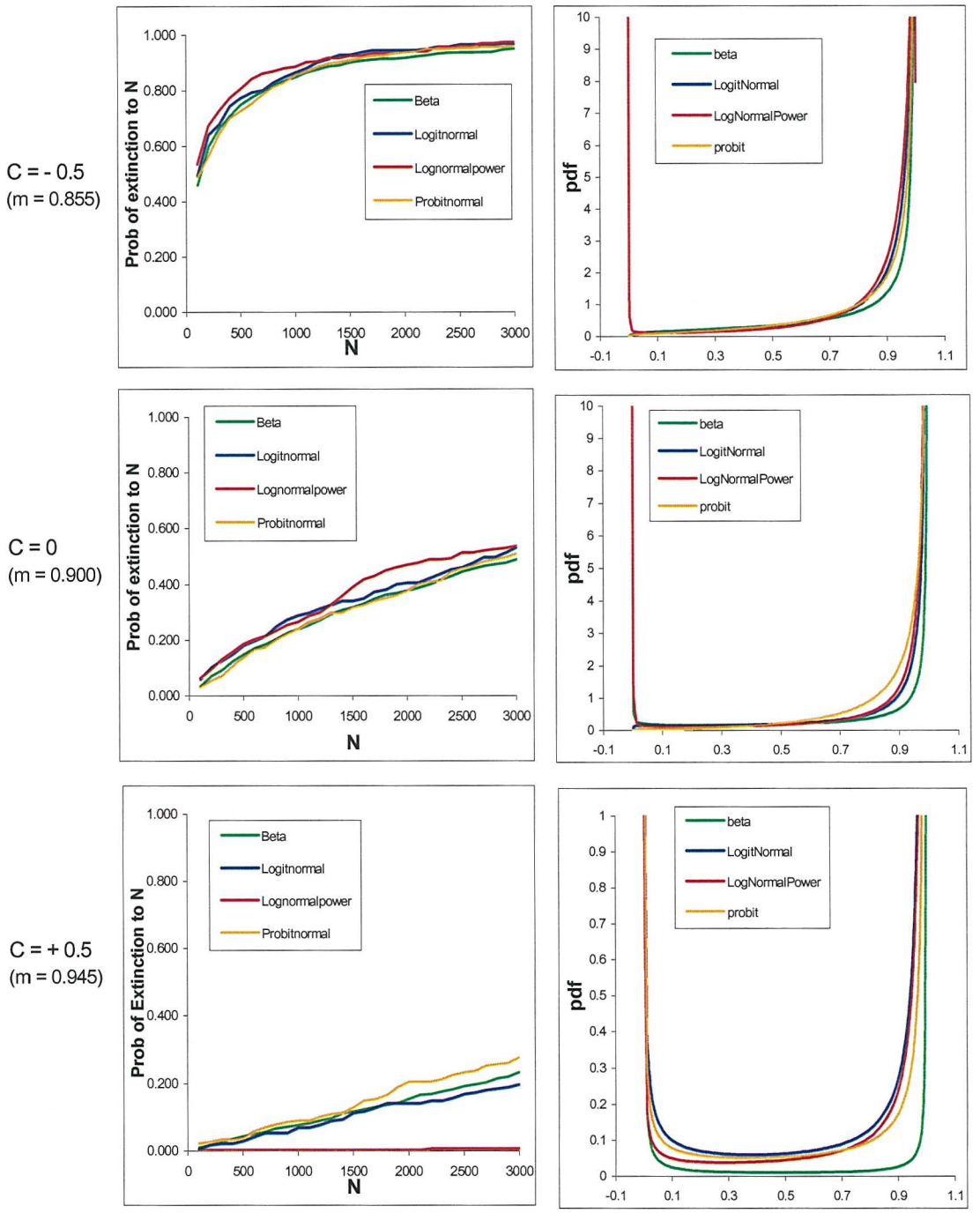

Figure 5.9: Probability of quasiextinction curves for different mean levels when *s* remains constant close to its possible maximum (s = 0.223; 98% of its possible maximum at C = +0.5). The corresponding pdfs are also shown. At the lowest mean level all the distributions are J shaped (except lognormalpower distribution), while at the highest level of mean all distributions are U shaped. This figure shows that a higher mean produces a lower risk even if pdf the changes from a J shape to a U shape. (The risk curve for lognormalpower distribution is invisible at highest mean as it is on the horizontal axis).

5.7 Summary and conclusion

In this study I have not considered all the distributions that can be used to represent the environmental variability, and therefore cannot rule out the possibility of other distributions being better in some respects than the ones considered in this study. To my knowledge no prior efforts have been devoted to explicitly assess the effect of the choice of distribution on the population outcome. It is rare to find literature where the selection of the distribution is explicitly justified. The choice of distribution appears to be driven by custom, or researcher's convenience and experience. For this reason it is impossible to see how compatible the results presented here are with those of others.

The beta distribution has several advantages over the other distributions considered. Parameters can be specified analytically from the mean and standard deviation, and so the model implementation is quicker and not dependent on the convergence of numerical procedures. Because parameter specification is exact, the generated random variates will have the intended statistical properties (if used with a reliable random number generator). Since the lower and upper boundaries of the vital rate can be fixed at desired levels, the distribution can be used with vital rates that are distributed over any interval. All combinations of mean and standard deviation (that can be defined on the interval of interest) can be used with the beta distribution.

The beta distribution also has the advantage that the shape of the distribution can be determined in advance from the mean and standard deviation. This helps in avoiding the use of unrealistic combinations of mean and standard deviation when other available information suggests certain shapes are impossible. For example, if available information suggests a certain shape is unlikely for the population under consideration, then measures can be taken to avoid combinations of the mean and standard deviation that lead to the unlikely shape being used in the model.

Although the beta distribution can take a wide variety of shapes depending on the mean and standard deviation, it cannot take all the shapes that are useful in environmental modelling, e.g., the bimodal shape may be of interest when alternating between 'good' and 'bad' years. A composite beta distribution, which is a

combination of more than one ordinary beta distribution, can be used to get shapes like this that cannot be achieved by a single beta distribution. This idea of composite distributions is common in modelling catastrophic events (e.g., Breen et al. 2003).

Both the logitnormal and probitnormal distributions perform similarly to the beta distribution in most respects. Some difficulties with them are that their parameter specification is numeric and not exact, and is slow to implement in a population model. In addition, their shape cannot be determined in advance from the mean and standard deviation. However, their advantage over the beta distribution is that correlated data can be generated from these distributions.

Finally, the lognormalpower distribution, which is the distribution proposed in the latest NZSL model, 'performs poorly' when compared to other distributions examined. In addition to the difficulties seen with logitnormal and probitnormal distributions, this distribution has an inflexible and undesirable shape, and often predicts a different fate for the population.

6 Density Dependence

6.1 Introduction

6.1.1 Introduce the chapter

In previous chapters we looked at how environmental stochasticity can be represented in population models. This chapter focuses on another similarly important characteristic of population, density dependence (DD). I start by making a few introductory remarks about density dependence and outlining the scope of the chapter. I start from the fact that density dependence is generally represented in models by specifying one or more of the vital rates as functions of population density. Here I aim to develop an ecologically motivated criterion that can be used in objectively selecting a function for that purpose. With this aim in mind, I first examine the desirable properties of DD functions for both count-based and demographic models. Then I explore possible methods that can be used in testing a given function for each of those properties.

6.1.2 Introduce density dependence

As mentioned in chapter 3, no population can continue to grow indefinitely. As a population approaches the limits of its resources, or as the impact of natural enemies intensifies, the annual population growth rate (N_{t+1}/N_t) responds, mainly by declining and approaching unity so that the population growth ceases. Density dependence is

the change in individuals' performance as the density of the population changes, as a result creating a dependence of a population's performance and its growth rate on its density. The concept of density dependence first came to light when Thomas Malthus (1798) recognized that any population growing at a constant rate would ultimately result in it outstripping its resources. Morris and Doak (2002, p36) stated that, "probably the single most important factor that biologists want to include in PVAs but lack data to estimate properly is density dependence".

Murray (1994) wrote that by density dependence most ecologists mean "some dependence of population growth rate on past or present densities", meaning the relationship is causal and can be either positive or negative. Huffaker and Messenger (1964) defined density dependence as "... the actions of repressive environmental factors, collectively or singly, which intensify as the population density increases and relax as this density falls....". This definition implies that the relationship is negative. Positive relations have been observed at low densities in reproduction (e.g., difficulty in finding mates at low densities) and survival (e.g., weakening the group effect in defence as suggested by some of the hypotheses for brood amalgamation), (Runge and Johnson 2002). However, density dependent models generally tend towards negative relationships between population density and the rate of increase of the population, (Varley et al. 1973, Krebs 1985, p325). Royama (1977, p26) defined density dependence as "the net rate of change of a population being dependent on the present or past density, with some form of negative feedback process", which imply causation. The earliest known record of negative feedback mechanism in biological populations is that of Verhulst in 1838 (Bellows 1981). In general terms, density dependence is the phenomenon by which the population parameters (such as growth rate, survivorship or fecundity) depend on the density of the population (Burgman et al. 1993, p74). Eberhardt (1970) stresses that density dependence is not simply an association between performance (measured by either vital rates or growth rate) and density, but is rather a causal relationship between them. Royama (1977) cites a long list of works for the definition of density dependence as causal relationships as well as associations. Royama (1992) discusses the concept of density dependence in terms of casual versus correlation connotations. As Solomon (1958) discussed, the meaning of DD varies in the literature. In this chapter we consider only negative density dependence with causal relationships.

Negative density dependence generally represents resource limitations (food, space, etc) or competition among individuals for those resources. Limited resources means a reduced per capita share for higher population levels, which in most cases leads to reduced performance of individuals (i.e., reduced contribution to population viability), or in other words, enhanced performance at lower densities. This enhanced performance at lower population densities is the biological justification for sustainable population harvesting.

Negative density dependence is an important factor to consider in assessing population viability for two reasons. On one hand, negative density dependence places a limit on how far a population can grow away from the extinction threshold. On the other hand, the growth we measure at the current population size might underestimate the growth rate the population would experience if it were reduced to a smaller size. That is, the population might be more buffered against extinction than growth rates measured at intermediate densities would imply. Therefore predictions from density independent population models could be more conservative than those that would be obtained from density dependent models (Morris and Doak 2002, p101). The opposite is true if positive DD is present. The presence of density dependence can cause a density independent PVA to either over-or underestimate the true viability of a population depending on the specifics of how the growth rate responds to density. Therefore, representing DD in population models in its 'real form' is important for reliable predictions.

6.1.3 Representing density dependence in population models

In count-based population models, density dependence is represented by specifying the growth rate as a function of population size, without direct reference to the vital rates. Demographic models, on the other hand, represent DD by specifying one or more of the vital rates as functions of population density. Even though demographic models can yield more informative predictions for most species, better predictions occur only when we have enough data to estimate the greater number of parameters that those models require. With less information, simpler count-based models can

give more or equally reliable results even for species with highly structured populations (Ludwig and Walters 1985, Morris and Doak 2002, p9). Furthermore count-based models offer an opportunity to study various ways of building density dependent population models and assessing their consequences in a simpler context. For this reason, in this chapter I consider DD in both of these groups of models.

It is relevant here to see what is meant by density. The term 'density' does not necessarily mean the size of the population or part thereof, but is only an index of the size of the portion of the population that contributes to density dependence. For example, in harvest models (e.g., fisheries, plantation) density could be measured by the biomass of the harvestable part of the population or the dry weight of the harvest, which is (in most cases) a non-linear function of the population size (Watkinson 1980). Some population models ignore the fact that the density dependence may be restricted to specific age/stage classes. A management consequence of this is that, for example, harvesting density independent classes will not activate the density dependent population regulatory mechanisms as expected by harvest models (DeMaster 1981). The majority of models assume the density dependent mechanism only acts through part of the population. In those situations 'density' could be a suitably weighted combination of sub-populations (Caswell 1989,p231). In many wild bird and mammal populations, where adults are long lived and juveniles compose a small fraction of population, it can be reasonably assumed, at least approximately, that all density dependence is exerted by the adult population density (Lande et al. 2003, p70). In subsequent discussions in this thesis I use population size as a measure of density, but the concepts are applicable for any other suitable definition of density.

6.1.4 Assumptions for the rest of the chapter

This chapter examines the properties of functions that are used to represent the density dependence. It is easier to examine them, especially in the initial stages, if those relationships are free of some of the complications that exist in ecological data, which are often correlated and influenced by measurement/sampling errors. The following sections of this chapter treat only the true density dependence. This is neither an assumption that the usefulness of examining density dependence in the

presence of correlation, measurement errors, and time lag is unimportant, but rather it facilitates the development of the chapter.

The growth rate or vital rates of a population do not response to changes in density instantaneously. For example, a population will perform more or less independently of its density until the availability of resources becomes a limiting factor, meaning there could be a long delay in responding to population changes at lower densities. While acknowledging the importance of incorporating this time lag into DD modelling, I do not intend to discuss it in this chapter.

Density dependent responses cannot always be distinguished from the stochastic variations that were discussed in chapters four and five. Even if the natural world were 100 percent predictable, the dynamics of populations with density dependent regulation could nonetheless in some circumstances be indistinguishable from chaos, if the growth rate was large enough (May 1974). Since the environmental stochasticity and density dependency are likely to interact in terms of their effects on population growth, the effect of DD on population growth is not easy to analyse (and interpret) when both stochasticity and DD are present simultaneously. McLeod (1997) showed that the concept of carrying capacity could be validly applied to deterministic or low-variance environments, but not to the dynamics in highly variable stochastic environments. Using situations without stochastic variations will simplify the discussion in and understanding gained by the thesis, and so for that reason this chapter considers the effect of DD in the absence of stochasticity.

Estimating density dependent parameters has become a problem for most researchers due to measurement errors in observations (Barker et al. 2002). This leads to uncertainty in the estimated parameters (section 3.5.2). Even though the uncertainty of the parameters related to density dependence is an important component of a population model (Slooten et al. 2000), it is not the focus of this chapter. One of the primary concerns in modelling density dependence is not having measurements for a range of densities that is large enough to estimate DD parameters (Runge and Johnson 2002). For example, no one believes that, at present, good enough data are available to identify the forms of density dependent relationships associated with the NZSL population. None of the models proposed for this population have been able to justify

the choice of relationship used. This leads to misspecification of the functional form of the existing relationship. This chapter focuses on the issue of possible misidentification of the functional form and its consequences.

Cross correlation between two or more vital rates is believed to be common in biological populations. If cross correlation is present, the value of a vital rate in a given year is dependent not only on the density but also on the values of other vital rates. Therefore the density dependent relationship interacts with the cross correlation. For the purpose of this chapter I do not consider such cross correlations. That will simplify and focus the discussion in this chapter.

6.1.5 Graphically representing density dependent data

A variety of methods appear in the literature to graphically present density dependence. The aim of these methods is to demonstrate whether or not the data show evidence of a density dependent relationship, and if so to demonstrate the properties of that relationship. A few such common methods are listed here because they are useful to generate discussion in section 6.2.

The first method is to plot population size in one year against what it was one year before, (N_{t+1} vs N_t), where any deviation from a straight line through the origin indicates density dependence. Such deviations usually show either a monotonic curve or a humped curve (Figure 6.1, panel A), corresponding to two different forms of competitions, contest and scramble, described in section 6.2.2. The slope of the relationship shows the population multiplicative rate which varies with population density.

The second method is to plot population growth ($N_{t+1} - N_t$) against the corresponding density (N_t). As in the first method, any deviation from a straight line shows evidence of density dependence, with the slope corresponding to the population growth rate. These plots generally show a positive relationship at low densities and negative relationship at larger densities (Figure 6.1, panel B).

Because no demographic information is represented in these two plots, they cannot be used in demonstrating the properties of density dependence in demographic rates, and hence can only be used for count-based models.

The third method is to plot the density dependent function against the corresponding density. In count-based models the density dependent function is the rate of population increase, $f(N_t) = \frac{N_{t+1}}{N_t}$, while in demographic models it is the value of the vital rate (S_t), where subscript t represents the time. In these plots, any deviation from a line with zero slope shows evidence of density dependence. In the presence of negative DD, such plots either decrease monotonically with increasing density or show a sigmoid relationship (Figure 6.1, panel C).

The fourth method presents the above density dependent function on the logarithmic scale. Let the 'kvalue' at the population level of N_t be defined as $k_t = -\ln(f(N_t))$ in an analogy to the kvalue of Haldane (1949) and Varley and Gradwell (1960)[1]. When k_t is plotted against $ln(N_t)$, density dependence is shown by deviation from a line with zero slope. When DD is present, the relationship often becomes linearly or exponentially (but not logarithmically) increasing (Bellows 1981), (Figure 6.1, panel D).

The fifth method is to plot k_t against N_t. Density dependence is shown in these plots by deviating the relationship from a line with zero slope. When density dependence is present, such plots usually show either a logarithmically increasing or exponentially increasing shape (Figure 6.1, panel E).

We will come back to these plots in section 6.2 to see how they can be used to explore some desirable properties of DD functions.

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[1] Varley and Gradwell (1960) defined the kvalue (meaning 'killing power' in the context of mortality event) as "the difference of logarithms of number of individuals before and after an event". Therefore, $k = \ln(N_t) - \ln(N_{t+1})$, or $k = -\ln(SurvivalRate)$. Varley et al. (1973) applied it to reproductive rate, and Reeve et al. (1998) used it in both count-based and demographic contexts. I used the analogy of it to define the kvalue in general as $k = -\ln(f(N))$, where $f(N)$ refers to DD function.

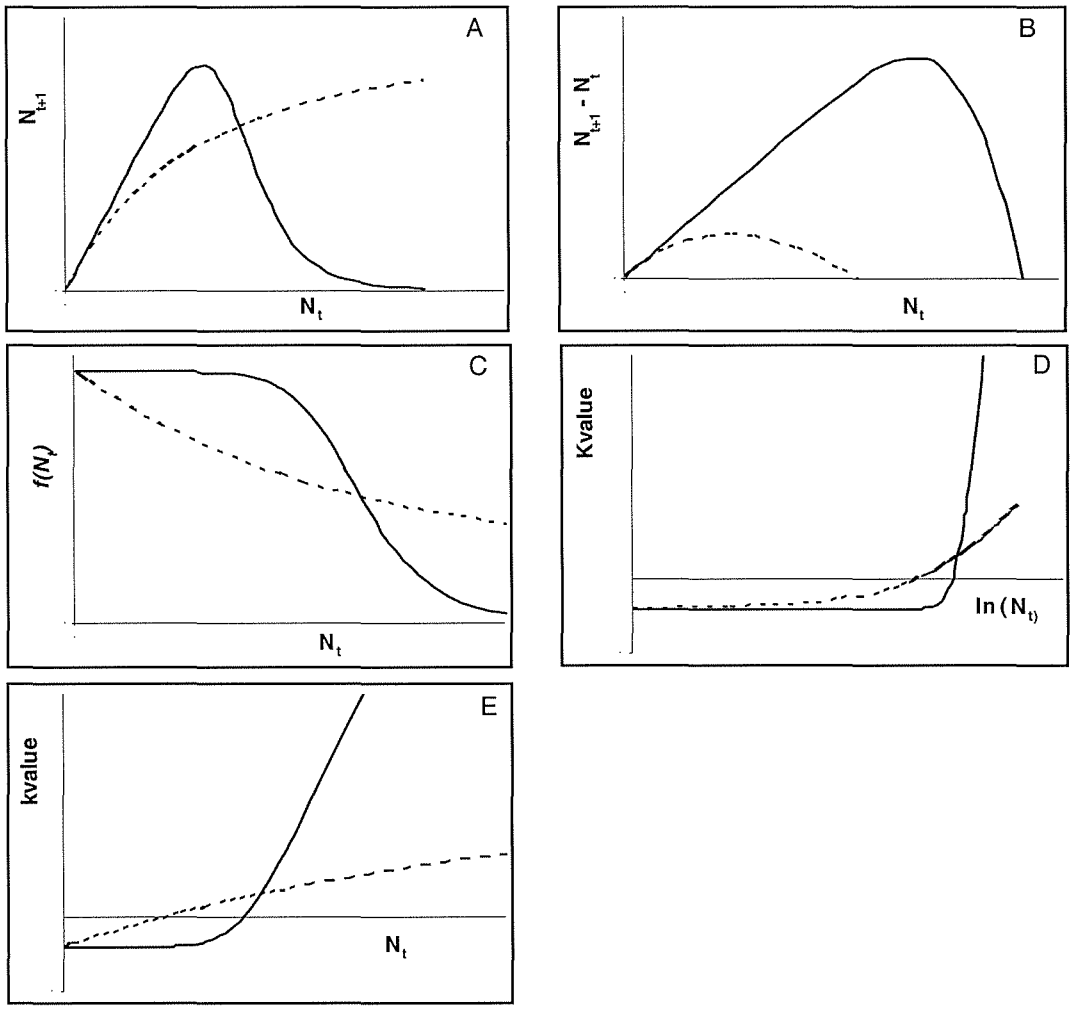

Figure 6.1: Sketch plots showing five different ways of graphically representing density dependence (section 6.1.5). These plots depict the theoretical relationships correspond to the DD function, not to the relationships observable in field data that are usually subjected to time lag. The curves in each panel show two common shapes each plot could take. The shapes shown in dotted lines mostly associated with contest competition, while the solid lined shapes mostly associated with scramble competition (as described in section 6.2.2).

6.1.6 The flow of the chapter

As stated in section 6.1.3, density dependence is represented in population models by specifying vital rates (or growth rate) as functions of population density. In reality different vital rates may be affected differently by the density of different stages (Caswell 2001, p512), so the same DD function or the same measure of density may

not be suitable for all vital rates even in the same population. So how do we know which function best describes this relationship for a given vital rate in a given population? Theoretically, this question could be addressed by estimating the DD curve if we have estimates for that vital rate for a range of corresponding densities. While it is better to estimate a population specific function whenever possible, such estimates are generally difficult to obtain as they would require density and vital rate data covering whole range of population sizes that can be experienced by the population in the future. Even if such data were available, they usually pose numerous well-known problems during the estimation process (Bellows 1981, Pollard et al. 1987, Barker et al. 2002), which we do not intend to discuss here. Therefore modellers are often compelled to use their own subjective judgments in deciding which function is best suited in describing these DD relationships. In such situations it is useful to have an objective criterion to select a function. The aim of the rest of this chapter is to develop such a criterion that can be used to distinguish between appropriate and inappropriate functional forms when there is no sufficient information in the data to make a selection.

With that aim in mind, in section 6.2 of this chapter, I explore the desirable properties for DD functions. In the light of these properties I evaluate commonly used DD functions in chapter 7, in order to help modellers in selecting one function over another.

6.2 Desirable properties of DD functions

Density dependence is represented in population models by specifying one or more vital rates (or the growth rate in the case of count-based models) of the population as a function of its density. These functions take the general form of $S_t = f(N_t)$, where N_t is the population density and S_t is the density dependent vital rate, both at time t. In the case of count-based models, S_t is the population multiplication rate, which is customarily denoted by λ_t (or the population growth rate of $ln(\lambda_t)$), and hence the population model becomes $N_{t+1} = f(N_t)N_t$. I refer to $f(N)$ as the density dependent

function (DD function), and the rest of this chapter focuses on properties that are desirable to have in this function.

Available ecological data (i.e., estimates of vital rates and corresponding population densities) are often insufficient to understand the nature of the DD function. Difficulties in estimating parameters for DD functions from those data are very common, and I do not intend to discuss these difficulties in this thesis. Data can often be fitted to several differently-shaped functional forms equally well. These functions can take quite different shapes beyond the data range, and this can have important impacts on the fate of the population (Runge and Johnson 2002)[2]. This is largely due to the fact that the range of densities covered by the data is often small. Therefore, the statistical fit of a function to the data is not a suitable basis for selecting one DD function over another. In those situations, even though data exists, they are of little help in selecting a function, compelling modellers to make subjective judgements about their suitability. The aim of this part of study is to develop an objective criterion to assist with that selection by using biological knowledge to bracket possible functional forms (or to rule out inappropriate functional forms). First I first consider, on the basis of biological acceptability, what properties are desirable in a DD function in depicting the nature of density dependence, and then I examine how can we determine whether a given function possesses those properties.

A given population will not necessarily have all the characteristics that we discuss in this chapter. Therefore, a DD function for a given population need not be able to represent all the possible characteristics, but only characteristics present in that population. That is, the properties of the function need to be matched to the population characteristics. Here I will develop a method of matching functional properties to population characteristics. However, when modelling a population with unknown characteristics (which is a very possible scenario) there is no way of matching the functional properties to population characteristics. In such situations either we have to experiment with different functions, or else use a DD function that is flexible enough to represent all possible characteristics of the population (or as many as possible). Although there is no guarantee that a function capable of

[2] This is analogous to the well known statistical fact that no regression can be extrapolated beyond the range of the data.

representing all characteristics exists, at least we should be able to eliminate identifiably 'inappropriate' functions. For that purpose, I will now develop a way to assess DD functions on their properties.

6.2.1 Shape of the function

Density dependent relationships can be expressed in terms of the shape of the DD function (i.e., the shape of the curve that depicts the relationship between the value of the function and the density; see Panel C in Figure 6.1). If the density dependence represents the resource limitations, it does not activate until the population reaches a certain level, the *critical density* (Hassell 1975). Therefore, the strength of the DD can be assumed to be minimal at the smallest density, and increases with increasing density. The slope of the DD curve represents the strength of the DD relationship, because the larger the slope, the more sensitive the vital rates are to changes in density. This implies that the slopes of DD curves are likely to be minimal at the lowest density, and become larger (in magnitude) with increasing density (Figure 6.1, panel C, solid curve).

If the slope continues to increase in this way, the vital rate ultimately becomes zero at a certain (sufficiently large) density, leading population to collapse. While this can plausibly happen, and has been observed with count based data, e.g., *Stegobium paneceum* population in (Bellows 1981) and winter moth (*Operophtera brumata)* population in (Varley and Gradwell 1968), there are populations that behave differently. In those populations, at least a small fraction of individuals survive and reproduce even at very large densities. This is the likely situation for most long-living k-selected species. This implies, in those populations, the strength of DD (the slope of the DD curve) does not continue to increase with increasing density. Slope becomes maximal at the density where competition for resources becomes substantial, and decreases at larger densities.

We are interested here in the characteristics of a general DD function that can describe a wide range of dynamics of populations. Such a function should be able to

explain both of the above dynamics. Therefore, it may be more plausible to assume that the effect of density under such a function sets in at a maximum rate around some characteristic density (Getz 1996), and gradually reduces when the density is further away from that characteristic density (see Figure 6.1, panel C, solid curve). That implies the vital rate of the population has an inverted sigmoidal form as a function of population density. This does not mean non-sigmoidally shaped DD functions are not plausible. In fact a wide range of dynamics can be described by a collection of non-sigmoidal functions. What is meant here is that a single sigmoidal function can describe a wider range of dynamics than can be described by a non-sigmoidal function, and are therefore more suitable as a general function[3].

The sigmoidal property can be examined visually from plot 3 in section 6.1.5 (Figure 6.1, panel C). They can also be tested using derivatives of DD functions with respect to population density. For sigmoidal shaped functions, the first derivative should be zero at both zero density and the largest density, and at its maximum (in magnitude) at a certain intermediate density, which is usually near the carrying capacity (Bellows 1981, Getz 1996). Also, the second derivative of the function changes from negative to positive with increasing density. In chapter 7 I evaluate this property for some commonly used functions.

6.2.2 The nature of competition: contest and scramble

Nicholson (1954) identified two extreme forms of intraspecific competition between individuals for resources, and termed them as contest competition (i.e., compensatory competition) and scramble competition (i.e., over-compensatory competition). In contest competition, individuals compete (i.e., contest) for resources on a 'win or fail' basis. Each 'winning' individual gets the full share of resources it requires, any animal who doesn't get enough resources will 'fail' to become successful (fail to survive or reproduce), (Varley et al. 1973). Competition among some breeding birds

[3] For example, DD becomes stronger with increasing density under the θ-logistic function when $b > 1$ (section 7.3). As opposed to sigmoid-shaped functions, the strongest DD occurs at the largest density instead of at a intermediate density. This agrees with the concept of critical density, and seems biologically plausible for populations that collapse at high densities, which is a feature generally associated with scramble competition (section 6.2.2). What we are looking for are properties of a general function that can be used for populations with both contest and scramble properties. Sigmoidal shape is a desired property for such a general function.

for a limited number of nesting sites is an example of this (Lebreton and Clobert 1993, Broom et al. 1996, Kemp and Wiklund 2001, Kokko et al. 2004, Vogel 2005), Assuming no other limiting factor, the birds' reproduction will not be affected as long as the number of breeding pairs is lower than the number of available nesting sites (i.e., the critical density). However when the number of pairs exceeds the number of nesting sites, the reproductive ability of those excess pairs will be seriously affected. A general feature of this form of competition is that once the critical density is exceeded, the number of successful individuals is determined by the resource level, and is independent of the number of individuals that entered the competition. That is because each additional entry is compensated by a failure. Therefore the population does not collapse due to a very high number of individuals entering the competition (Broom et al. 1996). However, in practice strict context competition is unlikely be observed; once the all optimal quality cites are occupied additional pairs move to lower quality cites resulting reduced reproduction instead of no reproduction.

In contrast, in a scramble form of competition, individuals share resources equally. If the share per individual is sufficiently large (which happens below the critical density) they will all be successful (survive or reproduce), whereas above the critical density none will get a sufficient share so all (or most) individuals will fail to succeed (fail to survive or reproduce). The general feature in this form of competition is that the population could collapse if a large number of individuals enters the competition (Figure 6.2). Whether or not a large number of individuals enters the competition is largely determined by the shape of the DD function (assuming no external inputs like large immigration pulses). For example, as shown in the second panel of Fig 6.2, if the population growth rate is large enough in previous years, it is possible to have a population size much larger than can be supported by available resources. Such shaped DD curves can lead populations to an unstable status or bifurcation (May and Oster 1976). The existence and intensity of density dependent negative feedbacks governs the stability of the system and its ability to withstand perturbations (Lebreton and Clobert 1993). The range of parameter values of some functions (hence the resultant shapes) that lead populations to unstable and bifurcation phase are discussed in the literature (e.g., Alsop 1998, Caswell 2001). Having a large growth rate, a very large number of individuals entering the competition, and consequently the

population entering the bifurcation phase are artifacts of the shape of the DD function which the dynamics is subjected to.

According to this description, populations do not show any difference between contest and scramble competitions until the critical density is reached, and until then, all populations show density independent dynamics. In practice, both in contest and scramble, there is a smooth transition region from density independent to density dependent dynamics. Also, no population is either 100% contest or 100% scramble, rather, most populations are at a point on the contest-scramble continuum, but generally having features of one form of competition more than the other. Therefore it is more meaningful to place each population and corresponding DD function at a suitable place on a continuous contest-scramble scale, rather than grouping them to one of two distinct groups of either contest or scramble. However, to my knowledge, there is no standard way that can be used to measure the contest-scramble status of different populations or different functions, and hence in practice each function is said to be either contest or scramble. Describing a population in one of these forms only implies this form of competition is more prominent; it does not mean they do not have the other form of competition.

Scramble competition can contribute a more complex component to population dynamics than contest competition. Sometimes this complexity is very difficult to distinguish from the effect of inherently unpredictable forces such as environmental stochasticity (Morris and Doak 2002, p323). For this reason, populations subjected to contest and scramble competitions predict quite different dynamics. For example, in chapter 7 we will see that, when all other factors remain the same, the Beverton and Holt function (which is contest) predicts an asymptotically stable population, while the Ricker function (which is more scramble) can predict a fluctuating population even without any form of stochastic variation. If one wants to build a model to predict the dynamics of a population where the form of competition is unknown, the DD function used in the model should be able to (or be flexible enough to) describe both of these forms of competitions. Therefore we now see how a given DD function can be examined for the nature of competition represented by it.

Three methods can be suggested to identify the form of competition described by a DD function (as detailed below, and summarized in Table 6.2). Since no population is purely contest or purely scramble, any method used to identify the nature of competition has to consider a sensible 'cut-off limit' that demarcates 'mostly contest' from 'mostly scramble'. Because of the essentially arbitrary or incompatible nature of such limits, these methods yield to different conclusions in some situations. Each method is related to one of the DD plots introduced in section 6.1.5. Interestingly, the plot that is most commonly used to describe the shape of the function (the *f(N)* vs N plot, plot 3 in section 6.1.5) does not clearly distinguish contest competition from scramble.

6.2.2.1 Method 1

This method is based on the relationship between N_{t+1} and N_t, the relationship shown by the first plot in section 6.1.5 (Figure 6.1, panel A), and hence can only be used for count-based models. Note that plots in Fig 6.1 depict the theoretical relationships corresponding to the DD function, not to the relationships observable in field data that are usually subjected to numerous sources of variations, specifically to time lag in density dependent feedback mechanism. If the dynamic is density independent then N_{t+1} and N_t are linearly related. If the competition is contest the curve monotonically increases towards an asymptote. If the competition is scramble, overcompensatory nature produces a bloom hence the curve becomes a humped shaped and falls down to zero at high densities. Since populations generally have a combination of these two extreme types of competitions, the curves usually take an intermediate shape (Figure 6.2).

Therefore the slope of this curve, or the value of first derivative of N_{t+1} with respect to N_t evaluated at high densities, is useful in determining the contest/scramble status[4]. If it has a large negative value then the competition is scramble. If it is positive and approaches zero, the competition is contest. If it is negative and approaches zero then the nature of competition is not clearly distinguishable.

[4] Here the term 'high density' implies population size is large enough to activate the density dependence in full, which is clearly larger than the critical density (Varley et al. 1973).

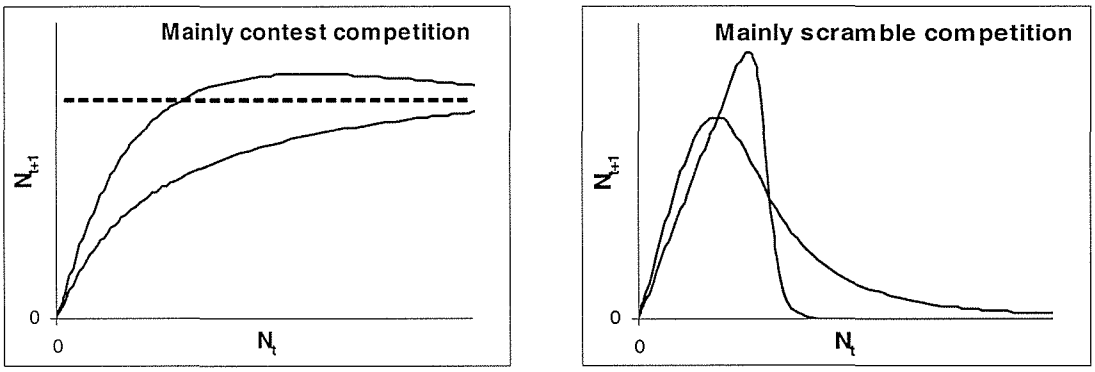

Figure 6.2: A sketch showing examples of possible relationships between N_{t+1} and N_t. The left and right panels show mainly contest and mainly scramble competition respectively. Two curves in a single panel show the same form of competition with different strengths.

A function depicts one form of competition more than the other depending on the relative closeness of the slope (at high densities) to two extremes. Suppose we want to find the form of competition represented by a set of data, can we use the estimated slope of the curve at high densities? How far away from zero should it be in order to determine if it is scramble? Do we know the slope for a function that represents 50% contest and 50% scramble as the cut-off point? For this reason this method is not a straight forward way of identifying the nature of competition. As we see later, similar weaknesses exist in other methods too.

6.2.2.2 Method 2

This method is based on the relationship shown by the fourth plot of section 6.1.5. In these plots density independence is indicated by zero slope, and negative density dependence is represented by increasing relationships. Data plotted in this way often produce linearly or exponentially (but not logarithmically) increasing curves (Figure 6.1, panel D). When the slope of this curve at high densities is near unity, contest competition is implied (Varley et al. 1973, p26). If the plot increases exponentially (when the slope becomes much larger at large density) scramble competition is implied (Varley et al. 1973, Hassell 1975). As the slope changes from 1 to infinity the model turns gradually from 100% contest to 100% scramble. Therefore the slope (i.e., the magnitude of first derivative evaluated at large N) can be helpful in determining

the nature of competition. Reeve et al (1998) used this method with both count-based and demographic data to determine the nature of density dependent competition. Similar to method one, there is no objective 'cut-off slope' that demarcates contest competition from scramble competition.

6.2.2.3 Method 3

This method is based on the fifth plot of section 6.1.5, the relationship between kvalue and population density (Figure 6.1, panel E). Any deviation from zero slope shows some form of density dependence. The contest competition is represented by logarithmically increasing plots while exponentially increasing plots represent scramble competition (Bellows 1981). This implies that the second derivative of kvalue with respect to density is negative for contest competition, and positive for scramble competition. When it is zero no particular form of competition is prominent. This provides a cut-off level that represents both forms of competition in equal proportion (provided the first derivative is not zero which implies density independence). A general DD function should be able to describe both of these types of competitions, so the second derivative of kvalue should be able to take both negative and positive values at different levels of the parameters.

6.2.2.4 Interrelationship between methods 2 and method 3

One notable feature of method 3 compared to the other two methods is that it uses a specific cut-off level to demarcate mostly contest from mostly scramble. We can use this to derive a corresponding cut-off for method 2.

The cut-off used in method 3 is $\frac{d^2k}{dN^2}=0$, where k represents the kvalue. $\frac{d^2k}{dN^2}=0$ implies $\frac{dk}{dN}=C$, which leads to $\frac{dk}{d\ln(N)}=CN$, where C is a positive constant because k and N are positively related. This means that the slope of the relationship between kvalue and ln(N), which is the slope of the plot used for method 2, increases

proportionally to N. This provides a cut-off level for method 2 that is comparable to what is used in method 3:

- if the slope of the kvalue vs ln(N) curve is increasing faster than N, (or equivalently, if the curve increases faster than exponentially) then competition is scramble,
- if the slope is increasing slower than N then the competition is contest,
- if the slope increase is proportional to N then both forms of competitions are equally represented.

Note that this cut off level derived for method 2 does not provide a specific 'cut-off slope' rather it gives a 'cut-off rate for the slope's increase'. For this reason, the use of this cut-off is not simple. Therefore, when method 2 is used, it is common to ignore this cut-off and to say, when slope goes from one to infinity competition goes from purely contest to purely scramble (e.g., Varley et al. 1973).

Method 3 is easier than method 2 because it has an objective cut-off, and needs to find only the sign of the derivative, and not the magnitude. In the next chapter we meet a few situations where methods 2 and method 3 lead to different conclusions. Therefore, in that chapter we use combinations of these methods to evaluate the nature of competition described by commonly used DD functions.

6.2.3 Population stability

Population regulation and stability is a combined result of density dependent effects in all vital rates. Due to this combined effect, it is impossible to explain the population stability through the DD effect of a single vital rate independently of the others. If only one vital rate is density dependent (i.e., all others are density independent) then the population stability can be discussed in terms of the DD function more easily. The most straightforward situation is in count-based population models where the combined effect of all vital rates is condensed within the DD of the growth rate. In this section I discuss population stability in context of count-based population models.

Population regulation and population stability, which some populations naturally achieve through a density dependent process, is a main focus of some population

dynamics studies. Density dependent regulation usually assumes that the population tends to adhere to a fixed-point attractor, such as carrying capacity or equilibrium population level (Caswell 1997). In count-based DD functions, the equilibrium population level (K) is defined as the value of N when $f(N)=1$. Such a population level may or may not exist for a given DD function[5]. A population is said to be locally stable at K if it tends to return to K when perturbed, or is said to be unstable at K if it tends to move away from K. Population is neither stable nor unstable if it fluctuates between two or more population levels; i.e., there is no such fixed population level if the population is subjected to limit cycles.

Not all animal populations have the same stability property. They could show more complex forms of dynamics, such as stable and unstable equilibriums, attractors, periodic or damping cycles, bifurcation, and chaos (Caswell 1997). The relationship of density dependent functions to various dynamical behaviours has been discussed in the literature (e.g., May et al. 1974, May 1976, May and Oster 1976, Royama 1977). For a population model, we need to select a DD function that is able to describe the dynamics of the population. In situations where we do not know the dynamics of the population (which is the usual case in population modelling), the DD function should be able to describe any of these possible dynamics and stability properties. Therefore we must have a way to know what sort of stability can be described by a given DD function.

There are two ways to evaluate a DD function in order to examine the stability of a population at an equilibrium point. The first is related to the derivative of N_{t+1} with respect to N_t, which in graphical format, is the slope of the first plot in section 6.1.5. May and Oster (1976) showed that, if this derivative at K is between negative one and positive one, then the fixed point K is 'locally attracting or stable'. If the derivative is between negative one and zero, the population is oscillatory damping (i.e., N_t vs t relationship is oscillating with decreasing amplitude thus approaching the stable level), while when derivative is between zero and one, the population is monotonically damping (i.e., N_t vs t relationship is either monotonically increasing or decreasing towards the stable level). When the slope steepens beyond one (either positive or negative), K becomes 'repelling' or 'unstable'. If the magnitude of the

[5] In the case of the DD functions used in demographic models, $f(K)$ need not be unity, instead it will be large enough just to balance death and birth. Since births and deaths are balanced at K, in demographic models, the value of $f(K)$ depends on the level of other vital rates.

slope is unity, there is not enough information to determine the stability (Allman and Rhodes 2004, p23). Therefore, this criterion can be used to find the range of parameters of a given DD function that corresponds to various stability properties (see chapter 7 for applications to some commonly used functions).

The second method relates to the derivative of k_t with respect to $\ln(N_t)$, which in graphical format is the slope of plot 4 in section 6.1.5 (May et al. 1974). As seen from Appendix 6A, it can be shown that the two derivatives used in methods 1 and 2 evaluated at K add to unity (i.e., the slopes of the two plots add to unity at K). Therefore,

$$\left.\frac{dN_{t+1}}{dN_t}\right|_K + \left.\frac{dk_t}{d\ln(N_t)}\right|_K = 1 \cdot$$

Therefore, like the first method, we can use $\left.\frac{dk_t}{d\ln(N_t)}\right|_K$ to evaluate neighbourhood stability at K as in Table 6.1. Even though the local stability of unstructured populations is able to be examined analytically in this way, the process is not as straightforward for structured models and numerical investigations are usually easier (Gurney and Nisbet 1998, p32, 37, 240).

Table 6.1: Evaluating neighbourhood stability at equilibrium population level.

Value of $\left.\frac{dN_{t+1}}{dN_t}\right\|_K$	**Value of** $\left.\frac{dk_t}{d\ln(N_t)}\right\|_K$	**Neighbourhood stability at K**
Less than -1	More than two	Unstable
Between -1 and 0	Between +1 and +2	Stable at K with oscillatory damping.
Between 0 and +1	Between 0 and +1	Stable at K with monotonic damping.
More than + 1	Less than zero	Unstable
+1 or -1	zero or two	Information insufficient

6.2.3 (a) Stability for multiple time periods

If an equilibrium population level K exists, *f(K)* =1, meaning that a population at K returns to the same level at each subsequent time period. Even if it does not return to the same level at each time period, some populations return to the same level periodically after a certain number of time periods. If it returns to the same level once every second year ($N_{t+1} \neq N_t$, but $N_{t+2} = N_t$), then the population has a two-year periodic cycle (i.e., population oscillates between two levels). Therefore, the population will have a p-year periodic cycle if it returns to the same level after p time periods, $N_{t+p} = N_t$. Will such a population be stable? Will it return to the same cycle if perturbed? Will it be determined by the density dependent function? To answer these questions we can generalise the above stability evaluation to multiple time periods as shown in appendix 6B.

As seen from this generalisation, the stability of the population at time period p at population level $N_{K,p}$ can be examined analytically by evaluating $\left(\left.\frac{dN_{t+1}}{dN_t}\right|_{N_{K,p}}\right)^p$ and using the single-time-period criterion given in Table 6.1, where $N_{K,p}$ is any population level that satisfies $N_{t+p} = N_t$.

6.2.3 (b) Relationship of population stability to other population characteristics

(i) Nature of competition and stability

Scramble competition could cause large populations to collapse whereas they could tolerate high population levels under contest competition. Thus, in count-based models, it seems like that the contest and scramble competitions are mostly associated with the stable and unstable statuses respectively. But, as will be seen in chapter 7, populations can be driven to unstable status under the contest competition (e.g., Haldane function), and to stable status under the scramble competition (e.g., logistic function). Therefore the nature of competition (contest versus scramble) and stability are not the same population characteristic stated in a different way.

(ii) Sigmoidal shape and stability

The negative density dependence (smaller vital rates at larger densities) tends to push the population towards a stationary population level, which is necessary for population regulation. If the DD is strongest near a certain population level, the elasticities of vital rates to changes in density are larger at that level, meaning they respond more strongly (proportionally) to density changes. This dynamic supports establishing a stable equilibrium at that level. The stronger the DD, the larger the slope in the DD curve. Sigmoid-shaped density dependent functions have their largest slopes near K. Therefore sigmoid shape and stable equilibrium appear to be compatible concepts. However, some sigmoidal functions could lead a population to an unstable status (e.g., Maynard Smith and Slatkin function, see section 7.8). In other words, it is possible to have regions of parameter space that are not asymptotically stable. Similarly, there are non-sigmoidal functions that could lead a population to a stable status (e.g., the logistic function). Therefore, the functional shape and stability are not the same property stated differently.

6.2.4 Asymmetric population growth

The term population growth is commonly used to mean the increase in population size or its biomass. The relationship between population growth and population density is quite different to the relationship between the population growth rate and density. For most populations, growth is minimal at its lowest density and at the carrying capacity, and becomes maximum at an intermediate density level (say N^*). Harvest models that aim for a maximal harvest, attempt to manage the population closer to this N^* level. Models that aim to rebuild depleted populations also try to manage the population near this level (e.g., Cooke 1994, Slooten 1996). Where is this population level relative to the carrying capacity? Is it being dictated by the function used to describe the density dependence?

If the growth curve (the curve between growth and population size as shown by the second plot in section 6.1.5) is symmetric, then maximum growth occurs at K/2. Not all growth curves are symmetric, and there is no reason for all populations to have their maximum growth at the same fractional density (Thomas et al. 1980).

Symmetric growth curves are associated with linear density dependent functions (such as the logistic function, see section 7.2). Asymmetric growth and non-linearities in growth dynamics have been observed for a variety of organisms. The maximum productivity level is usually below K/2 for most fast-breeding, short-lived, r-selected species, while for slow-breeding, long-lived, k-selected species it is often between K/2 and K (Schaefer 1954, Pella and Tomlinson 1969, Pomerantz et al. 1980), even though examples exist to the contrary. Marine mammals generally show an asymmetric population growth, with maximum growth occurring near K (Cooke 1994, Wade 1998, Givens 1999), but it is impossible to assume this situation is common for all marine mammals. For example, we have no idea of that population size for NZSL relative to its K. Population models developed for such populations should be able to represent this possible asymmetry. For example, any model that predicts a symmetric growth, or the maximum population growth at a level below K/2, is probably unsuitable for most long-lived species. The level of non-linearity of the DD function used in the model determines the population level where maximum growth occurs. A density dependence function used for a population where the nature of the population is unknown should be flexible enough to represent any of above growth curves (i.e., symmetric, asymmetric r-selected, asymmetric k-selected). A general DD function should also be flexible enough to allow the population model to achieve maximum growth at any fractional density of K. In this section I consider how to assess the flexibility of DD functions to represent various degrees of asymmetry in population growth.

A density dependence function is fully flexible in representing any form of asymmetric growth if N^* can be at any population level between zero and K. This N^* can be found in terms of model parameters by using the first derivative of the growth function $(N_{t+1} - N_t)$, with respect to population density, where $N_{t+1} = f(N_t)N_t$, and $f(N_t)$ is the DD function. It is then possible to find the minimum and maximum population level this N^* can take by varying the model parameters. This procedure allows us to see if the density dependent function can represent the maximum population growth at any population level over the entire range of populations from zero to K. In chapter 7 we use the above criterion to evaluate the ability of some DD functions that are commonly used to represent this asymmetric growth. There we

observe that, for some functions, the resultant expression cannot be solved analytically so either an approximation or numerical methods have to be used.

In this section we presented the 'asymmetric growth' concept in the context of population growth in count-based models. This idea can be generalised to demographic rates in demographic models if the DD is represented by a single vital rate. For example, if the survival rate is represented by the DD function in a demographic model, at what level of population will the highest number of survivors be expected? Similarly, at what population level will the greatest number of newborns be achieved? These are analogous to the population level corresponding to maximum growth in count-based models. Therefore, if the term 'growth' is defined appropriately, this property is relevant to demographic models too. However, when the 'growth' is influenced by more than one density dependent vital rate, the discussion becomes more complicated – that is why we selected a simpler count-based model context. Later, in section 7.2 (using the logistic function), we specifically show that this idea can be generalised to unstructured demographic models if the DD is represented only through a single vital rate.

6.2.5 Summary of properties

In this chapter I discussed at least four properties that are important for a general DD function to have if it is to describe the DD relationship in population models. They are related to the shape of the function, the nature of competition, population stability, and asymmetric population growth. I have discussed the possible ways these properties can be assessed in any given function. In that way I produced some criteria for assessing the suitability of a given function to represent the DD relationship. Before heading to the next chapter, I summarise the above properties and assessment techniques in the Table 6.2.

In the next chapter I consider some commonly used DD functions, and evaluate them using the above criteria to assess their ability to represent these properties.

Table 6.2: Summary of methods that can be used in evaluating the properties of DD functions. The sketches of plots referred to in first two columns are shown in Figure 6.1

Graphically presenting DD		Evaluating shape of DD function (Sigmoidal?)	Evaluating nature of DD competition (Contest or Scramble?)	Count base models	
Plot number (As in section 6.1.5)	Description of plot			Evaluating stability at equilibrium (K)	Evaluating asymmetric growth
1	N_{t+1} vs N_t	-	First derivative at large N_t is: large and negative ==>scramble, positive & approaches zero ==>contest, negative & approaches zero ==>undecidable.	stable at K if first derivative at K is between -1 and +1.	-
2	$(N_{t+1} - N_t)$ vs N_t	-	-	-	First derivative can be zero at various fractional density if asymmetry is flexible.
3	$f(N_t)$ vs N_t	second derivative change from negative to positive with increasing N if sigmoid.	-	-	-
4	k_t vs $\ln(N_t)$	-	First derivative at large N_t is: near one ==> contest, much larger & positive ==> scramble.	stable at K if first derivative at K is between 0 and 2.	-
5	k_t vs N_t	-	Second derivative is: negative ==> contest, positive ==> scramble, zero ==> none is prominent.	-	-

7 Properties of Commonly Used Density Dependent Functions

7.1 Introduction

In the previous chapter we discussed the properties of a general DD function that are important in describing the various forms of DD relationships in population models. Each property is related to the ability of the function to represent one or more characteristics of the population, and its flexibility in doing so. Namely, those characteristics are:

- the way that the strength of DD relationship varies with the increasing density (i.e., the shape of the function),
- the nature of competition among individuals (contest versus scramble),
- the stability of the population in the neighbourhood of the carrying capacity,
- the flexibility in asymmetric population growth.

On the basis of that, I developed some criteria for assessing the suitability of a given function in representing DD relationship (Table 6.2). In this chapter I consider some commonly used DD functions, and evaluate them using these criteria to assess their ability to represent these population characteristics in population models.

The DD functions used in population models vary widely in their structure and complexity. Not only are numerous functions in use, but there are also variations of the same function, using different parameterisations and transformations. Runge and Johnson (2002) grouped DD functions into four groups (linear, exponential, hyperbolic, and power) based on the shape of the $f(N)$ vs N relationship, and

commented that functions that belong to the power group are not biologically acceptable, and so the members of the other three groups cover the full spectrum of plausible non-depensatory functions. For this study I selected a sample of commonly used functions covering a wide range of functional forms, and this chapter evaluates the suitability of each of those functions using the criteria developed in chapter 6. A full summary of properties of all functions is given in Table 7.1 in page 203.

The format of the same function can be quite different in the literature, depending on whether it is used in continuous-time or discrete-time models. I use only discrete-time versions here, to be consistent with the rest of the thesis. Also, some of these functions are in the literature in specific formats that are more appropriate for count-based models (e.g., they use growth rate and/or population carrying capacity as parameters). I use a notation that can be used in both count-based and demographic models.

7.2 Logistic function

Gompertz (1825) introduced the concept of density dependence to population modelling through a differential equation in which the instantaneous growth rate declined with density in a logarithmic fashion. This approach has found only little favour among population biologists. Verhulst (1838) proposed a mathematically simpler version known as the continuous-time logistic differential equation for population growth. For a considerable period in the past, most continuous population growth models were based on the logistic equation or on its extensions (Yodzis 1989). The logistic model is probably the most well known population model for density dependence. The continuous version of it assumes logarithmically declining instantaneous rates with increasing population size, while the discrete version used in most matrix population models assumes a linear relationship between vital rates and population density.

The discrete version of the function can be given as

$$f(N) = d\left(1 - \frac{N}{a}\right),$$

where *f(N)* is the value of the density dependent rate at density N, *d* is the value that *f(N)* approaches when density approaches zero, and parameter *a* is the density when the rate becomes zero due to density dependent effects (Figure 7.1, panel C). When this is applied to count-based models, *f(N)* is the population multiplication rate usually denoted by λ, and *a* is the density at which λ becomes zero (i.e., the population becomes extinct). In count-based models *f(N)* = 1 at the carrying capacity (K). Therefore $K = a\left(\frac{d-1}{d}\right)$.

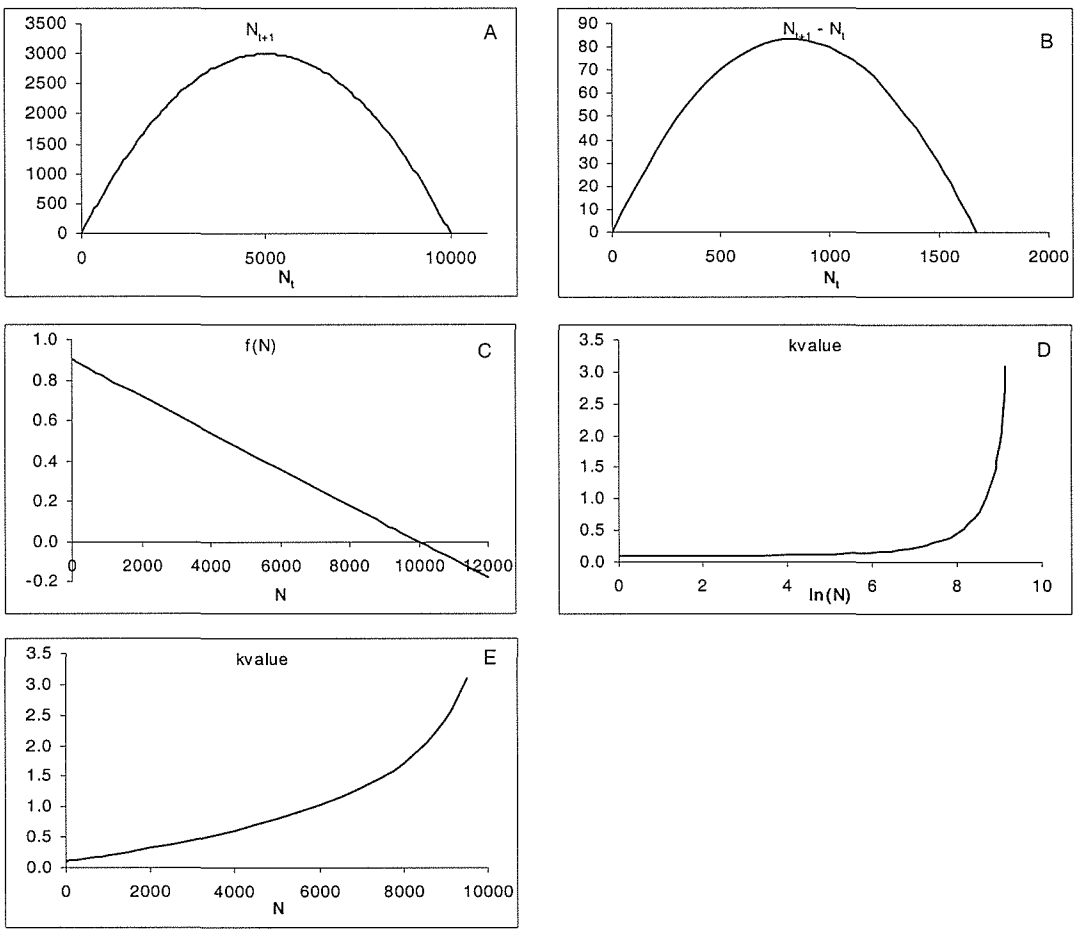

Figure 7.1: An example of density dependence under the logistic function. Panels A and B use the parameters d = 1.2 and a = 10000 to represent a growing population in a count-based model. Note that N_{t+1} is zero when $N_t = a$, and the growth is zero at a lower density, the carrying capacity. The last 3 panels use parameters d = 0.9 and a = 10000 to represent a demographic rate lower than one, such as survival rate.

7.2.1 The shape of the function and the form of competition

The plots in Figure 7.1 show the main properties of the function. These 5 plots are the same plots that were explained in section 6.1.5 in a general context. The function has a linear relationship (panel C), and so the strength of DD is the same at any population level, and there is no specific population size that the strength of DD becomes maximal as is characteristic in sigmoid shaped functions. The second derivative of *f(N)* with respect to N is zero for all N, implying that a sigmoid shape is not possible.

At large N the first derivative of N_{t+1} with respect to N_t is large and negative. The first derivative of kvalue with respect to *ln(N)* is large and positive at large N, and the second derivative of kvalue with respect to N is positive for any N, therefore this function can only represent scramble competition.

7.2.2 The stability and symmetry

In count-based models the carrying capacity is given by $K = a\left(\frac{d-1}{d}\right)$. Note that $K < a$, and no positive K exists if $d < 1$. The first derivative of kvalue with respect to *ln(N)* evaluated at K is (*d*-1). Therefore, according to the criterion in Table 6.1, the population is

- stable at K with monotonic damping if $1 < d < 2$,
- stable with oscillatory damping if $2 < d < 3$,
- Information is insufficient to determine if $d = 1$ or $d = 3$
- and unstable otherwise.

Therefore the parameter *d* governs the dynamics of the population[1].

[1] Although the discrete-time version of the function exhibits this complicated stability behaviour, the continuous logistic model does not (Edelstein-Keshet 1988, p55, Alsop 1998, p112). One explanation for this is that the time lags inherent in discrete time steps often mean the quantity being modelled cannot 'figure out' by how much it should change quickly enough, so that it overshoots its 'goal'. However more complicated continuous time models can also produce cyclic and chaotic behaviour (Allman and Rhodes 2004, p39).

Maximum growth occurs at the population size of $N^* = K/2$ or equivalently at $N^* = \frac{a}{2}\left(\frac{d-1}{d}\right)$, while the maximum growth rate occurs at the lowest density. Therefore populations characterized by asymmetric population growth cannot be represented by this function in count based models.

The above stability and asymmetric growth properties were derived assuming the model is a count-based one. In fact that restriction can be relaxed to cover all unstructured models (both count-based models and demographic unstructured models) provided the entire density dependence can be represented by a single vital rate. For example, if DD is represented by the survival rate in a demographic unstructured model then the corresponding density dependent population multiplication rate is $f(N) = S_t(1+R)$, where S_t is the density dependent survival rate at time t, and R is the density independent reproductive rate. S_t here is given by $S_t = d\left(1 - \frac{N_t}{a}\right)$, where the parameters d and a in this expression are different from those in corresponding count-based expression. Starting from this, and repeating the same steps as above, we can derive similar stability and asymmetry properties (see appendix 7). The point is that the applicability of representing stability and asymmetric growth properties by DD functions is not confined to count-based models even if they were originally presented in the context of count-based models.

The logistic model is seldom considered as a realistic biological model. Non-linearity in density dependent relationships is being observed in more and more populations, especially with marine mammal populations (Ragen and Fowler 1992). Although this is probably the most well known density dependent model that has been used in the past, it is highly unlikely it will suit a marine mammal species where the maximum net productivity level (MNPL) is generally believed to be large. This might be the reason for this function not having been suggested for NZSL population. However, it serves as a heuristic example that exhibits important features that are common to more realistic models (Wade 1998).

- First, the existence and stability of steady states and periodic solutions changes as a critical parameter is varied.

- Second, the number of parameters affecting the qualitative features of a model is smaller than most other models.
- Third, the function demonstrates an interesting biological phenomenon in density dependency: it is possible for a population to exhibit cyclical behaviour even though the environment is completely unchanging, and the population may never reach a single equilibrium level if it has a sufficiently high rate of increase (Allman and Rhodes 2004, p25).

The above properties are summarised in Table 7.1 in page 203 in comparison with the properties of other functions examined later in the chapter.

7.3 θ–logistic function

Gilpin and Ayala (1973) presented a population model based on θ-logistic function introducing an additional parameter to the logistic function to incorporate non-linearity in density dependence, enabling it to describe the asymmetry in population growth[2]. The θ–logistic function can be considered as a reparameterised (and simplified) version of the Pella and Tomlinson (1969) model that is employed to investigate surplus production versus stock size relationships in fisheries (Pitcher and Hart 1982, Hilborn and Walters 1992). Sometimes this function is referred as the generalized logistic function (e.g., Wade 1998, Wade and DeMaster 1999, Runge and Johnson 2002). It and its linear transformations are commonly used in the area of animal population modelling, especially in fisheries modelling (e.g., Chivers 1999, Runge and Johnson 2002, Saether et al. 2002).

Generally this function is given in the literature with quite different parameterizations, depending on whether the population model is continuous-time or discrete-time, and

[2] The name θ–logistic was given due to the introduction of the θ parameter to the logistic function, but for consistency with the notation used in the rest of this thesis I replace θ with *b*.

In fact Verhulst's (1838) original DD model contains a parameter for non linear growth (Pomerantz et al. 1980), therefore, the concept of non-linear density dependency had been introduced long before the θ–logistic function. Also, later in this chapter, we meet with several non-linear DD functions that were introduced before the θ–logistic function. Hence Gilpin and Ayala (1973) did not introduce the non-linearity, but rather incorporated non-linearity to logistic function.

demographic or count-based. In this thesis we consider only discrete-time models. Using the notation introduced in the logistic function, this function can be given as

$$f(N) = d\left\{1 - \left(\frac{N}{a}\right)^b\right\},$$

where *f(N)* is the density dependent rate either in count-based or demographic, *d* is the limiting value of function when density approaches zero, *a* is the density when the rate become zero due to DD effects, and *b* is a parameters that determines the curvature of the DD relationship.

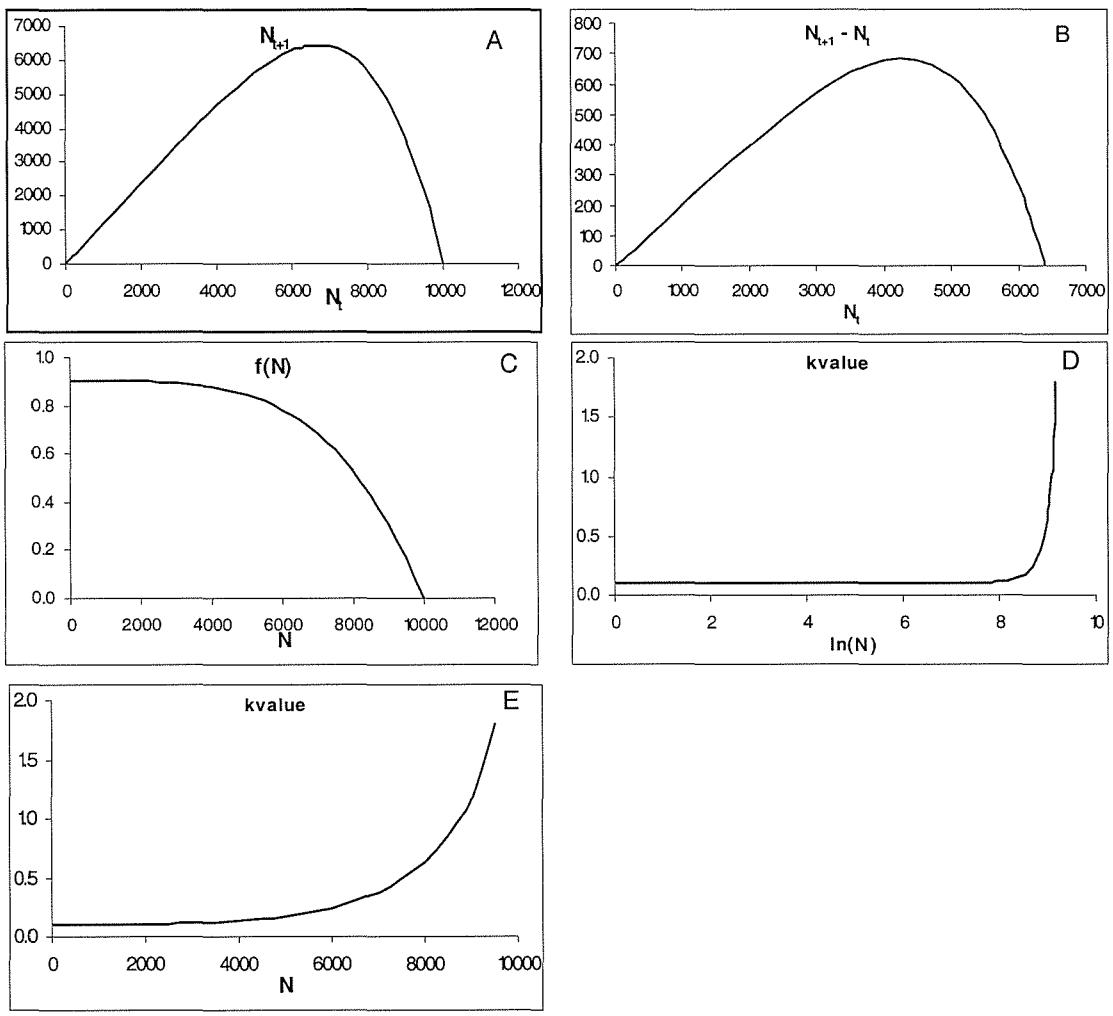

Figure 7.2: An example of density dependence under the θ-logistic function. The panels A and B use the parameters d = 1.2, a = 10000, and b = 4 to represent a growing population in a count-based model. Note that N_{t+1} is zero when $N_t = a$, and the growth is zero at a lower density, the carrying capacity. The last 3 plots use parameters d = 0.9, a = 10000, and b = 4 to represent a demographic rate lower than one, such as survival rate.

When this is applied to count-based models, *f(N)* is the population multiplication rate usually denoted by λ, and *a* is the density at which the population becomes extinct. The function has a value of unity at the carrying capacity (K), therefore

$$K = a\left(\frac{d-1}{d}\right)^{1/b}.$$

The Figure 7.2 (that corresponds to the plots described in section 6.1.5) shows the shape of the function, and some important features of the population dynamics under this function.

7.3.1 The shape of the function and the form of competition

This is a monotonically decreasing function with increasing N, and the parameter *b* governs its shape (Figure 7.2, panel C). The second derivative of the function is positive if $b<1$ (i.e., the function is concave up), negative if $b>1$ (i.e., the function is concave down), and zero if $b=1$ (i.e., function is linear). It does not change from negative to positive with increasing N, hence a sigmoidal shape is not possible. The slope of the function, which is indicative of the strength of DD, is maximum at $N=0$ if $b<1$, and at $N=a$ if $b>1$. Therefore there is no specific population level between zero and *a* that maximises the strength of DD as there is in sigmoidally shaped functions. As *b* approaches infinity, the function remains fairly density independent until N nears to *a* at which point it reduces sharply.

At large *N* the first derivative of N_{t+1} with respect to N_t is large and negative. The first derivative of the kvalue with respect to *ln(N)* is large positive at large *N*. The sign of the second derivative of the kvalue with respect to *N* is positive at any level of *N*. These three features all imply that this function can represent only scramble competition.

7.3.2 Population stability

The stability of the population at $N = K$ can be determined by using the first derivative of N_{t+1} with respect to N_t evaluated at K, or the first derivative of kvalue

with respect to ln(N) evaluated at K, which are $\{1-b(d-1)\}$ and $b(d-1)$ respectively for count-based models. Therefore, using the criterion in Table 6.2, the population is,

- stable at K with oscillatory damping if $1 < b(d-1) < 2$,
- stable at K with monotonic damping if $0 < b(d-1) < 1$,
- Information is insufficient to determine if $b(d-1) = 0$ or $b(d-1) = 2$,
- and unstable otherwise.

7.3.3 Asymmetric growth

It can be seen by taking the derivative of the expression for the population growth ($N_{t+1} - N_t$) with respect to N_t that the growth becomes maximum at the density of

$$N^* = a\left(\frac{d-1}{d(1+b)}\right)^{1/b} \text{ or equivalently at } N^* = K\left(\frac{1}{1+b}\right)^{1/b}.$$

Therefore this function allows the maximum growth to occur at different fractional densities of K (Getz 1996). Note that:

- N^* =K/2 when $b = 1$. This is the case of the logistic function that growth becomes symmetric.
- $K/2 < N^* < K$ when $b > 1$,
- $K/e < N^* < \frac{K}{2}$ when $0 < b < 1$, where e is the base of natural log.

Therefore the parameter b has important implications in managing harvestable populations.

N^* asymptotically approaches K when b approaches infinity, while it approaches K/e when b approaches zero[3]. Therefore, under the θ-logistic function

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[3] Say $Y = \left(\frac{1}{1+b}\right)^{1/b} = (1+b)^{-\frac{1}{b}}$. Then N^* = YK. $\log(Y) = -\frac{1}{b}\log(1+b)$. Using L'Hôpital's rule, $\lim_{b\to 0}\log(Y) = \lim_{b\to 0}\frac{-\{1/(1+b)\}}{1} = -1$. Therefore $\lim_{b\to 0} Y = \frac{1}{e}$, and so $\lim_{b\to 0} YK = \lim_{b\to 0} N_{\max} = \frac{K}{e}$.
Similarly, $\lim_{b\to\infty}\log(Y) = \lim_{b\to\infty}\frac{-\{1/(1+b)\}}{1} = 0$. Therefore $\lim_{b\to\infty} Y = 1$, and $\lim_{b\to\infty} YK = \lim_{b\to\infty} N^* = K$.
Hence $K/e < N^* < K$.

$$K/e < N^* < K.$$

Since $N^* > K/e$ for all combinations of positive parameters, this implies that this function cannot describe the density dependence of a population where the growth rate is large enough to have maximum growth at a level smaller than K/e (Gilpin et al. 1976, Pomerantz et al. 1980, Thomas et al. 1980). Even if it does not make biological sense of having maximum growth at a density of near zero, populations with $N^* < K/e$ are quite plausible (Gilpin et al. 1976). Also, because $K/e < N^* < K$, this function can be a potential candidate for the NZSL population. In fact it has been used in at least two population models proposed for the NZSL population (e.g., Hilborn and Wade 1999, Breen et al. 2003).

Fletcher (1978) observed that for the θ–logistic model, N^* approaches zero as *b* approaches -1. Therefore, with the exception of a singularity at *b* = 0, N^* can range from zero to K as *b* varies from -1 to infinity. However, any *b* below zero implies a positive density dependence. Therefore under this model N^* cannot be below K/e with negative density dependence. One of the attractive features of this model is that by knowing *b*, N^* can be localised to one of three regions as below;

Value of b	Range of N^*
$-1 < b < 0$	$0 < N^* < \frac{K}{e}$
$0 < b < 1$	$\frac{K}{e} < N^* < \frac{K}{2}$
$1 < b < \infty$	$\frac{K}{2} < N^* < \infty$

Depending on the values of the parameters, this function can represent various forms of dynamical stability. It can represent the asymmetric growth within some limitations (i.e., N^*>K/2), but it does not possess the ability to represent either a sigmoid shape or the contest form of competition. Also, difficulties are often experienced when fitting this model to data (i.e., parameter estimation is difficult) especially when *b* is small (Pomerantz et al. 1980). Nevertheless it has become one of the most commonly used functions among fisheries biologists.

7.4 Ricker function

The Ricker (1954) function was primarily a stock recruitment model in fishery populations (Pitcher and Hart 1982), and later became a density dependent function both in count-based and demographic models. In demographic models it is more commonly used to represent the density dependence in reproduction, although it has occasionally been used for other vital rates (e.g., Neubert and Caswell 2000, for transition rates). The logistic function can be considered as a Taylor series approximation of this function (Eberhardt 1977).

The Ricker function can be seen in the literature in a variety of parameterizations depending on the context of its use (Hannesson 1993, Gurney and Nisbet 1998). In the area of fisheries, where it is sometimes used as a stock-recruitment model or as a surplus yield model, parameterization of the model is so different that the model appears to be a completely a different one (e.g., Hannesson 1993, p56). In count-based population models it is very common to parameterize it in terms of population growth rate and carrying capacity as $f(N) = e\,\mathrm{xp}\left\{r_0\left(1-\frac{N_t}{K}\right)\right\}$, (Pitcher and Hart 1982, p184). However I use the parameterization of

$$f(N) = d\exp(-aN)$$

because it is consistent with the notation used in rest of the chapter, and because it can be used equally well in both count-based and demographic models. This parameterization has been used in count-based models (e.g., Hassell et al. 1976, May and Oster 1976, Caswell 1997), and for reproductive rate in demographic models (e.g., Ricker 1954, Pitcher and Hart 1982, p184, Caswell 1997), and for survival rate (e.g., May et al. 1974, Bellows 1981).

7.4.1 The shape of the function and the form of competition

The plots in Figure 7.3 (corresponding to the five plots introduced in section 6.1.5), show some of the properties of the function and some features of the resultant dynamics. The *f(N)* function has a monotonic, decreasing, concave-up shape, with the

highest slope of the curve (greatest strength of density dependence) occurring at zero density (plot C in Figure 7.3). The second derivative of *f(N)* with respect to N is positive for all N, implying this function cannot represent a sigmoid shape.

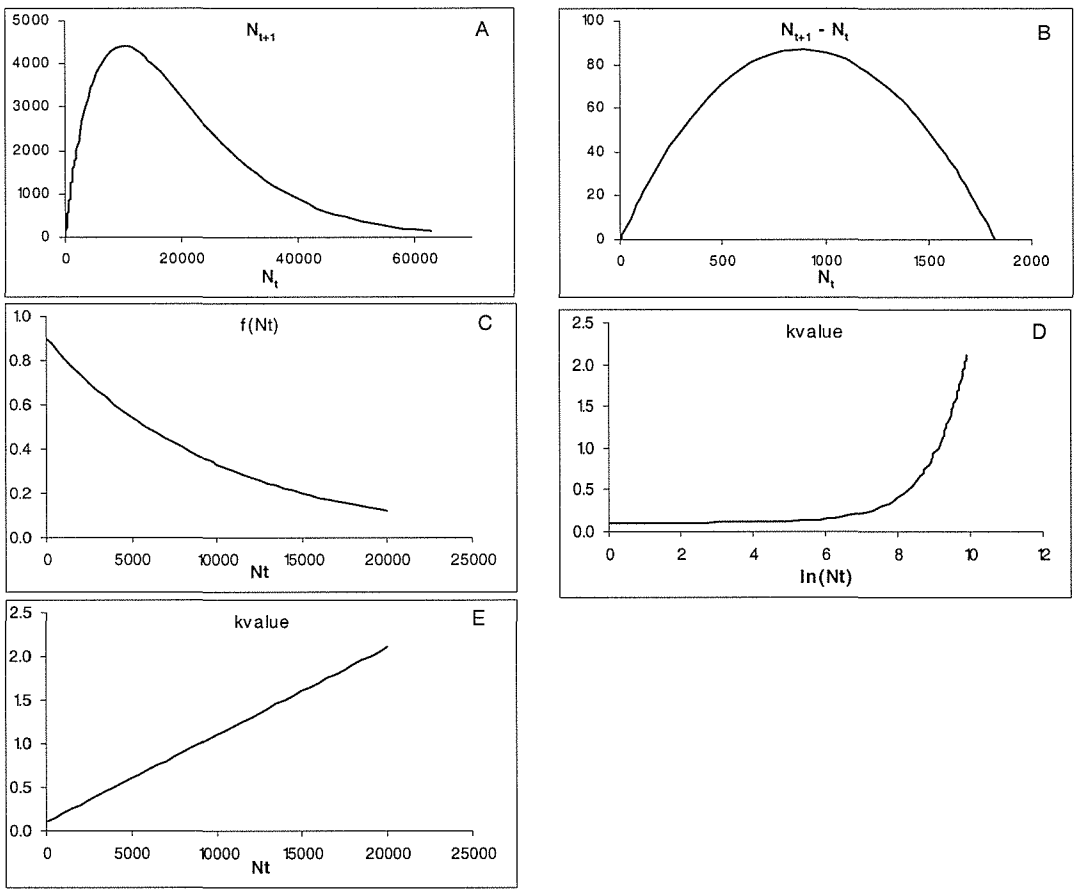

Figure 7.3: An example of density dependence under the Ricker function. Plots A and B represent a growing an initially growing population in a count-based model with parameters d = 1.2 and a = 0.0001. The other three plots use parameters d = 0.9 and a = 0.0001 representing a demographic rate lower than one.

$\frac{dN_{t+1}}{dN_t}$ is positive if $N_t < \frac{1}{a}$, and negative otherwise. Therefore the relationship of N_{t+1} vs N_t (in plot A) takes a humped shape. Although N_{t+1} does not become zero at a large finite density (as happens under the logistic and q-logistic functions) it approaches zero when N_t becomes very large. Both these features are usually associated with scramble competition. The first derivative of this relationship is negative and approaches zero at large N, therefore it does not help in deciding the nature of the competition. The second derivative of the kvalue with respect to N is zero, (in

agreement with the linear relation in plot E), implying that this function can be considered as one that represents both forms of competitions (contest and scramble) equally. The first derivative of the kvalue with respect to ln(N) is *aN,* which means that the slope increases linearly with N. This can be interpreted in more than one way.

The slope *aN* can increase without bound with increasing N, hence according to the section 6.2.2.2 we could conclude this function represents scramble competition. However, in the case of count-based models, the parameter *a* can be specified in terms of the carrying capacity (K) as given by the relationship, $aN = \frac{N\ln(d)}{K}$. From this we can reasonably assume that the slope will not be very large when N remains within practical limits (even if N becomes several times larger than K), and hence might not reach scramble level, particularly for species where the parameter *d* is usually small. In addition, according to the cut-off suggested in section 6.2.2.4 we could decide that this represents both contest and scramble competitions equally. Therefore we can conclude that the function represents both forms of competitions equally (unless *d* is large or the population level increases to an unrealistically high level)[4].

7.4.2 The stability

In count-based models, the population is stationary at the density of $K = \frac{\ln(d)}{a}$. $\left.\frac{dN_{t+1}}{dN_t}\right|_K = 1 - \ln(d)$, and $\left.\frac{dk}{d\ln(N)}\right|_K = \ln(d)$. Therefore, as per the criterion in table 6.2, the population is stable at K if and only if $0 < \ln(d) < 2$. This condition[5] holds for all values of *d* that are between 1 and 7.4. As observed under the logistic function, the parameter *d* governs the dynamics of the population (May 1974). Since parameter *a* has no effect on the stability at K (Neubert and Caswell 2000), by specifying *d* in terms of demographic rates, we can separate combinations of demographic rates that

[4] This conclusion can be subjected to criticism. The Ricker function is generally considered as a scramble function (e.g., Gurney and Nisbet 1998, Caswell 2001, Wikan 2004). The different conclusion here is due to the specific 'cut-off' used.

[5] When *d* is only slightly lager than one (that is the likely situation for most large mammals), using the approximation of $\ln(d) = (d-1)$, the condition $0 < \ln(d) < 2$ translates to a stable population at K if $1 < d < 3$, which is the same stability condition obtained under the logistic function.

correspond to stable populations from those that correspond to unstable populations (Gurney and Nisbet 1998, p127).

7.4.3 Asymmetric growth

It can be seen by taking the first derivative of the expression for the population growth ($N_{t+1} - N_t$) with respect to N_t that the growth becomes maximum at the population size that satisfies the condition $d(1-aN) = e^{aN}$. This expression cannot be solved analytically for the population level that corresponds to maximum growth. However, as shown in appendix 8, under the assumption of $d \approx 1$ (assumed to be a realistic assumption for most long-lived species such as NZSL) this population level (N^*) can be estimated to be confined to the narrow interval,

$$\frac{K}{e+\ln(d)} < N^* < \frac{K}{2+\ln(d)}.$$

By allowing d to vary between one and infinity, this interval can have a maximum possible range of zero to K/2. But, because this interval was obtained under approximations (i.e., keeping a gap in both sides of N^*), the above statement does not mean to say that N^* can be anywhere in this interval. It is safer to say that this function does not allow maximum growth beyond this range.

For long-living populations, where parameter d is usually not far from one, the lower limit of this range will not fall far below K/e, narrowing above interval to $\frac{K}{e} < N^* < \frac{K}{2}$ which says the growth function can deviate from symmetry only slightly. Since the function cannot exhibit the maximum growth at population levels above K/2, this function does not seem to be a suitable candidate in modelling long-lived slow-growing animal populations such as the NZSL.

The Ricker function is common in representing density dependence in reproduction. One of the attractiveness of this function is its longer right tail (in plot A, Figure 7.3) that allows some reproduction even at very large densities. The function has some drawbacks such as being non sigmoid, and insufficiently asymmetric. Nevertheless it

can describe various dynamical stability properties. It is quite simple, and having only two parameters parameter estimation becomes somewhat easier.

7.5 θ–Ricker function

Seven years after introducing the θ–logistic model, Thomas et al. (1980) introduced the θ–Ricker model by adding an additional parameter to the Ricker function, with the intention of eliminating some of the drawbacks in the Ricker model – In particular, its inability to adequately represent asymmetric growth[6]. This function is sometimes known as the generalised Ricker function (e.g., Getz 1996, Yearsley et al. 2003), and is commonly used to represent DD in both count-based and demographic models. Bellows (1981) and Getz (1996) discussed some of the properties of this model.

Similar to the functions described earlier, this function appears in the literature in quite different forms. In count-based models the function is commonly parameterized in terms of growth rate and carrying capacity. Among the widely varying forms available[7] $f(N) = \exp\left[r_0\left\{1-\left(N/K\right)^{\theta}\right\}\right]$ is quite common. However, I use the parameterization of

$$f(N) = d\exp(-aN^b)$$

where *f(N)* can be growth rate in count-based models or any vital rate in demographic models.

7.5.1 The shape of the function

The Figure 7.4 shows the shape of the function and some important features of the dynamics under the θ-Ricker function. The first derivative of *f(N)* with respect to N is

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[6] Both Thomas et al. (1980) and Bellows (1981) claim this function is new to their papers.

[7] For example, Getz (1996) reparameterised this to $f(N) = \exp\left\{r_0\left(1-N/K\right)^{\theta}\right\}$, where θ > 1.

zero when the density is zero or infinity, and negative at all other levels of density. It becomes maximal (in magnitude) at an inflection point, when

$$N = \left(\frac{b-1}{ab}\right)^{1/b},$$

where the density dependence becomes strongest. Such a point may not exist if $b < 1$, and so the shape of the function is dependent on the value of b. The second derivative of the θ–Ricker function with respect to N changes from negative to positive at this inflection point as N increases - hence a sigmoid shape is possible only if $b > 1$.

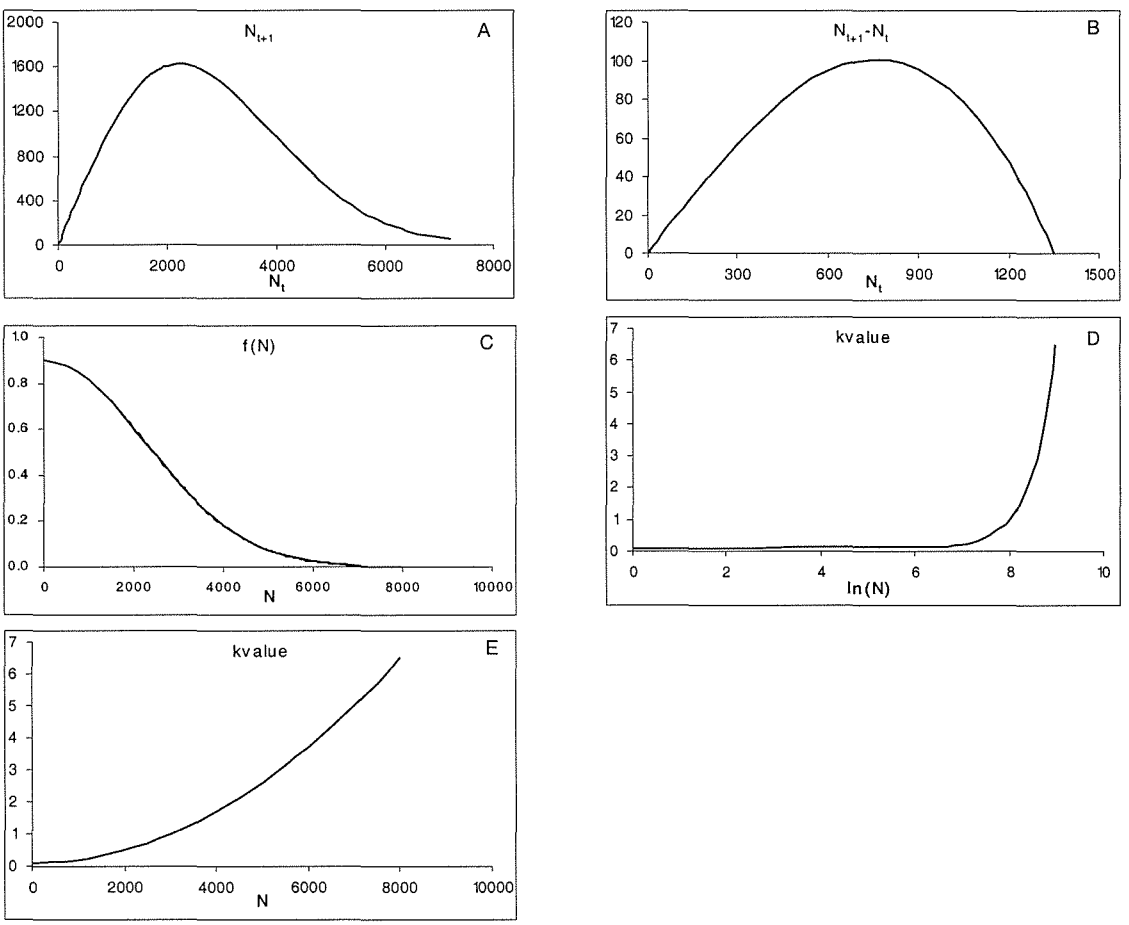

Figure 7.4: An example of density dependence under the θ-Ricker function. Plots A and B use the parameters $d = 1.2$, $a = 0.0000001$, and $b = 2$ to represent a growing population in a count-based model. N_{t+1} approaches zero when N_t is large. The growth is zero at the carrying capacity (at N = 1350 for these parameters). In the last three plots, the parameter d is changed to 0.9 to represent a demographic rate lower than one.

7.5.2 The nature of competition

The first derivative of N_{t+1} with respect to N_t is $\left(1-abN_t^b\right)d\exp\left(-aN_t^b\right)$, which is small and negative at large N, so it does not provides a clear indication about the nature of the competition. The second derivative of kvalue with respect to N is $ab(b-1)N^{b-2}$, which is negative if $b < 1$ implying contest competition, and is positive if $b > 1$ implying scramble competition (as per the criterion in Table 6.2). The derivative of kvalue with respect to *ln(N)* is $\frac{dk}{d\ln(N)} = abN^b = b\ln(d)\left(\frac{N}{K}\right)^b$, which can increase without limit with increasing N providing evidence of scramble competition (section 6.2.2.2). However, under most realistic conditions N is not much greater than K, hence the right hand side of this expression is not much larger than $b\ln(d)$. Therefore, unless parameters b and/or d are large enough it is unlikely for this function to represent scramble competition. For long-lived slow-growing species the parameter d is usually near unity. Therefore, for such species this function is most likely to represent contest competition (unless the parameter b is very large). That is the likely situation for the NZSL.

Further, as above expression shows, the increase in derivative with N is slower than linear if $b < 1$, implying contest competition is possible (section 6.2.2.4). If $b = 1$ the increase of the slope is linear implying that both types of competitions are equally represented. If b > 1 the increase is faster than linear and so the competition is scramble. The *f(N)* function also shows that sufficiently high values of b can cause the population to decline sharply, a phenomenon usually associated with scramble competition. Therefore the nature of competition is decided by the parameter b. Increasing values of parameter b imply movement along the continuum of contest-scramble competition towards more severe scramble.

7.5.3 The stability

In count-based models the population is stationary at the density $K = \left(\frac{\ln(d)}{a}\right)^{1/b}$.

$$\left.\frac{dk}{d\ln(N)}\right|_K = b\ln(d) \approx b(d-1), \qquad \text{and} \qquad \left.\frac{dN_{t+1}}{dN_t}\right|_K = \{1 - b\ln(d)\} \approx \{1 - b(d-1)\}.$$

Thus the condition for neighbourhood stability at K is $0 < b(d-1) < 2$. Therefore:

- if $0 < b(d-1) < 1$ then the population is stable at K with monotonic damping.
- if $1 < b(d-1) < 2$ then the population is stable at K with oscillatory damping.
- if $b(d-1) = 0$ or $b(d-1) = 2$ then the information is insufficient to determine the stability.
- the population is unstable at K otherwise.

Consequently, the stability is decided by the interaction of parameters *b* and *d*, independent of the parameter *a*. Because *d* is usually small for long-lived animals, the strength of the DD effects around K is largely governed by the parameter *b*, and so Getz (1996) referred to *b* as the abruptness parameter. The larger the value of *b*, the weaker the effect of DD at population densities below K, the more rapidly DD sets in around K, and the more severe the effects of DD above K. The DD function is less sensitive to changes in N near K when b is small[8]. This characteristic is different to what was seen for the θ–logistic function (Getz 1996).

7.5.4 The asymmetric growth

For the θ–Ricker function, the level of the population that produces the maximal growth (N^*) cannot be obtained in terms of K analytically. Pomerantz et al. (1980) investigated the asymmetry property of this function by fitting it to data from a large number of insect species, and observed that maximum growth occurs well below K. Although the θ–logistic function yields symmetric growth when *b* = 1, there is no such value of *b* where the θ–Ricker model yields a symmetric growth. Using numerical methods Thomas et al. (1980) showed that as *b* varies from zero to infinity,

[8] When *b* is small N_{t+1} responds to changes of N_t less abruptly (i.e., more slowly), hence it take several time steps to produce the same response in N_{t+1} that occurs in a single time step when b is large (when response is abrupt). Therefore the effect of a small *b* is similar to the effect of time delay (Getz 1996).

N^* ranges from zero to K. This flexibility of permitting asymmetric growth dynamics is important due to the observation that it is biologically unreasonable to expect all species to experience maximal growth at the same fractional density of K (Gilpin et al. 1976).

7.6 Haldane function

Haldane (1953) gave one of the simplest functions for density dependence. It was in frequent use in the early days (Varley and Gradwell 1960, Varley et al. 1973, Fowler 1981). Even though its appearance is rare in recent literature, it is useful as a reference point in studying more realistic functions as it exhibits some important features of a DD function. The function is simpler than others having only two parameters, but it still has the ability to exhibit the existence and stability of steady states, and stability that varies as a critical parameter is varied. Under this function, it is possible for a population to exhibit cycles even if the environment is completely unchanging (May et al. 1974).

The form of the function is quite different between continuous-time and discrete-time versions of models. To be consistent with the rest of the thesis I consider only the discrete-time version, which is given as

$$f(N) = dN^{-b}.$$

Negative values of b imply positive density dependence, b = 0 implies density independence, and positive b means negative density dependency. The magnitude of b represents the strength of the density dependence. The parameter d represents the value that the population multiplication rate (in count-based models) or the vital rate (in demographic models) approaches when the density goes to zero. In the count based context this function is given in the literature in various forms, for example as $kvalue = b\ln(N) - \ln(d)$ in Varley and Gradwell (1968), and as $\ln(N_{t+1}) = \ln(d) + (1-b)\ln(N_t)$ in Morris (1959). These versions have been used to detect the presence of DD in count-based data by estimating the parameter b. Varley and Gradwell (1960) and Bellows (1981) used this function in demographic models.

The parameter b is generally small (much smaller than one). Larger values of b are characterized by sharply declining $f(N)$ functions, which results in unrealistically small function values at realistic levels of N. In count-based models $f(N) = 1$ at the population size of $K = d^{1/b}$, and so the population is stationary at this level of N. For populations where d is not much larger than one, the parameter b should be small for K to be realistically large. Therefore, I consider only the situation where b is small[9].

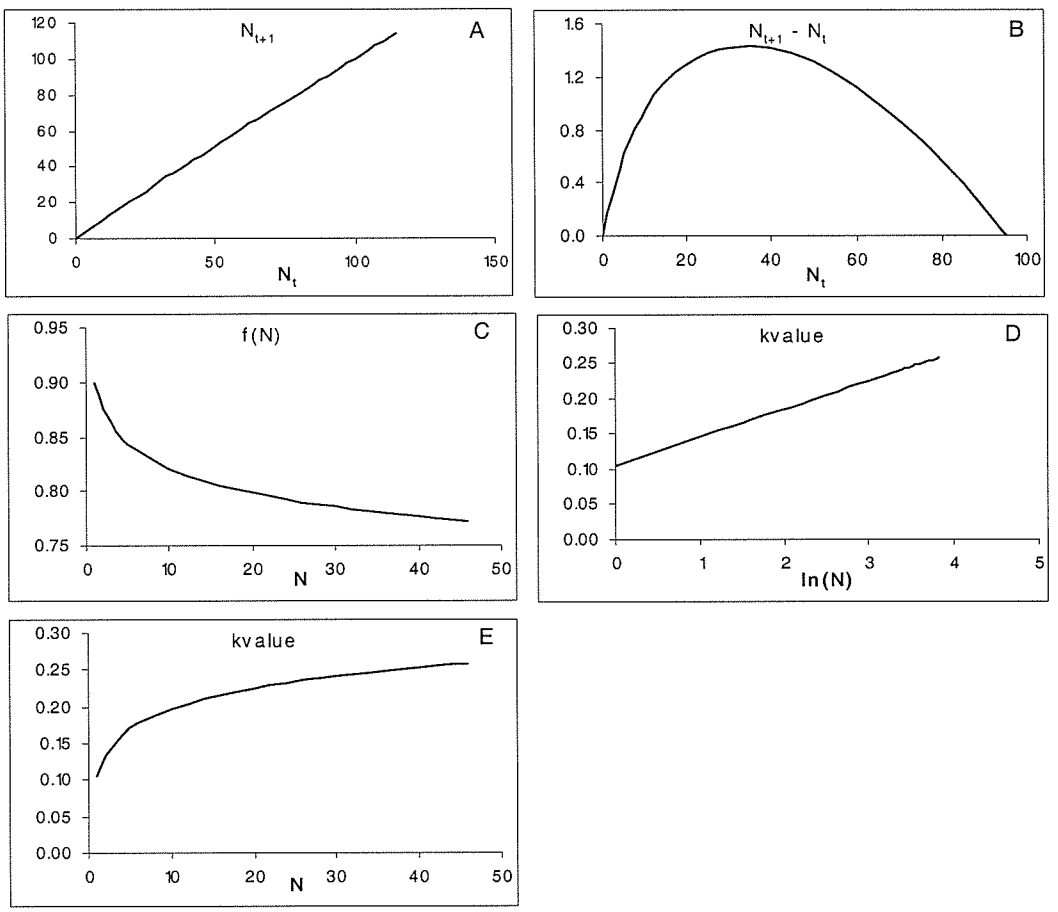

Figure 7.5: An example of density dependence under the Haldane function. The panels A and B use the parameters $d = 1.2$ and $b = 0.04$ to represent the dynamics of a growing population in count-based models. (Due to the small value used for b, N_{t+1} appears nearly proportional to N_t). For these parameters, the growth becomes zero at the carrying capacity of N = 95. In the last three plots, the parameter d was changed to 0.9 to represent a demographic rate over the unit range.

[9] In exception to this, May et al. (1974) discuss situations where b is larger, such as 2.5.

7.6.1 The shape of the function

Some features of this function are shown in the Figure 7.5. The first derivative of *f(N)* is negative and the second derivative is positive for the entire range of N, so the function is shaped concave-up, monotonically decreasing, and asymptotically approaching zero at large N. the slope is maximum (i.e., the density dependence is strongest) at the lowest density, and near zero (i.e., nearly density independent) at larger densities. Therefore, a sigmoid shape is not possible. Since the parameter *b* is generally small, N_{t+1} is nearly linearly related to N_t, as van be seen from panel A (i.e., the DD relationship is generally weak).

7.6.2 Nature of competition and stability

The first derivative of N_{t+1} with respect to N_t is $\frac{d(1-b)}{N^b}$, which is positive and approaches zero at large N when $b < 1$ (in fact *b* is much smaller, see later). Also, $\frac{d^2k}{dN^2} = \frac{-b}{N^2}$, which is always negative irrespective of the value of *b* or level of N. Both these results suggest contest competition. The slope of the relationship between the kvalue and ln(N) is *b*. Therefore the slope of this plot is not increasing with N, also implying contest competition (section 6.2.2.4). According to section 6.2.2.2, one could interpret this result as the function being scramble if b > 1, and contest if b < 1. However, this function cannot represent scramble competition because *b* is usually much smaller than one.

The derivative of the kvalue with respect to *ln(N)* is *b* while the derivative of N_{t+1} with respect to N_t is *(1-b)* at the population level of *K*. That is; $\left.\frac{dk}{d\ln(N)}\right|_K = b$, and $\left.\frac{dN_{t+1}}{dN_t}\right|_K = 1-b$. Therefore, in theory, the magnitude of parameter b governs the neighbourhood stability at K under this model. The population is stable at K if $0 < b < 2$. Since *b* is usually very small, the population is always stable at K.

7.6.3 Asymmetric growth

This model predicts a maximum growth at the population level of $N^* = K(1-b)^{1/b}$, which does not exist if $b > 1$. When b varies from zero to one, N^* varies from K/e to zero (as shown below[10]). Therefore, depending on the value of b, the maximum growth occurs at a population level between zero and K/e, (Figure 7.5, panel B). Since N^* cannot exceed K/e, this model may not be a suitable candidate for large mammal species (e.g., NZSL) because for most of those species the maximum growth generally occurs near K. As seen in section 7.3, maximum growth occurs at a population level of between K/e and K under the θ-logistic function, the range which is exactly the complement of the range under this model.

7.7 Beverton and Holt function (B&H function)

Similar to the Ricker function, the Beverton and Holt (1957) function is used very commonly in fishery models to describe the density dependent relationship between stock (i.e., the parental population size) and recruitment (Gulland 1988), and also to model the harvestable growth (Hannesson 1993). Although both of these functions were originally derived from different theoretical analyses, it is a common practice to choose the one which fits the trend of the data best (Pitcher and Hart 1982, p182)[11]. Both of these functions are widely used to represent the DD in demographic rates (e.g., Bellows 1981), in count-based models (e.g., Getz 1996), and in biomass dynamics models (e.g., Pitcher and Hart 1982, p221).

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[10] Say, $Y = \ln\left((1-b)^{1/b}\right) = \dfrac{\ln(1-b)}{b}$. Therefore $N^* = Ke^Y$.

$\lim_{b\to 0} Y = \lim_{b\to 0} \dfrac{\ln(1-b)}{b} = \lim_{b\to 0}\left(\dfrac{-1}{1-b}\right)$ = -1 (Using L'Hôpital's rule).

Therefore, $\lim_{b\to 0} e^Y = e^{-1}$, which leads to $\lim_{b\to 0} K(1-b)^{1/b} = \lim_{b\to 0} Ke^Y = K/e$.

Furthermore, $\lim_{b\to 1} K(1-b)^{1/b} = 0$, so $0 < N^* <$ K/e.

[11] As stated in sections 6.1.4 and 6.2, one of the motivations to this chapter is the inappropriateness and impracticality of selecting a DD function based on a statistical fit.

The B&H function can be seen in various forms in the literature (e.g., May et al. 1974, Pitcher and Hart 1982, Getz 1996, Alsop 1998), and various reparameterizations of the same form can also be seen. Using the notation of this thesis it can be given as

$$f(N) = d(1 + aN)^{-1}$$

where parameter d is the value that vital rate approaches when N approaches zero, and parameter a determines the strength of density dependence.

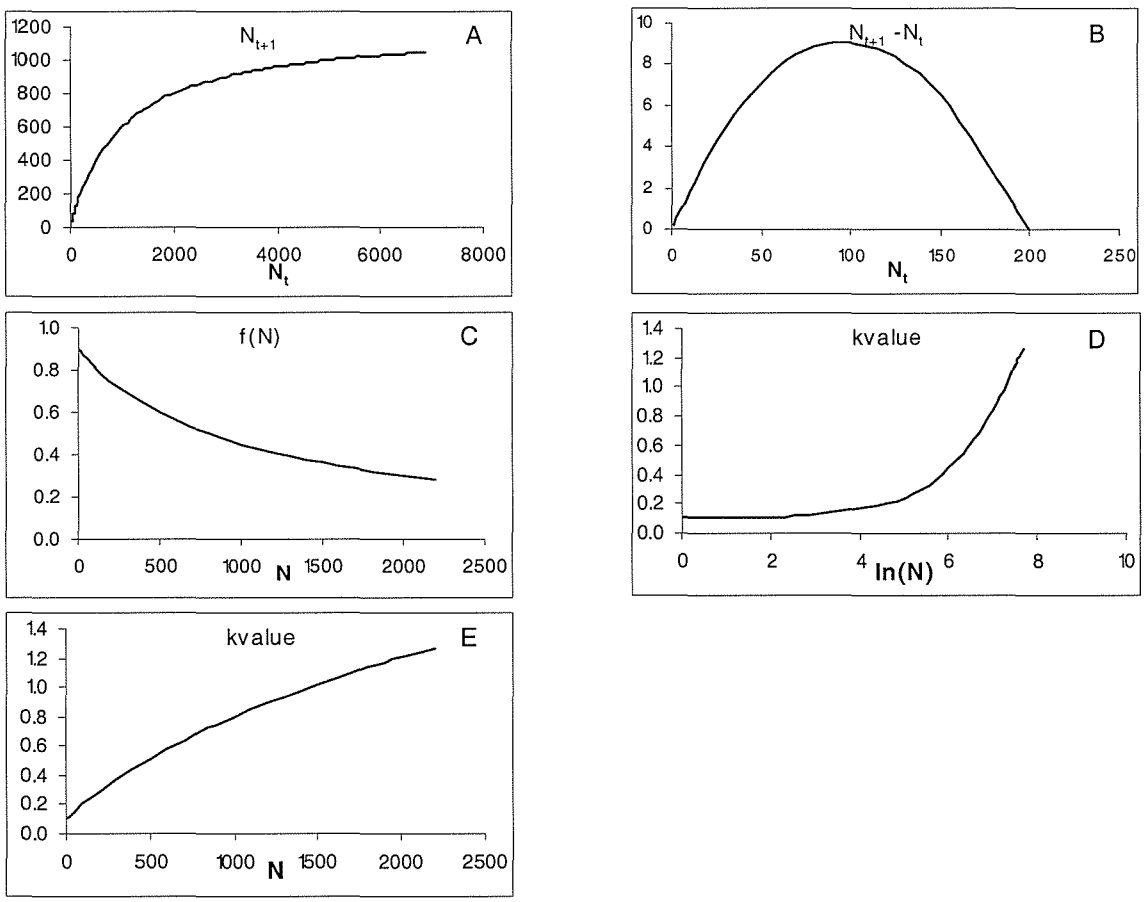

Figure 7.6: An example of density dependence under the B&H function. The panels A and B, with parameters of d = 1.2 and a = 0.001, show the features of the dynamics in count-based models. The other three panels use parameters d = 0.9 and a = 0.001 representing a demographic vital rate.

The parameter a is generally small (much smaller than one). Larger values of a are characterized by sharply declining *f(N)* functions, which results in unrealistically small function values at realistic levels of N. In count-based models *f(N)* = 1 at the population size of $K = \frac{d-1}{a}$, and so the population is stationary at this level of K. For

populations where d is not much larger than one, the parameter a should be small if K is to be realistically large (that could be the likely situation for most marine mammals, including the NZSL). Therefore, I consider the situation where a is small. (In exception to this, May et al. (1974) discuss situations where a is larger, such as 2.5).

In count-based models, the function $f(N)$ becomes one at the population level of $K = \frac{d-1}{a}$, the carrying capacity. Therefore, the parameter a should be very small relative to d in order to have a realistically large value for K (assuming K is measured by number of individuals).

7.7.1 The shape of the function and the form of competition

Figure 7.6 shows some important properties of the function. The function is monotonically decreasing, asymptotically approaching zero at large N. It is concave-up shaped, with the magnitude of the slope decreasing with increasing density, implying that density dependence is strongest at the lowest density, and weakest at the largest density. The second derivative of $f(N)$ with respect to N is positive for all N, so a sigmoid shape is impossible to represent.

The derivative of N_{t+1} with respect to N_t is positive and approaches zero at large N, that is; $\lim_{N\to\infty} \frac{dN_{t+1}}{dN_t} = \lim_{N\to\infty} \frac{d}{(1+aN)^2} = 0$. Also, $\frac{dk}{d\ln(N)} = \frac{aN}{1+aN}$, which approaches one at large N. It is less than one at all realistic values of N. Further, $\frac{d^2k}{dN^2} = \frac{-a^2}{(1+aN)^2}$, which is negative for all values of N. All these results suggest the function can only represent the contest competition.

7.7.2 Stability

The derivative of the kvalue with respect to *ln(N)* is $\left(\frac{d-1}{d}\right)$ while the derivative of N_{t+1} with respect to N_t is $\left(\frac{1}{d}\right)$ at the population level of *K*. That is; $\left.\frac{dk}{d\ln(N)}\right|_K = \frac{d-1}{d}$, and $\left.\frac{dN_{t+1}}{dN_t}\right|_K = \frac{1}{d}$. As per the criterion in Table 6.1, both these results lead to the conclusion that the population is stable at K if $d > 1$, as agreed by Gurney and Nisbet (1998, p127). This result is unusual given that no population is stable when *d* is very large under any of the other functions examined. This result is a special case of the stability conditions obtained for the more general MS&S and Hassell functions (see sections 7.8 and 7.9). The stability is not influenced by the parameter *a*. Of course, for any viable population, $d > 1$ in count-based models, because no population can avoid extinction if d < 1. Therefore, this function represents stable dynamics for all viable populations.

7.7.3 Asymmetric growth

In count-based models, the growth becomes maximal at the density of $N^* = \left(\frac{d^{1/2}-1}{a}\right)$. Since $K = \frac{d-1}{a}$, N^* can be give in terms of K as $N^* = \frac{K}{d^{1/2}+1}$. Therefore, neither positive K nor positive N^* exist if $d < 1$, which is intuitive to understand.

When *d* approaches unity *N** approaches *K/2* while when *d* approaches infinity *N** approaches zero, that is; $\lim_{d\to 1} N^* = \frac{K}{2}$, and $\lim_{d\to\infty} N^* = 0$. Therefore, under this function, maximum population growth can occur at population levels between zero and K/2 only. For this reason, this model cannot represent the dynamics of populations where the maximum growth occurs near K. This is consistent with the fact that this function has not been used in any of the models proposed for the NZSL population. This could also be a reason why this function is so common in fisheries models where it is usual for the maximum productivity to be generated at densities of less than half their

carrying capacity. This is in contrast to the observation that under the θ–logistic function, which is also commonly used in fisheries models, maximum growth occurs only at population levels that are above K/e (section 7.3).

7.8 Maynard Smith and Slatkin function (MS&S function)

This function was first introduced by Maynard Smith and Slatkin (1973) in modelling density dependent reproduction. They presented this model for a non-overlapping population with a life cycle where individuals breed at age one and then die so that newborns become the whole population in next year i.e., $N_{t+1} = RN_t$, a pattern of life cycle that is approximately correct for some species (Maynard Smith 1974). In this context it can be considered as a DD function in a count-based model. The density dependency in reproductive rate (R) was given by the function

$$R = R_o \left\{ 1 + (R_0 - 1)\left(\frac{N}{K}\right)^b \right\}^{-1}$$

where R_0 is the rate of reproduction at the smallest density, and K is the level of population where R becomes one. This function can be seen in subsequent literature with quite different parameterisations (e.g., May and Oster 1976, Getz 1996, Yearsley et al. 2003). For this thesis I have chosen the parameterisation of

$$f(N) = d\left\{1 + (aN)^b\right\}^{-1}.$$

Reeve et al. (1998), May et al. (1974), Bellows (1981), Bellows (1982), and Alsop (1998) use the parameterization in this form[12]. This form is consistent with the notation in the rest of this chapter, and it can be used for count-based models as well as for demographic rates. The Beverton and Holt function is a special case of this function, hence this function is sometimes referred as the generalised Beverton and Holt function (Getz 1996).

[12] Getz (1996) used the same parameterization as used in this thesis but with an added condition of b > 1. Possible reasoning for this condition could be that it is a requirement for function to be sigmoid shaped, and to represent both types of competitions (see later).

Yearsley et al. (2003) reparameterized this function into a form that can be used in logistic regression to estimate its parameters, where the special case of d =1 is particularly useful for vital rates over the unit interval, such as survival rate.

Bellows (1981) examined the descriptive abilities of seven different density dependent functions, and found that the MS&S function is the most flexible, and has the ability to describe a wider range of data sets than the other functions examined. (Most data sets used are from insect populations where the parameter d can be assumed to be large.) Further, Getz (1996) suggested that this function can be modified to $f(N_t) = d\left\{1 + (aN_{t-1})^b\right\}^{-1}$ to include a time delay in DD responses. The biological basis of changing N_t to N_{t-1} in the denominator is that in some populations, for example, resource limitations imposed on juveniles result in less reproductive adults a few years later (Alsop 1998).

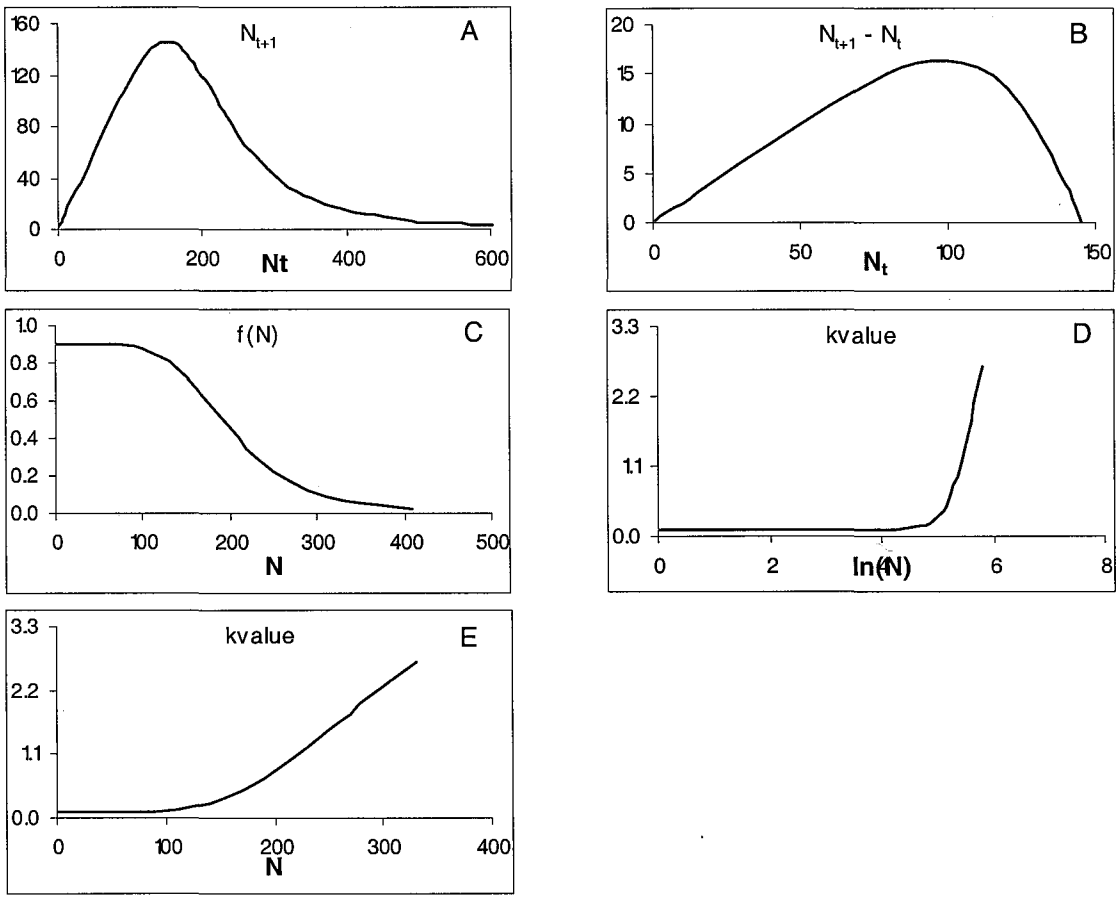

Figure 7.7: An example of the density dependence given by the MS&S function. The panels A and B use the parameters $d = 1.2$, $a = 0.005$, and $b = 5$ to represent the dynamics of a growing population in count-based models. The large value of b used in this example causes the N_{t+1} vs N_t curve to be hump-shaped (i.e., Ricker typed shaped). If b was smaller, the curve would take an asymptotically increasing shape (i.e., Beverton and Holt type shape). The growth can be quite asymmetric (panel B). In the last three plots, the parameter d is changed to 0.9 to represent a demographic vital rate over the unit interval. The shape of the curve is sigmoid in panel C, but it can be a decaying curve if parameter b is smaller.

The parameter a needs to be small for $f(N)$ to be reasonably large at large levels of N. Further, in count-based models $f(N)=1$ at carrying capacity, and so the population is stationary at the carrying capacity of $K=\frac{(d-1)^{1/b}}{a}$. If parameter a is large K will be unrealistically small.

7.8.1 The shape of the function

Some features of this function are shown in the Figure 7.7. The shape of the function is influenced by the value of parameter b. If $0<b<1$, the function has a monotonically decreasing concave up shape (non sigmoid). If b > 1, the sign of the second derivative of the function with respect to N changes from negative to positive with increasing N (as shown in appendix 9), implying that function is sigmoid shaped. Therefore this function has the flexibility to represent both sigmoid and non-sigmoid shaped relationships. When it is sigmoidal, there is an inflection point at $N=\frac{1}{a}\left(\frac{b-1}{b+1}\right)^{1/b}$. The density dependence is strongest at this population level. Note that this population level is independent of the parameter d. The inflection point does not exist (i.e., the function is not sigmoid) if $b<1$. This could be a possible reason for Getz (1996) imposing an additional condition of $b>1$ to make sure the function could represent the sigmoid shape.

When b approaches one, this inflection point approaches zero, and hence the sigmoid shape vanishes. This agrees with the shape of B&H function, which is a special case of the MS&S function (with b = 1) that does not have a sigmoid shape. When b approaches infinity the inflection point approaches $1/a$, which is large, hence strongest density dependence doesn't activate until the population level becomes large.

7.8.2 The form of the competition

The slope of the N_{t+1} vs N_t curve at large values of N is useful in deciding the contest/scramble status. The first derivative of N_{t+1} with respect to N_t can be shown to be small and positive at large N_t if $b < 1$. This means that the relationship between these two variables (N_{t+1} and N_t) is asymptotically increasing, and the competition is contest. The relationship becomes humped shaped if $b > 1$, and the derivative can shown to be negative at large N. Whether it is large negative or small negative is unclear from the derivative. Therefore, if $b < 1$ the function represents contest competition, while the form of the competition is unclear from this relationship when $b > 1$.

However, when looking at the relationship between the kvalue and ln(N), $\frac{dk}{d\ln(N)} = \frac{b(aN)^b}{1+(aN)^b}$, which is approximately equal to b at large N. Therefore the kvalue is (nearly) linearly related to ln(N) at large N. This can be interpreted as saying that the function describes contest competition when the parameter b is near one, while it describes scramble competition when b is much larger (section 6.2.2.2). Bellows (1981), Bellows (1982) and Reeve et al. (1998, p439) agree with this interpretation.

The second derivative of kvalue with respect to N is negative if $b < 1$, meaning that only contest competition can be represented, which agrees with the above result. The second derivative can be either negative or positive when $b > 1$ implying both forms of competition can be described by the function. But it is not straightforward to find levels of b that correspond to each form of competition separately as it interacts with N. Instead, as shown in appendix 10, we can find the level of population that corresponds to each type of competition for a given level of b. Given that $b > 1$, competition is scramble if $N < \frac{(b-1)^{1/b}}{a}$, while it is contest otherwise, so scramble competition becomes contest at large N. There is no such a transitional population size if $b < 1$.

If b is only slightly larger than one, then $\frac{(b-1)^{1/b}}{a}$ becomes very small (near zero), therefore the competition is contest over the most of the realistic range of N. On the other hand, if b is large, $\frac{(b-1)^{1/b}}{a}$ becomes large, so scramble competition occurs until the population grows to this level. Therefore, it is reasonable to conclude that for practical purposes, a smaller b leads to contest competition while a larger b leads to scramble.

In conclusion, this function describes contest competition if $b < 1$ or N is large, and scramble competition if $b > 1$ and N is small.

7.8.3 Stability

For this function, $\left.\frac{dk}{d\ln(N)}\right|_K = \frac{b(d-1)}{d}$, and $\left.\frac{dN_{t+1}}{dN_t}\right|_K = 1 - \frac{b(d-1)}{d}$. Both of these expressions lead to the conclusion that the population is

- stable at K without oscillation if $0 < \frac{b(d-1)}{d} < 1$,
- stable at K with convergent oscillation if $1 < \frac{b(d-1)}{d} < 2$,
- information is insufficient to determine the stability if $\frac{b(d-1)}{d} = 0$ or $\frac{b(d-1)}{d} = 2$,
- and unstable otherwise.

A density dependent function has full flexibility in representing asymmetric growth if it can represent the maximum population growth at any population level between zero and K. Since the growth function corresponds to MS&S function has no closed form solution N^* cannot be evaluated analytically.

7.9 Hassell function

I have considered one generalization of the Beverton and Holt function, the MS&S function. Hassell (1975) introduced another generalization of the B&H function, and Hassell et al. (1976) used it to discuss possible dynamical behaviours of populations.

Similar to other functions, various parameterizations of this function can be seen in the literature. I use the

$$f(N) = d(1+aN)^{-b}$$

parameterisation, hence in the count-based models, the dynamics equation becomes $N_{t+1} = N_t d(1+aN_t)^{-b}$. This parameterization was used by Hassell (1975), Hassell et al. (1976), Watkinson (1980), Bellows (1981), Alsop (1998), and Reeve et al. (1998). In this format, $a = 0$ or $b = 0$ implies density independence. The larger the values of a and b the stronger the effect of DD.

7.9.1 The shape of the function

Some features of this function are shown in Figure 7.8. The first derivative of *f(N)* with respect to N is maximum (i.e. DD effect is strongest) at lowest density. The second derivative of *f(N)* with respect to N is positive for all N, so as its shape is only concave up, a sigmoid shape is not possible to represent.

7.9.2 The form of competition

The derivative of N_{t+1} with respect to N_t can be given as $\frac{dN_{t+1}}{dN_t} = \frac{d + daN(1-b)}{(1+aN)^{b+1}}$, which is positive, and approaches zero at large densities if $b < 1$, giving evidence for contest competition. If $b > 1$, this derivative is negative and approaches zero at large densities, which does not provide evidence for either form of competition.

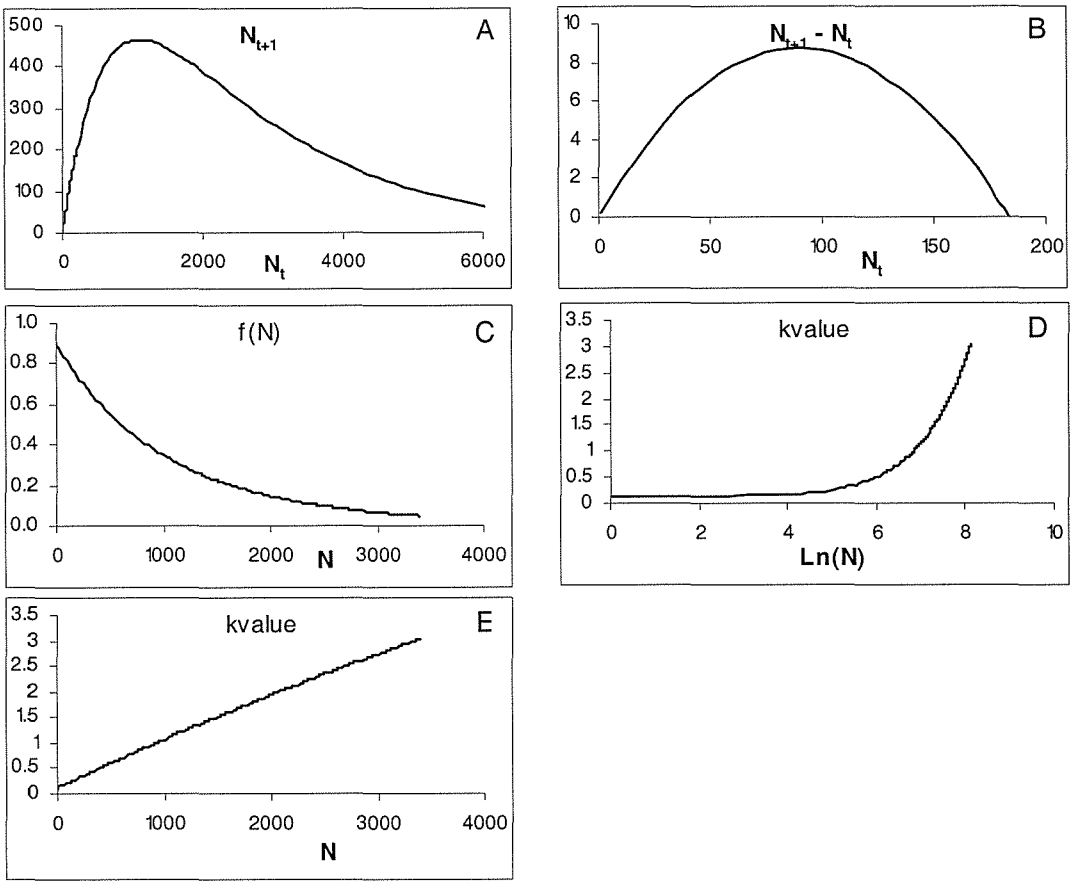

Figure 7.8: An example of the density dependence given by the Hassell function. The panels (A) and (B) use the parameters $d = 1.2$, $a = 0.0001$, and $b = 10$ to represent the dynamics of an initially growing population in a count-based model. The large value of b used in this example causes the N_{t+1} versus N_t curve to have a humped (i.e., Ricker type) shape. If b was smaller the curve would take an asymptotically increasing shape (i.e., Beverton and Holt type shape). In the last three panels, the d was changed to 0.9 to represent a demographic rate over the unit range. The function is non-sigmoid and concave up shaped (panel C).

The second derivative of the kvalue with respect to N is negative for any value of N, irrespective of the values of the function parameters. This implies that only contest competition can be represented by this function. Bellows (1981) agrees with this result. However, $\frac{dk}{d\ln(N)} = \frac{abN}{1+aN}$, so $\lim_{N\to\infty} \frac{dk}{d\ln(N)} = b$. In agreement with Bellows (1981), this can be interpreted as contest competition because the slope of this relationship does not increase with increasing N (section 6.2.2.4). However, on the other hand, this implies that as b changes from one to infinity the function changes from representing 100% contest competition to 100% scramble competition (section 6.2.2.2). Hassell et al. (1976, p473) and Alsop (1998) agree with this result. The two

results are compatible when b is small, but not when b is large. This is an example of obtaining incompatible results when used different measures to distinguish two forms of competitions.

The above inconsistency can easily be examined in the context of count-based models. In those models, the critical density is given by $\frac{1}{a}$ as shown by Hassell (1975). Also, $f(N)=1$ at the equilibrium population size (K), therefore $K=\frac{d^{1/b}-1}{a}$. It is fair to assume that the critical density is smaller than the equilibrium population size, thus $d^{1/b}-1>1$. This translates to $b<\frac{\ln(d)}{\ln(2)}$ providing an upper limit for the parameter b. Therefore, parameter $b<1$ if $d<2$. This is the likely situation for most long-lived species. Small b corresponds to contest competition under the both above results. Therefore, for most long-lived species, this function likely to represent only the contest competition.

In summary, the function represents contest competition when b is smaller or close to one. It is not very clear which form(s) of competition can be represented when b is larger because it is dependent on the specific cut-off level used to distinguish between the two forms of competitions.

7.9.3 Stability

When using the function in count-based models, $f(N)=1$ at the carrying capacity (K), therefore $K=\frac{d^{1/b}-1}{a}$. Form this we can see that $\left.\frac{dk}{d\ln(N)}\right|_K=b\left(1-d^{-1/b}\right)$. Similarly, $\left.\frac{dN_{t+1}}{dN_t}\right|_K=1-\left\{b\left(1-d^{-1/b}\right)\right\}$. Both of these lead to the following results on the neighbourhood stability of the population at K.

- If $0<b\left(1-d^{-1/b}\right)<1$ the population is stable with monotonic damping
- If $1<b\left(1-d^{-1/b}\right)<2$ the population is stable with oscillatory damping

- If $b\left(1-d^{-1/b}\right)=0$ or $b\left(1-d^{-1/b}\right)=2$ the information is insufficient to determine the stability.
- The population is unstable otherwise.

Therefore the function can demonstrate different stability conditions.

It is not possible to analytically find the level of N that corresponds to the maximum growth. The growth function (N_{t+1} –N_t) cannot be solved for the N_t that maximises growth. It is not clear whether the maximum growth can occur at any level of N between zero and K (i.e., at any fractional density of K).

7.10 Usher Function

Ullyett (1950) introduced this function to illustrate density dependence by fitting it to mortality data from a fly population. Later Pennycuick et al. (1968) and Usher (1972) generalized its use for other demographic variables and formally incorporated it into structured population models. May et al (1974) used it in count-based models.

In its most simple and general form the Usher function can be given as

$$f(N)=d\left\{1+\exp(bN-a)\right\}^{-1}$$

Similar to all of the other functions we considered so far, the range of *f(N)* is from zero to *d*. But, under this function, of course depending on the values of the parameters, it is possible that $f(N)\approx d$ when the population is below a certain level, and $f(N)\approx 0$ at higher population levels (Figure 7.9 for example). This knife-edge onset of density dependence seems biologically unacceptable in some situations. Also, it is common to use linear transformations of this function to cover different ranges, say from *d1* to *d2* (e.g., May et al. 1974). The following discussion is based on the untransformed version of the function. I will discuss some consequences of using transformed versions later in section 7.11.

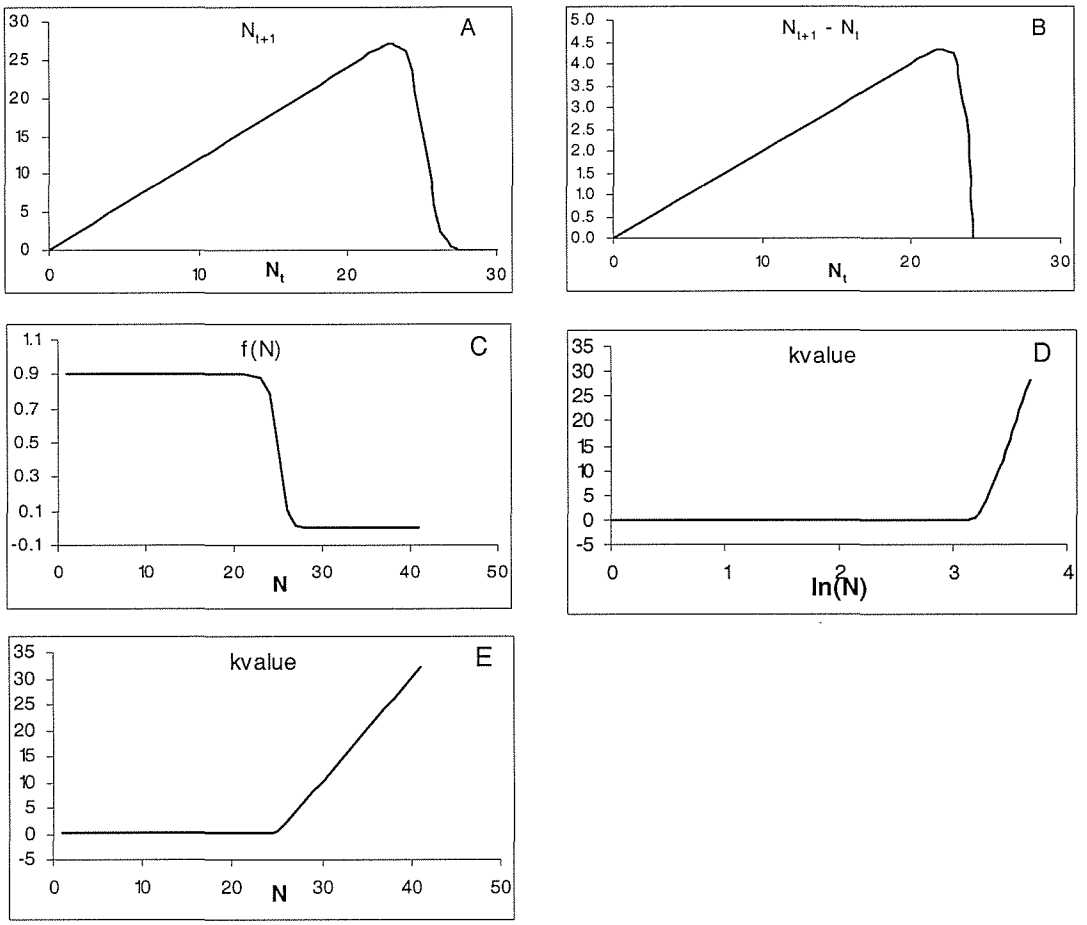

Figure 7.9: An example of the dynamics given by the Usher-function-based density dependence. The panels (A) and (B) use the parameters d = 1.2, a = 50, and b = 2 to represent the dynamics of an initially growing population in a count-based model. In the next three panels the parameter d is changed to 0.9 to represent a demographic rate on the unit range. Panel (C) shows the shape of the function while panels (D) and (E) show the relationship of kvalue to ln(N) and N.

7.10.1 The shape of the function

Some features of this function are shown in Figure 7.9. With the parameter values used in the figure, N_{t+1} is nearly proportional to N_t (i.e., population growth is almost density independent) until it reaches near carrying capacity, where it then drops abruptly to near zero. The growth also follows a similar pattern, so the growth is asymmetric.

The function *f(N)* =1 at $N = \frac{a + \ln(d-1)}{b}$. This is the carrying capacity (K) in count-based models. (Therefore K does not exist if d <1, which is an intuitive result.) Therefore, the parameter a is usually much larger than b so that K can be realistically large[13].

The first derivative of the *f(N)* function with respect to N approaches zero for large densities. It also approaches zero as N approaches zero if a is large[14]. The second derivative of *f(N)* with respect to N is negative if $N < \frac{a}{b}$, is positive if $N > \frac{a}{b}$, and is zero if $N = \frac{a}{b}$. Therefore, provided that the parameter a is large, the *f(N)* function has a sigmoid shape with an inflection point at $N = \frac{a}{b}$. Density dependence is most sensitive to changes in population level at the inflection point. The inflection point and carrying capacity are similar for most populations, especially for large mammals, because $\ln(d-1)$ is usually small. Therefore DD effects are greatest near the carrying capacity.

As seen from the Figure 7.9, the function is nearly density independent ($f(N) \approx d$) until N approaches near K, is highly density dependent around K, and again nearly density independent ($f(N) \approx 0$) soon after K. At the inflection point, $f(N) = \frac{d}{2}$, hence the function is symmetric around the inflection point.

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[13] K needs to be large under the assumption that N is measured by number of individuals, an implicit assumption used throughout the thesis. Suppose N is measured by a larger unit such as carrying capacity, parameters a and b need to be very similar.

[14] $\frac{df(N)}{dN} = \frac{-bd\exp(bN-a)}{\{1+\exp(bN-a)\}^2}$. Therefore $\lim_{N\to\infty} \frac{df(N)}{dN} = 0$. Also, $\lim_{N\to 0} \frac{df(N)}{dN} \approx 0$ if a>>0. Therefore a>>0 is a requirement for sigmoid shape.

7.10.2 The form of competition

As panel (A) of Figure 7.9 shows, N_{t+1} drops almost to zero if N_t increases beyond a certain level, indicating scramble competition. The slope of this plot is negative and approaches zero at large N, so it does not provide evidence of one form of competition over the other. For this function, $\frac{dk}{d\ln(N)} = \frac{bN\exp(bN-a)}{1+\exp(bN-a)}$, which increases without limit, with increasing N. Hence in one way this can be interpreted as representing scramble competition (section 6.2.2.2). However, on the other hand, $\lim_{N\to\infty}\frac{dk}{d\ln(N)} = bN$ can be interpreted as representing both forms of competition in equal proportion (section 6.2.2.4). (This is another example of obtaining incompatible results when used different measures to distinguish two forms of competitions).

The second derivative of the kvalue with respect to N is $\frac{b^2\exp(bN-a)}{\{1+\exp(bN-a)\}^2}$, which is positive for any combination of the parameters. This implies that this function, in its present form, can represent only scramble competition. However, in practice this function is used with linear transformations where contest competition too can be represented (see later, section 7.11).

7.10.3 Stability

The stability of the population at the carrying capacity can be examined by using the slope of kvalue vs ln(N), or the slope of N_{t+1} versus N_t at K. It can be shown that $\left.\frac{dk}{d\ln(N)}\right|_K = \frac{(d-1)\{a+\ln(d-1)\}}{d}$ and $\left.\frac{dN_{t+1}}{dN_t}\right|_K = 1-\frac{(d-1)\{a+\ln(d-1)\}}{d}$. Both of these lead to the same condition of stability at carrying capacity - that is, the population is stable if $0 < \frac{(d-1)\{a+\ln(d-1)\}}{d} < 2$. Note that the parameter *b* has no effect on the population stability at K.

The Figure 7.9 shows that asymmetric growth is possible under this function. But it is not possible to find the population level that corresponds to maximum growth analytically.

The properties of all the functions discussed in this chapter are summarized in Table 7.1 in page 203.

7.11 Linear transformations of functions

Some of these density dependent functions are used in practice as linearly transformed versions of the forms given in this chapter (even if the same parameterization is used). For example, Runge (2002) used the logistic function, Chivers (1999) used the θ-logistic function, and May et al. (1974) used the Usher function with linear transformations. In the untransformed form the value of these functions, *f(N),* has a range from zero to *d* (except with the Usher function when *a* is small). They can be used to represent any desired range, say from d_L to d_U, by using linear transformations of the form

$$f_1(N) = d_L + \left(\frac{d_U - d_L}{d}\right) f(N)$$

In this section I look at the robustness of the properties of DD functions for these linear transformations.

I use the Usher function as an example because it is very common to find examples of using this function with linear transformations, especially in count-based population models. The population dynamics given by this function in its present (untransformed) form could be unrealistic in count-based models at least for two reasons. First, the function is symmetric around the inflection point (i.e., $f(N) = d/2$ at $N=a/b$). Second, for most parameter combinations, the function is nearly density independent until the population level approaches near K, and then the population almost certainly collapses soon after K. This second unrealistic feature can be avoided by using linear transformed versions of the function like

$$f(N) = d_L + (d_U - d_L)\{1 + \exp(bN - a)\}^{-1}.$$

With the lower and upper limit of *f(N)* being specified by d_L and d_U, this version allows flexibility on the location and the range of the function[15] (e.g., May et al. 1974). This linear transformation simply shifts and re-scales the DD curve along the vertical axis without altering its shape qualitatively. Therefore it does not change its sigmoid shape.

In count-based models, the above linear transformation predicts a different carrying capacity for the population, namely $K = \frac{a + \ln\{(d_U - 1)/(1 - d_L)\}}{b}$. As with the untransformed version, the transformed version is nearly density independent ($f(N) \approx d_U$) until N approaches near K, is highly density dependent around K, and again nearly density independent ($f(N) \approx d_L$) soon after K. The inflection point at N= *a/b* is not changed by this transformation, where $f(N) = (\lambda_L + \lambda_U)/2$ (i.e., *f(N)* is still symmetric around the same inflection point). The density dependence is most sensitive to changes in population sizes around this inflection point which is not far from K.

For the transformed version, $\left.\frac{dk}{d\ln(N)}\right|_K = \frac{(1-d_L)(d_U-1)\left[a + \ln\{(d_U-1)/(1-d_L)\}\right]}{(d_U - d_L)}$.

Therefore the condition of stability at K is $0 < \frac{(1-d_L)(d_U-1)\left[a + \ln\{(d_U-1)/(1-d_L)\}\right]}{(d_U - d_L)} < 2$.

As with the untransformed version, the stability is independent of the parameter *b*. However, the stability of the population under this version could be different from that of the untransformed version because it is dependent on parameters d_U and d_L.

Unlike the original untransformed function, the population does not collapse under the transformed version when the density is larger than K, and instead remains close to the level of d_L. The transformation also changes the type of competition represented, as the second derivative of the kvalue of the transformed function can now be shown to be either negative or positive, depending on the values of the parameters d_L and d_U. (It was always positive in the untransformed function, always representing scramble

[15] Note that with this transformation the upper limit does not become d_U exactly. When density is zero $f(N) = d_L + (d_U - d_L)\{1 + \exp(-a)\}$. This is nearly d_U because the parameter *a* is usually large.

competition). Therefore, unlike the untransformed version, the transformed version can represent either scramble or contest competition.

In conclusion, the transformed and untransformed versions do not always have the same properties. Although this is only shown using a single function, it illustrates a general feature of these transformations that the properties of functions are sensitive to linear transformations. This result is important because modellers appear to use such transformations simply to cover a different range without realising the possible consequences. Therefore before using such a transformation, it is important to realize that these transformations could cause changes to some of the properties of the original function. If the form of the function is different from the one used here, the actual form of the function used in the population model (after any linear transformations) needs to be evaluated using the criteria given in chapter 6, rather than relying on the summary of results in table 7.1.

7.12 Summary of functions

As seen above, the properties of density dependent functions can represent a variety of population characteristics. Varying degree of nonlinearity in shape, and different combinations of contest and scramble forms of competitions are expressed by various functions. Functions can exhibit different stability properties at carrying capacity. Some functions represent the asymmetric growth in such a way that maximum population growth could occur at any population level between zero and K. Obviously there could be more population specific characteristics that are useful in a given situation, but which I haven't discussed here.

In chapter six I developed a guideline to select a DD function to suit the characteristics of the population. The objective of the present chapter was to demonstrate the practicality of that guideline, both in matching population characteristics to functional properties, and in selecting a general (i.e., flexible) function. In situations where we know the population characteristics, we can select a function with corresponding properties, whereas when dealing with a population of

unknown characteristics we may want to select a function that is general enough for its properties to be consistent with almost all population characteristics. To help with that decision, I produced a guideline (Table 6.2), and evaluated properties of some commonly used functions using that guideline (Table 7.1). I do not intend to suggest one function over the others because the most suitable function depends on specific population characteristics, but rather I present an objective approach that can be used in selecting a suitable function.

Not all functions are completely independent. It is obvious that the logistic and Ricker functions are special cases of the θ–logistic and θ–Ricker functions respectively, and that the B&H function is a special case of both the MS&S function and the Hassell function. One may assume that these special cases are not important here because they are already covered by more general functions. However, if the properties of a special case function are consistent with the characteristics of the population then the simpler function is preferable as fewer parameters are needed to be specified (or to be estimated from data).

Both the logistic and B&H functions are Taylor series approximations of the Ricker function. Similarly both the θ–logistic and MS&S functions are Taylor series approximations of the θ–Ricker function. One function being an approximation of another does not imply that their properties are similar, nor that they are based on similar assumptions about the underlying dynamics. As seen above, these approximations do change some of the main properties of the functions. Most of these approximations are valid only when the parameter d is small - i.e., in the case of count-based models when the growth rate is small, (Eberhardt 1977), as evidenced by the observation that the Ricker curve approaches the B&H curve only when d is small (Pitcher and Hart 1982, p188).

In this chapter we presented properties of different functions in their most basic form. In practice, various linear transformations are used with most of these functions. These linear transformations may cause changes to the functions' properties - such as changing the nature of competition, altering the stability status, and shifting the population size that corresponds to maximum growth either towards zero or K, thus altering any asymmetric growth. It is unlikely that a linear transformation will cause

any qualitative changes to the shape of the function, even if does make some quantitative changes. These changes can add more flexibility to the function when describing the dynamics of a population.

Out of the nine functions considered in this chapter, only three of them (θ–Ricker, MS&S, and Usher) can exhibit a sigmoid shape. Of these three, only the θ–Ricker and MS&S can represent both forms of competition. However, although the Usher function in its original form can only represent scramble competition, it can represent both forms of competitions in practice, because of the linear transformations used with it. Different forms of population stabilities can be expressed by all three of them by using different combinations of parameters. Therefore these three functions were selected for further studies in the next chapter where I will explore the behavioral differences of these functions in a population model.

Table 7.1: Summary of properties of DD functions.

			Shape Indicators			Form of competition indicators		Asymmetry indicators		Stability indicators	
Name	**function f(N)=**	**K**	**DD is maximum when**	**Sign of** $\frac{d^2 f(N)}{dN^2}$	**Sigmoid shape possible?**	**sign of** $\frac{d^2 k}{dN^2}$	**Contest/ scramble?**	**N at Maximum growth (N*)**	**Possible range for N***	$\left.\frac{dk}{d\ln(N)}\right]_K$	**Requirement for stability at K**
logistic	$d\left[1-\frac{N}{a}\right]$	$a\left(\frac{d-1}{d}\right)$	Never	zero	no	+	Scramble	$\frac{K}{2}$	only at K/2	$(d-1)$	$1<d<3$
θlogistic	$d\left[1-\left(\frac{N}{a}\right)^b\right]$	$a\left(\frac{d-1}{d}\right)^{1/b}$	N = 0 if b<1. N =*a* if b>1. never if b=1.	+ if b < 1. - if b > 1. zero if b = 1.	no	+	Scramble	$K\left(\frac{1}{1+b}\right)^{1/b}$	Between K/e and K.	$b(d-1)$	$0<b(d-1)<2.$
Ricker	$d\exp(-aN)$	$\frac{\ln(d)}{a}$	N = 0	+	no	zero	Both equally	$\frac{K}{e+\ln(d)}<N^*<\frac{K}{2+\ln(d)}$	Between zero and K/2	$\ln(d)$	$0<\ln(d)<2$
θRicker	$d\exp(-aN^b)$	$\left(\frac{\ln(d)}{a}\right)^{1/b}$	$N=\left(\frac{b-1}{ab}\right)^{1/b}$ if b > 1	change from - to + when N increases.	yes	- if b < 1. + if b > 1. zero if b = 1.	Scramble if b > 1. Contest if b < 1. Both equally if b = 1.	no analytical solution	Between zero and K.	$b(d-1)$	$0<b(d-1)<2.$
Haldane	dN^{-b}	$d^{1/b}$	N = 0	+	no	negative	Contest	$K(1-b)^{1/b}$	Between zero and K/e.	b	$0<b<2$
B&H	$d[1+aN]^{-1}$	$\frac{d-1}{a}$	N = 0	+	no	negative	Contest	$\frac{K}{d^{1/2}+1}$	Between zero and K/2.	$\frac{d-1}{d}$	$d>1$
MS&S	$d[1+(aN)^b]^{-1}$	$\frac{(d-1)^{1/b}}{a}$	$N=\frac{1}{a}\left(\frac{b-1}{b+1}\right)^{1/b}$ if b > 1	change from - to + when N increases, if b > 1.	yes	Positive if $N<\frac{(b-1)^{1/b}}{a}$. negative otherwise	Contest if b < 1 or N large. Scramble if b > 1 and N small.	no analytical solution	Not known	$\frac{b(d-1)}{d}$	$0<\frac{b(d-1)}{d}<2$
Hassell	$d[1+aN]^{-b}$	$\frac{d^{1/b}-1}{a}$	N = 0	+	no	negative	Contest only. May be scramble if b large?.	no analytical solution	Not known	$b(1-d^{-1/b})$	$0<b(1-d^{-1/b})<2$
Usher	$d\left[1+\exp(bN-a)\right]^{-1}$	$\frac{a+\ln(d-1)}{b}$	$N=\frac{a}{b}$	Change from - to + when N increases	yes	positive	Mainly scramble (both in transformed versions)	no analytical solution	Not known	$\left(\frac{d-1}{d}\right)[a+\ln(d-1)]$	$0<\left(\frac{d-1}{d}\right)[a+\ln(d-1)]<2$

Chapter 8 Effect of Density Dependence Function on Population Predictions

8.1 Introduction

8.1.1 Objective of the chapter

In chapter six I discussed the properties of density dependent functions in relation to their ability to represent population characteristics of dynamics models. I identified at least four important properties that a function should have in order for it to qualify as a possible candidate for a 'general DD function' that can be used in a model for a population whose characteristics are unknown. Namely, those properties are:

- ability to have a sigmoid shape,
- ability to represent both forms of competition (contest and scramble),
- ability to represent asymmetric population growth so that the maximum population growth can occur at any fraction of the population's carrying capacity,
- the possibility of representing various dynamics with regards to the stability of the population.

Where possible, analytical ways of verifying those properties for a given function are also presented in that chapter.

Chapter seven examined a range of DD functions on the basis of the above properties, which were selected to cover the full spectrum of functions presently in use. Out of the functions considered we identified a few functions that have enough flexibility in the representation of the various possible characteristics of a population with

unknown characteristics. It is not clear how to select one function over the others in modelling a given situation. We cannot use a statistical fit of the function to data as the basis for this choice when we do not have enough data to estimate the density dependence parameters for various candidate functions. Also, Runge and Johnson (2002) and Pascual et al. (1997) clearly showed that a statistical fit to data cannot provide a solid basis for selecting a DD function. This is because, since several functions can often be fitted to the data equally well, traditional model selection methods based on the parsimony principle are not practical in distinguishing between alternative functions. Even if different functions are statistically indistinguishable within the range of the data they can be very different outside the data range. Therefore the statistical fit can only be used to rule out inappropriate functions, not to select appropriate ones.

This makes it necessary to investigate the effect of the choice of DD function on model predictions. This is important given that the form of density dependence can be difficult to estimate from data. If the population inferences from models are insensitive to the choice of functional form then it relieves modelers from the burden of searching for a DD function suitable for the question in hand (because a subjective selection of a function does not influence the predictions). On the other hand, if population inferences are sensitive to the functional form, it becomes necessary to examine the possibility of representing DD in a relatively function-independent way.

This sets the theme for the present chapter. In the first half of this chapter I evaluate the effect of DD functions on model predictions. In the second half I examine the possibility of modelling density dependence in a relatively function-insensitive way, preferably using less input information. I consider the situation in which density dependence is known to exist in more than one vital rate, but where there is not enough information in the data to estimate parameters for DD functions for each vital rate separately. It could also be the possible that the strength of density dependence of one vital rate is influenced by the level of DD in other vital rates. This chapter introduces a way to incorporate DD into some situations like this.

The question of the effect of DD functions on the population predictions have been addressed in the literature. For example, Runge and Johnson (2002) demonstrated that

the model structure had a significant effect on population predictions. Their work is different to this work for two reasons. First, they used an unstructured model. The way that they select parameter values for DD functions cannot be generalised to structured models. Second, they selected parameters for different functions so that the population reaches the same carrying capacity for all functions. That does not ensure that all the functions represent the same strength of DD, so the results are not exactly comparable. Pascual et al. (1997) examined the same question using a structured model. But all the functions included in their study were simple two-parameter functions that were not selected for this analysis for reasons which are given below. Furthermore, their study was based on estimating parameters for all functions from a single set of data, therefore the difficulty of ensuring the same strength of DD was not applicable (see section 8.2).

8.1.2 Short list of functions

Out of the functions considered in previous chapter, three of them (θ-Ricker, MS&S, and Usher) were selected for further consideration in this chapter because of their flexibility in representing various population characteristics. However, this choice dose not mean that other functions are unsuitable or inappropriate for a given population. For example, if a certain population is known to produce maximum growth when the density is near its carrying capacity (K) then we do not have to look for a function that can produce maximum population growth at any level of density between zero and K. The θ–logistic function does not have a sigmoid shape, cannot describe contest competition, and is unable to produce maximum population growth below the population level of K/e. However, that function still has a wide history of having been used in population models. In particular, it is currently being considered in modelling the New Zealand Sea Lion (NZSL) population, (the Hilborn model and the Breen-Kim model proposed for the NZSL both use this function, see chapter 2). Therefore I also selected the θ-logistic function for comparison with the three functions chosen above.

8.1.3 The approach

The comparison is based on using these selected functions in a population model to predict population outcomes. It is easier to interpret outcomes from a simpler model than from a more realistic model which include characteristics such as stochasticity, senescence, catastrophe, etc. For example, if both density dependence and stochasticity were included in the model then we could examine the behaviour of the stochastic model under different DD functions in a more realistic situation than in a model with density dependence alone. Such an examination would be essential in practice if we intended to examine the fate of a given population, as the exclusion of stochasticity from the model could drag the analysis away from the real situation. In addition, any observable difference in outcomes of a model with no stochasticity but with different DD functions does not provide an assessment on how significant those differences are relative to other sources of variations such as stochasticity and uncertainty. However, density dependence, especially scramble competition, can contribute a complex component to population dynamics. Some times this complexity is very difficult to be distinguished from the effect of inherently unpredictable forces such as environmental stochasticity (Morris and Doak 2002, p323). If we include both stochasticity and density dependence in a population model, then we do not know whether any resultant variation is due to density dependence or due to stochasticity. In order to meet the objective of this chapter, it is easier to compare the effect of different forms of DD functions on the model predictions when stochasticity is absent, i.e., when different simulations differ only by the DD function. For this reason I opted to use a deterministic model with no representations of catastrophic events, senescence etc.

The intention of this exercise is to examine whether the choice of DD function has a significant influence on model projections, not to find a 'good' DD function. If the model outcomes are insensitive to the choice of DD function then we can ignore the differences between DD functions, and can use any of those functions in a model. Using simulations we can observe possible problems that could arise when particular functions are used, and see whether different functions lead to similar or dissimilar predictions. If the predictions are dissimilar between functions we have to select the one that makes more accurate and biologically realistic predictions. The accuracy

cannot be assessed in practice when the real outcome is unknown. Therefore, the way around this problem is to use functions with most flexibility in representing various population characteristics, and see which function leads to the most biologically acceptable outcomes. Therefore the simulation is not used here as a tool of assessing the suitability of functions, but rather it serves as a tool to demonstrate the importance of careful selection of functions. Very flexible functions tend to be more sensitive to choice of parameter values, thus when using such functions sampling error in parameter estimates could be more important. Therefore, as stated earlier in the thesis, selecting a flexible function is not an attempt to seek more flexibility than necessary. For example, if it is known the concerned population produces its maximum growth near K then we do not have to look for a function that has flexibility in producing maximum growth at very low levels of densities.

8.1.4 The flow of the chapter

In the first half of the chapter, I use different functions in the same population model to compare simulated population outcomes. To be consistent with chapter five, I opt to use a generic structured demographic model. In order to make simulated populations comparable I attempted to ensure that the strength of DD represented by all functions is the same. Section 8.2 explains how this was ensured. Section 8.3 presents the details of the simulations and the results. In section 8.4, I present a way in which density dependence can be modelled in more than one vital rate interactively in a situation where the available data is insufficient to model DD in individual vital rates independently.

8.2 Generating same level of density dependence from various functions

In the next section of this chapter, I use a case study to examine how the form of DD function influences the predictions of a population dynamics model. In order to ensure that the model outcomes are comparable between functions it is imperative that we

have the same level of density dependence used with each function. One way of doing this is to use a single data set to estimate DD parameters for all the DD functions, guaranteeing that all functions have the same level (or strength) of DD. The problems associated with estimating DD parameters from data are well known (e.g., Barker et al. 2002), and dealing with them is out of the scope of this chapter. Therefore I use another approach in forcing the DD in all the functions to have the same strength. In this approach one of the parameters of a DD function is used as an index for the level of density dependency. Then parameters for other functions with comparable strength can be specified in terms of that index. By using those parameter values, this makes sure that each function reflects the same level of density dependence[1]. In this section I explain this approach using an example. I first explain the term 'level of density dependence', then define what it means for two or more functions to have the 'same level of density dependence', and finally show how different functions can actually be made to have the same level of DD.

Let us consider a specific vital rate and any one of the DD functions, for example adult survival rate (S) and the MS&S function $S = d[1+(aN)^b]^{-1}$. Here the parameter d is a scaling constant which can be interpreted as the theoretical level of the vital rate at $N = 0$. Therefore the curve of the DD function (i.e., S vs N curve) goes through the point $(0, d)$, while the shape of the curve is determined by the other two parameters (a and b). Assume that the parameter d can be specified.

If we can specify the level of vital rate for some other level of N, then we fix another point on the curve. This could be the observed level of vital rate in field data (i.e., $S = S'$ at $N = N'$). Any DD curve used to represent density dependence of this vital rate should pass through both of these points irrespective of the DD function or DD strength (Figure 8.1, panel A). The selected function, and its a and b parameters determine the shape of the DD curve that passes through these two known points, and thus the strength of the density dependence is determined. But we can explicitly express one of the two parameters in terms of the other. For example, for MS&S

[1] This is comparable to the approach used in chapter 5 in forcing each stochastic distribution to have the same level of stochasticity. In that chapter the level of stochasticity was defined in terms of the mean and standard deviation of the vital rate. The parameters for each distribution were specified in terms of those pieces of information ensuring all distributions produced the same level of stochasticity.

function, $a = \frac{1}{N'}\left(\frac{d - S'}{S'}\right)^{1/b}$, hence the parameter b becomes the sole determinant of the shape of the curve. Therefore the DD curve can be fully specified by d, N', S', and b. By changing parameter b one can obtain DD curves from that function with different strengths as shown in Figure 8.1, panel A.

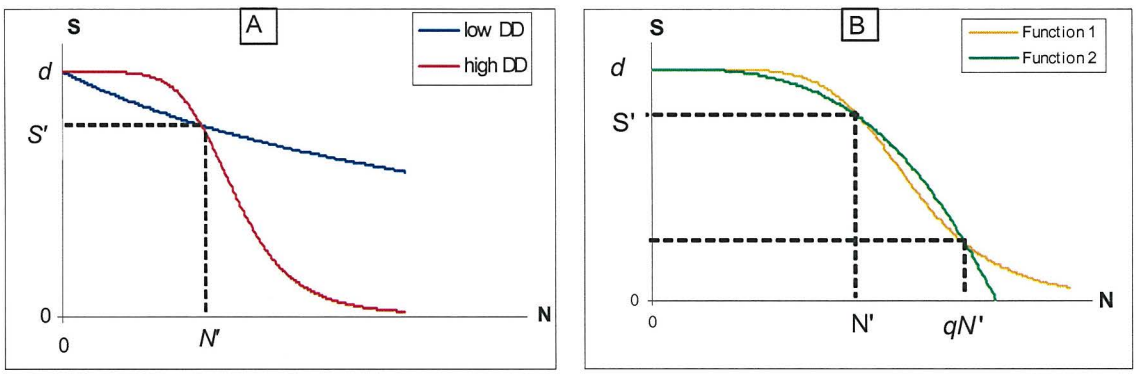


Figure 8.1: **Panel A:** Illustrative sketch of two curves of a same DD function (in this case MS&S function) having different strengths. Both curves pass through two specified points *(0, d)* and *(N', S')*. Parameter d is common for both curves. Parameter b determines parameter a, hence determines the strength of DD.
Panel B: Illustrative sketch of two DD curves from different functions (in this case from MS&S and θ–logistic functions) with the same strength. Curves pass through three common points. Parameter b for function1 is specified, and from that the corresponding b for function2 is derived as detailed in appendix 11.

Since any curve used to represent the DD relationship of this vital rate needs to pass through two fixed points, the above idea is applicable to any DD function. Therefore, we can obtain DD curves with different strengths by changing the value of the parameter b of the corresponding function (i.e., for each function, the parameter b defines the strength of DD)[2].

To ensure the comparison of population outcomes between DD functions is fair, we need to have the 'same' (or a comparable) level of DD under each function; consequently, the term 'comparable strength of DD' needs to be clarified. If the strength of the density dependence is the same under any two (or more) DD functions then the slopes of those curves are also the same at each level of N. Suppose that

[2] This requires expressing parameter a of the function in terms of b of the same function, as shown here for the example of the MS&S function. Although it can be done for most three-parameter functions, including all functions considered in this thesis, there is no guarantee that this can be done analytically for any given DD function.

those curves also go through a common point then the level of the vital rate will be the same under both of those functions at each level of N (i.e., the two curves will be identical). That implies, in a strict sense, that different functions cannot have the same level (or same strength) of DD. For this reason, we define the term 'same level of DD' with a slightly different meaning. We define *two functions have the comparable strength of DD (or same level of DD) if the curves of those functions meet at least at three specified population levels* (say, for example, at N = *0, N = N'*, and *N = qN'*, where *q* is a constant larger than one)[3]. *N'* could be the present (or observed) population level, therefore *qN'* is an arbitrary population level. This definition offers flexibility of adjusting the population range covered by the two comparable functions (i.e., flexibility in the range from *N = 0* to *N = qN'*), say, for example, from zero to carrying capacity K by selecting $q = \frac{K}{N'}$. I use the term 'same DD level' to imply DD levels are the same according to this definition (Figure 8.1, panel B).

We can use this definition to derive DD curves from each function so that all of them represent the same (specified) level of DD. Say one function is defined using two of its parameters (i.e., *d* and *b* parameters) and a point on its curve (*N', S'*). Then its curve passes through two points $(0, d)$ and (N', S'), and its strength is determined by the parameter *b*. We need to derive parameters for the second function with the comparable strength of DD and passing through the same two points as shown in panel B of Figure 8.1. Suppose, for example, if the second function is θ-Ricker where $S = d\exp(-\alpha N^{\beta})$ we need to specify its α and β parameters in terms of the parameters of the first function (parameter *d* is common for both functions). Appendix 11 shows how this can be done analytically for the four functions used in this chapter. In this way we can get DD parameters for all four functions for any given level of density dependence[4]. If the population model contains more than one density dependent vital rate (as in the population model used in section 8.3) the above process

[3] The usefulness of this definition depends on the choice of the three specified population levels. It is meaningful to choose population levels those are not too close to each other. The likely minimum and maximum population levels and the observed level could be one set of sensible choices. The present choice of N=0, N=N', and N=qN' is based on that.

[4] Although appendix 11 shows how this can be done analytically for all the functions used in this chapter, it does not guarantee that this can be done analytically for any arbitrary pair of functions. Numerical solutions could be used in cases where analytical solutions are not available.

can be repeated for each vital rate in order to obtain DD curves with comparable strengths.

8.3 Effect of DD function on model predictions

8.3.1 The population model and the simulation

All four DD functions are used in a population dynamics model to examine the effect of the choice of function on population predictions. The population model used was the same as the one used in chapter 5 (see section 5.2), but with the environmental stochasticity in vital rates was removed, and density dependence was included instead. It is important to recall that although the context of NZSL population was used here, the model is inadequate to represent the dynamics of the NZSL population realistically. Therefore the model outcomes cannot be interpreted as predictions for the NZSL population. The model is very simple, but it has the essential features of the Leslie matrix type models used in conservation biology. The model is stage-structured, deterministic, with birth pulse reproduction and a postbreeding census. The model is summarised as follows.

- Stage structure with three stages: stage1 (newborns), stage2 (juveniles), and stage3 (adults/breeders). The duration of stage2 is variable as only a fraction of stage2 animals become mature and move to stage3 each year. All surviving stage3 animals remain in the same stage, so there is no maximum age for animals.
- No stochasticity, allee effect, catastrophe, or senescence is included (for reasons given in section 8.1).
- Under this model, the population dynamics equation becomes (in the notation used in chapter 5)

$$\begin{bmatrix} 0 & 0 & S_3 R \\ S_1 & S_2(1-\gamma) & 0 \\ 0 & S_2\gamma & S_3 \end{bmatrix} \begin{bmatrix} N_{1,t} \\ N_{2,t} \\ N_{3,t} \end{bmatrix} = \begin{bmatrix} N_{1,t+1} \\ N_{2,t+1} \\ N_{3,t+1} \end{bmatrix}$$

This dynamic equation has five vital rates (S_1, S_2, S_3, R, and γ), with no density dependence in vital rate γ. This parameter is evaluated as in chapter 5 (formula 5.2).

Density dependence is present either in the reproductive rate (R), or in all stage specific survival rates (S_1, S_2, and S_3), or in both. For simplicity, I choose to have all three survival rates either density dependent or all three density independent. In that way, I have three models: DD in survival only model (DDS model), DD in reproduction only model (DDR model), and DD in both survival and reproduction model (DDSR model). I assigned three levels of density dependence (low, moderate, and high) within each of the three models, and within each of the four DD functions, resulting in 36 combinations of possible projections.

8.3.1 (a) DD parameter values used

As explained in section 8.2, the parameters of all four DD functions should correspond to the same strength of DD. For this reason I used the input parameters in Table 8.1 for the MS&S function, and parameters for other functions were derived from them using the method described in section 8.2. The parameter b = 2 refers to the lowest level of density dependence. For moderate and high levels of density dependence parameter b was changed to 4 and 6 respectively and parameters for the other three functions were re-evaluated.

Table 8.1: Parameters used for the MS&S function at the lowest strength of DD. Parameter b was changed to 4 and 6 respectively for moderate and high strengths of DD.

Parameter	Vital rate			
	S_1	S_2	S_3	R
d	0.80	0.85	0.90	0.45
S'	0.75	0.80	0.85	0.40
N'	6040	6040	6040	6040
b	2.0	2.0	2.0	2.0
q	2.0	2.0	2.0	2.0

Although I do not intend to model any specific population here, the above input parameter values were selected to roughly represent the demographic parameters of the New Zealand Sea Lion population (NZSL), which was introduced in chapter 2. The first row (d) of the table contains the assigned values of the corresponding vital rates at zero density. The next row (S') shows the likely levels of those vital rates at the population level given in the 3rd row. Therefore the first three rows of the table

specify the first two common points in each of the four DD curves at the lowest strength of DD (Figure 8.1). The population size of 6040 in the 3rd row was found by first estimating the stable-stage distribution and scaling it to 1400 newborns, and then taking the total population size, excluding newborns. That implies the term 'density' in DD functions means the total population size excluding newborns. 1400 is roughly the present annual production of female newborns, and 6040 roughly represents the present (female) population size of NZSL. The stable stage distribution was found using the vital rates in the second row, along with additional information of $\bar{t} = 2$ years and V(t) = 1.5 years, ($\bar{t}$ and V(t) are the mean and variance of the duration of stage2 respectively, as described in chapter 5). This stable distribution corresponded to $\gamma = 0.435$, meaning 43.5% of surviving stage2 animals progress to stage3 annually, which seems reasonable for the NZSL.

The population model was programmed in Matlab® and the population was projected forward covering three levels of DD under each DD function. Since all trajectories were observed to adhere to a fixed-point attractor, projection continued until the population reached a stationary level (K), making K the terminal population size. The resultant trajectories were compared between different functions using the predicted stationary population level and the time to reach K as performance indicators. The annual population growth rate, a commonly used performance indicator, was not used here because it is dependent on the chosen projection period for density-dependent models. Different indicators have been used in the literature for similar purposes. For example, Neubert and Caswell (2000) used population stability (convergence, chaotic behaviour, bifurcation, etc) as an indicator to compare the effect of DD on various vital rates. Such indicators are not applicable to long-lived slow-growing animals.

For each trajectory in this simulation I used the same DD function for all density dependent vital rates. I opted to do that because of the aim of this exercise is to examine the effect of the DD function on the outputs of the population model. This examination would have been quite complicated if the different vital rates followed different DD functions. For the same reason, I used the same strength of DD for all vital rates in each trajectory. This facilitated the interpretation of the effect of strength on population outcomes. However, in reality, DD curves in survival and reproduction need not follow the same function. In addition it is possible to have a high level of

density dependence in the survival rate while a low level in the reproductive rate, and vice versa. I haven't explored those situations because of the aim of this exercise is not to study the fate of a population but to explore the effect of the DD function on possible projections.

8.3.2 Simulation results

(a) Trajectories from the DDS model

In this model all survival rates are taken to be density dependent while the reproductive rate is taken to be density independent. There were 12 population trajectories under the DDS model (four DD functions at three DD levels). In these combinations of input vital rates each trajectory increases towards its own stationary population level (K) as shown in Figure 8.2. The level of K was dependent on the DD function and on the strength of the DD. This stationary level is smaller (see Figure 8.2) and is reached faster when the DD is stronger (see Figure 8.3). At all levels of DD, the MS&S function leads the population to K faster than the other functions. K is reached most slowly under the Usher function at lower DD levels, while it is reached most slowly under the θ-logistic at higher levels of DD.

The curves for the four DD functions used in the simulation for each density dependent vital rate at the lowest and highest DD levels are shown in Figure 8.4 (curves for the reproductive rate are also shown here as they were used in DDR and DDSR models later). They were produced using the same DD parameters that were used to produce Figure 8.2 (estimated as per appendix 11), and are therefore directly comparable to the above trajectories. They are useful in understanding how the differences between trajectories are related to the corresponding DD functions.

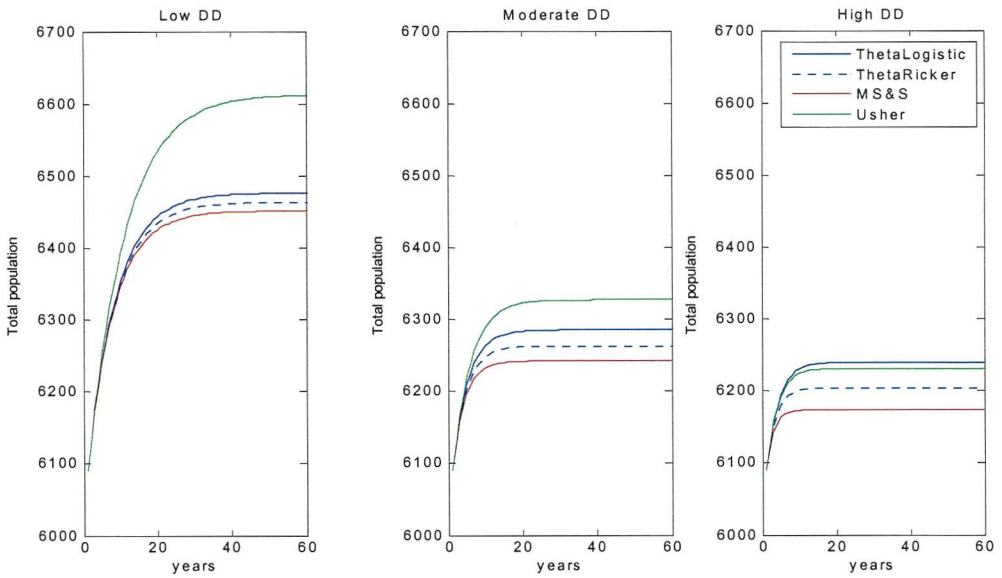


Figure 8.2: The trajectories under the DDS model. The higher the DD level the lower the K (i.e., the closer the K is to the starting population size), and the more quickly that K is reached.

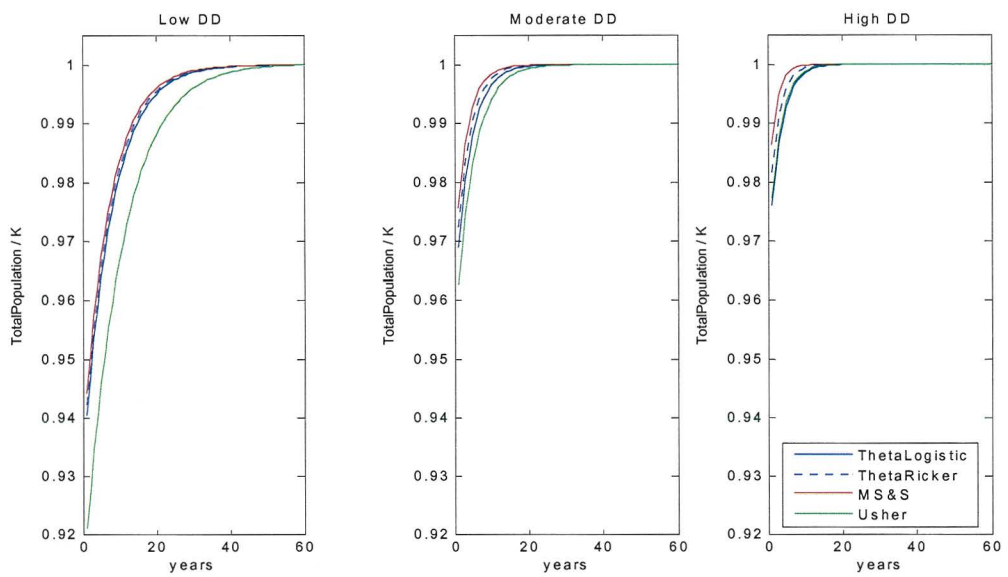


Figure 8.3: Trajectories in Figure 8.2 scaled by their own K. Since K is different for each trajectory, this scaling makes them easier to compare. Generally, under any function, K is reached faster when DD is stronger. Under the same level of DD some DD functions lead the population to K faster than others.

The top four panels of Figure 8.4 show that the vital rates simulated by the Usher function at the lowest density are different from their corresponding d parameters given in Table 8.1. The bottom panel does not show such a discrepancy. The reason for this can be seen from the formula of the Usher function which says $f(N) = d[1+\exp(-a)]^{-1}$ at $N = 0$. Here the parameter a is large at higher levels of DD, therefore $f(N) \approx d$. At lower levels of DD, a is smaller, and so $f(N) < d$. However this difference is at lower population levels, and it cannot explain the observed differences in trajectories, because Figure 8.2 shows that our population size ranges from about 6000 to 6600, which is not small.

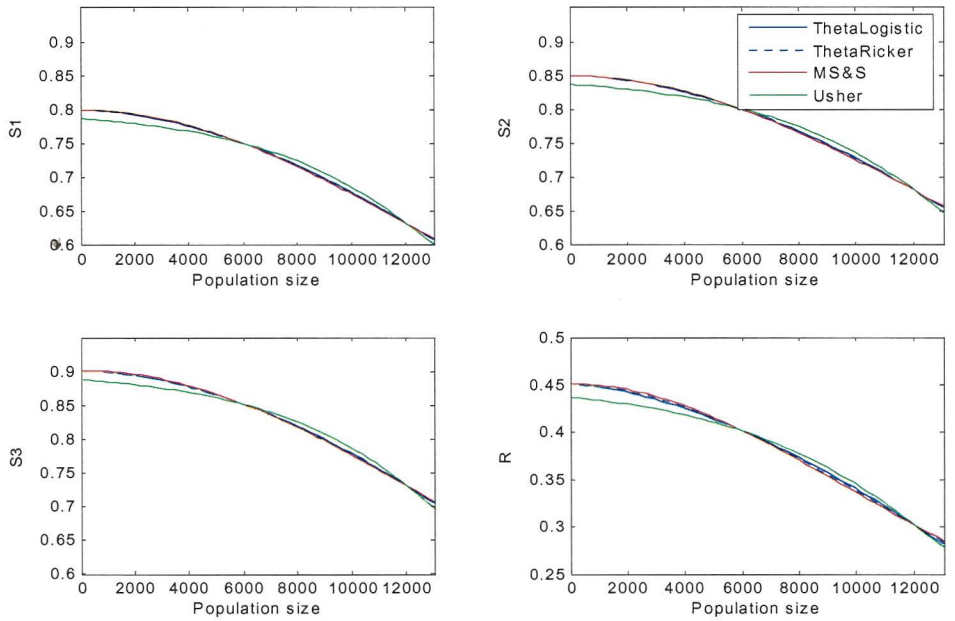
Apart from the above difference, the DD functions seem very similar at low levels of DD within the range of the population size. However those minor differences in DD functions have been reflected as large differences in the corresponding population trajectories (Figure 8.2), suggesting population trajectories are highly sensitive to the functional forms used to represent the density dependence in vital rates.

(b) Comparison of trajectories from DDS, DDR, and DDSR models

The population trajectories from the DDR model and the DDSR model are shown in Figure 8.5. It can be observed in comparison to the corresponding trajectories from the DDS model in Figure 8.2 that

- Under the DDR model, K is reached the most slowly, and the resultant K is largest.
- Under the DDSR model, K is reached the most quickly, and the resultant K is smallest.
- K in the DDS model is slightly larger than K in the DDSR model, and is much smaller than K in the DDR model. This model takes a slightly longer time to reach K than the DDSR model.
- These patterns are common to all DD functions and to all three DD levels used.

Low DD level



High DD level

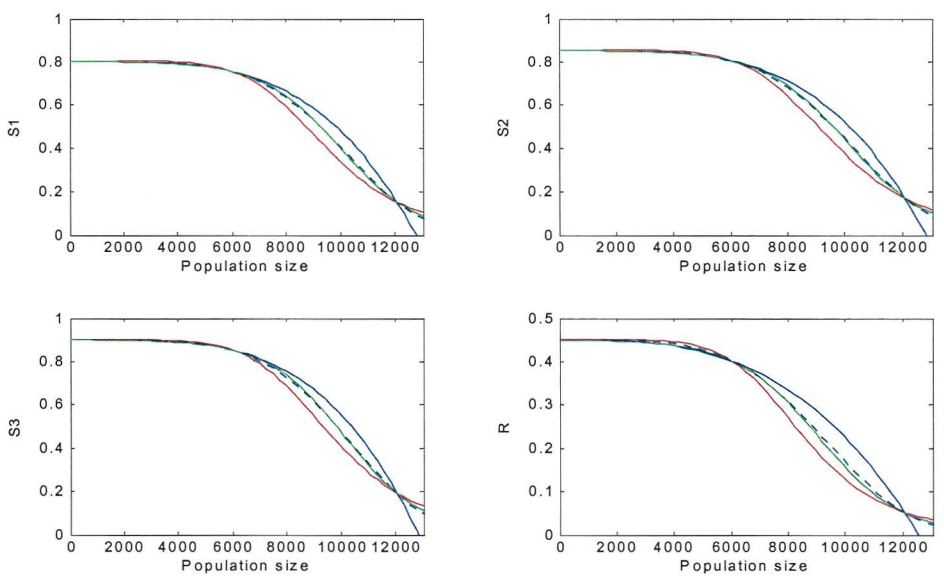


Figure 8.4: The DD functions used for each vital rate at C = 0 for low and high levels of DD. (note different scales in the upper and lower parts of each vital rate).

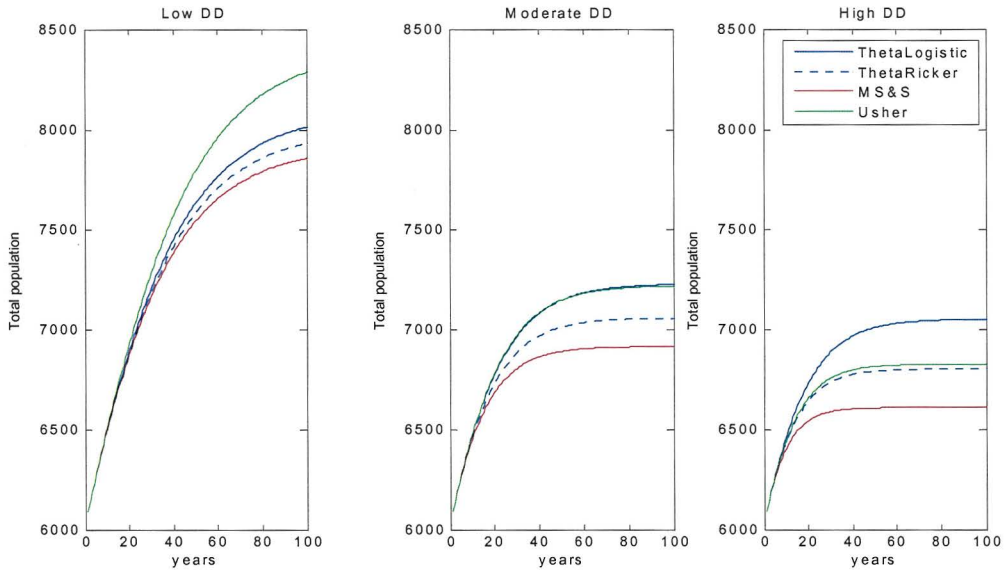
- This implies the effect of density dependence on the population is strongest when density dependence is present in both vital rates (survival rate and reproductive rate). It is weakest when density dependence is present only in reproductive rate. The presence of density dependence in survival rate is more effective than having it in reproductive rate. Introducing density dependent reproduction to a model that already has density dependence in survival rate will have only a small additional effect.
- Including DD in reproduction rate when it is already included in the survival rate has more effect at low DD levels than at high DD levels. This means that if the DD in survival is already sufficient enough to regulate the population then the additional DD imposed by reproduction has only a marginal effect.

The DDS, DDR, and DDSR models differ from one another by the vital rate(s) that are density dependent. The effect of density dependence through various vital rates can be examined by comparing the elasticity of model outcomes to changes in those vital rates. I haven't calculated those measures in the present experiment. Here I used the method in section 8.2 to ensure the DD levels are the same for different DD functions within a vital rate, but not between vital rates. Hence, the strengths of different vital rates are therefore not necessarily the same. Consequently, the trajectories from the DDS, DDR, and DDSR models are not strictly comparable, so the above observations from the trajectories should be used with caution.

(c) Trajectories from growing and declining populations

The purpose of parameter C in the model was to vary the reproductive rate and all the survival rates at the initial year by either increasing or decreasing them all. (This parameter and its role are explained in chapter 5. It is a constant between -1 and +1. If C is positive, input values for all vital rates go up in the initial year, they go down if C is negative). This parameter is useful to examine the effect of density dependent functions on growing and declining populations.

DDR model



DDSR model

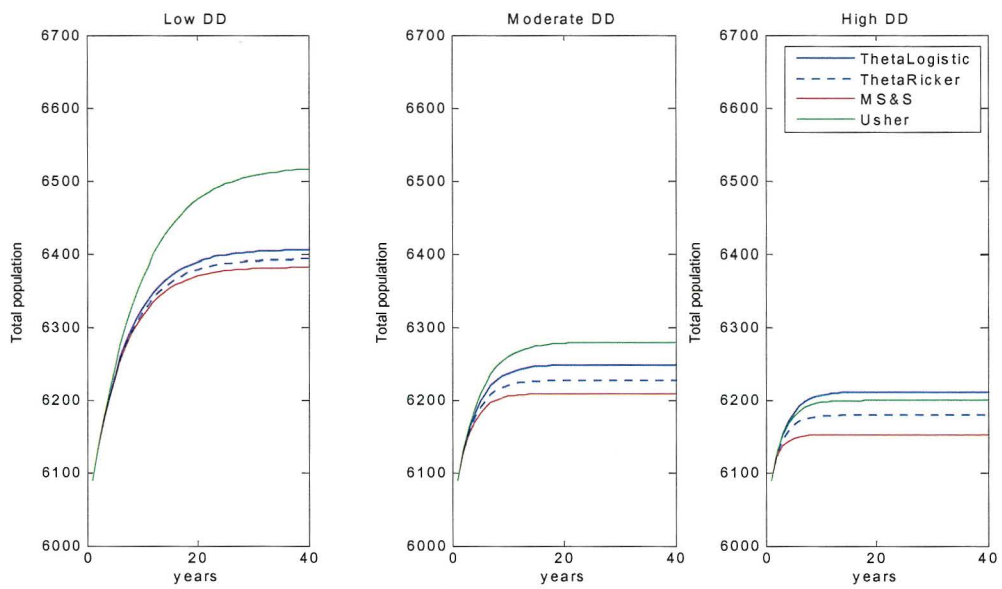


Figure 8.5: Trajectories using the DDR and DDSR models, and with different levels of DD. (Note the different scales in the top and bottom panels, and also in Figure 8.2 where corresponding trajectories are shown for DDS models).

When C was set to +1 (the largest possible value), the resultant trajectories were similarly shaped to those in Figure 8.2. As expected, the population grows to much larger levels (i.e., the K values were larger). The only noticeable qualitative difference from Figure 8.2 was that the Usher function had the largest K at all levels of DD. The Usher DD curves consistently produce larger vital rates for the whole range of population. Because of their similarity with Figure 8.2, those trajectories and the DD curves are not shown here.

Declining population trajectories can be produced by assigning lower (more negative) values to C. Such an example is presented in Figure 8.6 from which we can observe that:

- Similar to increasing populations, all declining populations settle at their own K, which is below the starting population size. When the DD level is higher, the K is reached faster.
- The population level is more resistant to change when the DD level is stronger.
- As with increasing populations, K is closest to the starting population in the DDSR model, furthest in the DDR model, and intermediate in the DDS model.
- The fate of the population is dependent on which vital rate is subjected to density dependent changes. If the DD acts on a more influential vital rate (for example, on the survival rate) the population moves to K more quickly, and that K is not very far from the starting population.

We change the parameter C to examine population trajectories with various levels of growth (and decline) rates. Even though quantitative differences were observed with every change of C, qualitative results were always similar. In all declining trajectories initial population level was above K. Although real populations likely to spend more time in the area below K than above K, the differences observed between DD functions when N is above K have practical important value because stochasticity could push the system to that area in various ways.

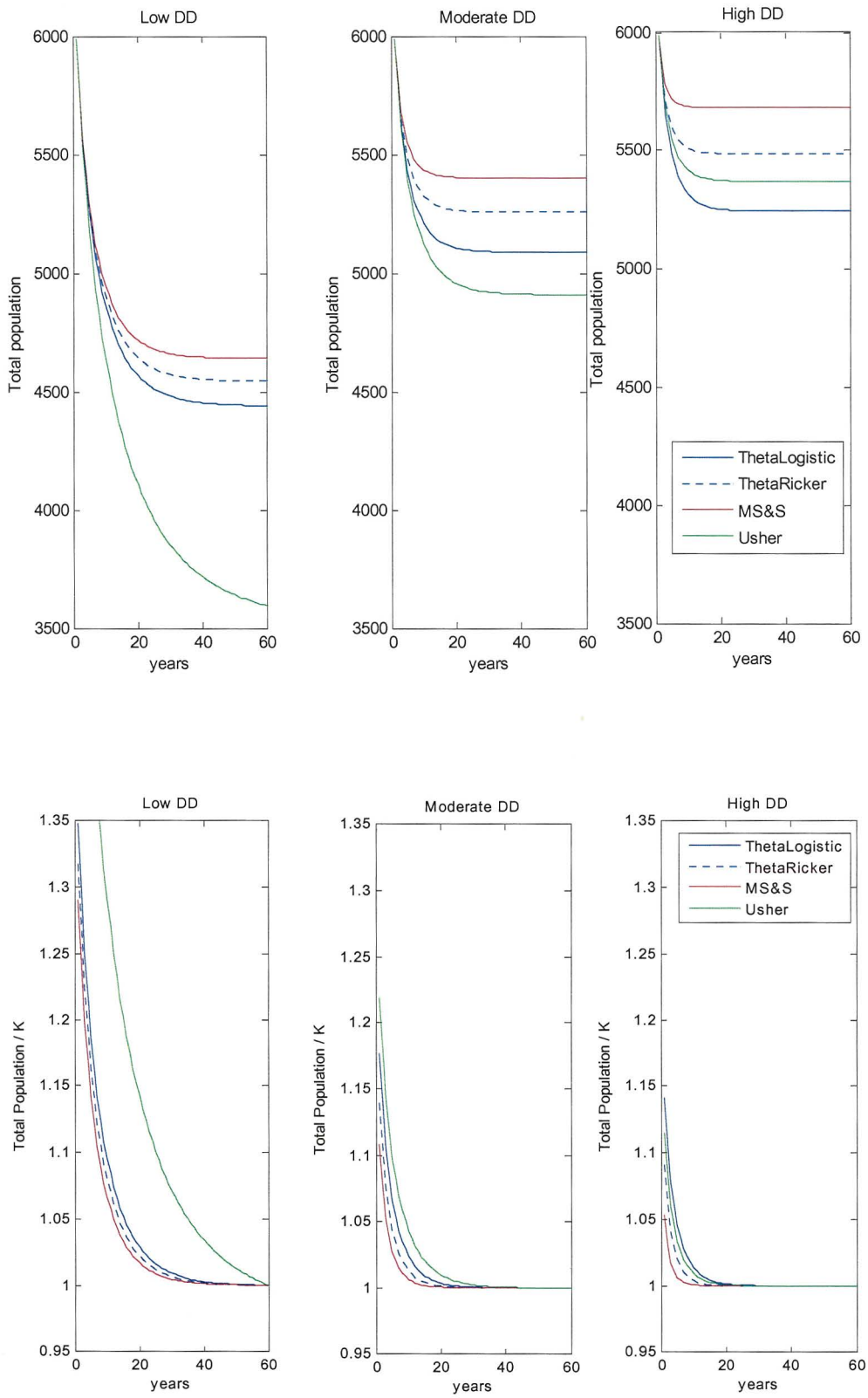


Figure 8.6: The top panel shows declining trajectories while bottom panel shows the same trajectories scaled by their respective equilibrium population levels. The higher the DD the closer the equilibrium population level to the starting population size, and the faster that the population reaches its equilibrium.

In declining populations, one could expect trajectories to decline faster under the Usher function because at small population levels this function tends to produce lower vital rates than are produced by the other functions (top panels of Figure 8.4). However, this was not observed in this exercise. The reason could be that in this simulation, none of the trajectories go down to very a small population sizes even when the smallest possible value (i.e., C = - 1) is used for C.

8.3.3 Discussion on comparison of DD functions

(a) Explanation of differences between trajectories from different DD functions

Carrying capacity (K) is the maximum level of population of a particular species that can be supported by a given habitat or area without damage to the environment (Hine and Martin 2004), and therefore a constant for a given population (at a given time). If so how can we explain trajectories of the same population reaching different levels of K under different functions? Suppose that K is specified explicitly in all the functions then the magnitude of the density dependent vital rates are determined by it, and so all of the trajectories can expected to reach the specified K. However, the carrying capacity is usually unknown and cannot be specified explicitly for most populations, meaning that its magnitude is determined by the vital rates, which are specified by the DD function as in this study. Therefore the resultant value of K is influenced by the choice of that function. Those values of K may or may not be different depending on the closeness of the vital rates determined by the various DD functions. In this study we observed that the resultant Ks are quite dissimilar between functions, even at the same level of DD. This implies that the vital rates realised from the DD functions could have been different. In fact, as is evident from Figure 8.4, vital rates realised at a given population level are dependent on the DD function.

According to Figure 8.2, the population ranges from about 6000 to 6600 individuals during the projection period. The differences in vital rates between the functions are very small in this range of population sizes, especially if the DD level is small (Figure 8.4). However these minor differences in vital rates are magnified considerably into quite large differences in population trajectories, suggesting that the population

trajectories are highly sensitive to the functional form of the density dependence. Such an observation is obvious in cases of density independent models (after enough time has passed) because differences between trajectories become widened with time in those models.

At lower DD levels K is considerably larger under the Usher function than under the other functions. This agrees with the top half of Figure 8.4 which shows the Usher function realises slightly larger vital rates than the other functions within this population range. Because the magnitude of the vital rates is positively related to the growth rate of the population, a function that produces larger vital rates throughout the population range tends to lead the population to a larger level. This explains the considerably larger K observed in Figure 8.2 under the Usher function at the lower DD level. Also the MS&S function which realises the smallest vital rates leads the population to smallest K.

In summary, the trajectories are different for the different functions, even though all the functions are at the same level of DD. Those differences reflect the differences between corresponding DD curves. It is worth recalling what I mean by the 'same level of DD'. Different functions are in fact not at the same level of DD in a strict literal sense; they are said to be 'equivalent' in a particular sense as defined in section 8.2. There are in fact minor differences between those 'equivalent' DD curves as seen from Figure 8.4, and population trajectories are highly sensitive to those differences. The observed differences between trajectories are consistent with the differences detectable in DD curves.

One might expect to see a difference in the trajectories produced by the θ-logistic function which has a differently (non-sigmoidal) shaped DD curve (chapter 7). However simulations do not show a marked difference in corresponding trajectories. This demonstrates the difference between the DD functions and the dynamics of the populations responding to those functions. As can be seen from Figure 8.4, in this particular example, non-sigmoid and sigmoid functions diverge at population levels above 12000, but none of the simulated populations grew to that level. Therefore, any differences in functional properties beyond the realistic population ranges do not have much practical value.

Above statement should not be interpreted as saying that NZSL population responds to the θ-logistic function and other DD functions in a similar way (remember that the θ-logistic function is used in the latest model proposed for the NZSL population). This is because, although I investigate DD functions in the context of the NZSL population, these simulations do not reflect the status of that population for various reasons given earlier.

(b) Effect of DD strength on the difference of trajectories produced by different functions

Figures 8.2 and 8.5 seem to suggest that the dynamics predicted by the four functions differ more at lower levels of DD than at higher levels. This is contrary to the expectation that any difference between DD functions would be more prominent at higher levels of DD than at lower levels[5]. This apparent discrepancy can be explained by their DD curves. The DD curves show larger differences between them when population levels are away from N' and qN' (i.e., from 6000 and 12000 in Figure 8.4). At lower DD levels, the population grows (or declines) to levels much further away from these two points than at larger DD levels, leading to larger differences between trajectories at the lower DD level. Suppose that the population level corresponding to one of the fixed points on the DD curves (i.e., N' or qN' in Figure 8.1) was closer to the K that was reached under the lower level of DD, then the curves would differ more at the larger level of DD, leading to more different predictions at large DD. Therefore our observation is probably an artefact of the way the 'same DD level' is measured. Therefore I am inclined not to consider the observation of more different predictions at lower DD levels as a general result.

In order to investigate this result further, q was adjusted so that qN' in Figure 8.1 lies in the vicinity of K at the lower level of DD (i.e., qN' is nearly 6500, or equivalently q is nearly 1.1). This ensures that the realised vital rates under different functions are same at this level of population because all DD curves meet at $N = qN'$. As noticed

[5] If the DD level is at the absolute minimum then all curves become density independent irrespective of the functions they represent, and so there is no difference between the curves of different functions. As the DD level increases the curves for the different functions become distinguishable from one another even if they all have the 'same DD level', hence a difference between functions begins to appear. This is consistent with the larger difference between the curves in the lower panel of Figure 8.4 than in those in the upper panel. Therefore one would expect to see a larger difference between trajectories at larger levels of DD.

earlier, K at higher levels of DD becomes smaller than the K at lower levels, therefore in this situation, the K at higher levels of DD would be between N' and qN' . The DD curves more likely to be different between functions in this range of population levels than at a population level closer to qN' (see Figure 8.1). Therefore, the projected trajectories would be expected to be more different between functions at the higher level of DD, than at the lower level. The trajectories observed by changing q from its present value of 2 to 1.1 confirmed with this expectation. Therefore, it is reasonable to say that the larger difference between the trajectories at the lower DD level in Figures 8.2, 8.5 and 8.6 is not a general observation, but rather a consequence of arbitrarily selecting the parameter q (i.e., arbitrarily selecting the population level qN').

This result reiterates the statement in section 8.2 that the usefulness of the definition of the 'same level of DD' is depends on the values chosen for N' and q. This result also leads to a serious question: are the observed differences between the various DD functions consequences of arbitrarily selecting three specified points on the DD curves (Figure 8.1)? In order to examine this possible effect, I repeated the model projections using different values of S' and q for all four vital rates (S_1, S_2, S_3 and R). This allows two of the three fixed points at which all DD curves meet (i.e., the two points other than the $(0,d)$ point in Figure 8.1B) to vary. Although these changes had quantitative effects on the resultant trajectories, the main qualitative observations remained the same. That is, the following qualitative observations were observed to be robust to values of above input parameters:

- Trajectories were different when different functions were used.
- Stronger density dependence corresponds to levels of K that are closer to the starting population size.
- Population reaches K faster under stronger density dependence.
- Density dependence in survival has stronger effect than density dependence in reproduction.

(c) Effect of DD strength on the resultant K

When DD is stronger, population growth (or decline) was observed to be smaller than what was observed at lower DD. This is because the predicted K is closer to the

starting population level when DD is stronger than when it is weaker in both growing and declining populations (Figures 8.2, 8.5, and 8.6). The strength of DD is shown by the slope of the DD curve; stronger DD corresponds to a larger slope, and therefore vital rates become more sensitive to changes in density (Figure 8.4). This can explain why the population does not grow (or decline) far away from the starting level when the DD is stronger. It can be seen from these graphs that, even after considering the shorter distance that higher DD level trajectories have to travel to reach their own K, they reach K faster (proportionally) than lower DD trajectories. In this sense, the strength of DD can be viewed as a 'resistance to change', or as an 'inclination to return when perturbed'. This view agrees with Lande et al. (2003) who defines the density dependence as the rate of return to equilibrium when perturbed.

(d) Time taken to reach K

Observed differences in the time to reach K under different functions (when other factors remain constant) could be explained from the relative shapes of the DD functions. The slope of the DD curve reflects the sensitivity of a vital rate to changes in population size. The MS&S function has the largest sloped DD curves at all levels of DD throughout the simulations (Figure 8.4). (Note that, as seen from Figures 8.2, 8.5, and 8.6 the population range covered by this simulation is from about 3500 to 8500 only, hence slopes beyond this range are irrelevant here). This function leads population trajectories to K faster than the other functions do. At the other extreme, the Usher function has the lowest slope and therefore reaches K the most slowly at lower DD levels. At large DD levels the θ-logistic function has the lowest slope and the slowest speed in reaching K. Therefore the observed differences in the time to reach K can be explained by the slopes of the corresponding DD curves. In general, all the differences we observed between population trajectories can be explained by the relative shapes of the DD curves.

(e) Relative effectiveness of DD in Reproductive rate and in Survival rate

We found that both the equilibrium population size and the time taken to reach that population size are more sensitive to the presence of density dependence in the survival rate than in the reproductive rate. This observation is consistent with results reported by Pascual et al. (1997) and Yearsley et al. (2003). For long-lived species, many authors have also noted a strong sensitivity of other population outcomes (e.g., growth rate) to survival rate and weaker sensitivity to fecundity for long-lived species (e.g., Weimerskirch et al. 1997, Crooks et al. 1998, Russell 1999, Heppell et al. 2000, Slooten et al. 2000).

Lande et al (2003) described density dependence as the elasticity of population growth rate with respect to changes in population density. Heppell et al (2000) showed that fast-growing mammal species (such as rodents and small carnivores) generally have relatively high elasticity of growth rate to reproduction and lower elasticity of growth rate to survival. This agrees with Neubert and Caswell (2000) who found that for fast-growing animals, density dependence in survival cannot control the population growth when the reproduction is high, and density dependent reproduction is more likely to cause chaotic dynamics than density dependence in other vital rates. On the other hand, slow-growing long-lived species (such as marine mammals) generally have a relatively low elasticity of growth rate to reproduction and high elasticity to survival. Lebreton and Clobert (1993) quantified this result for long-lived species by showing that the elasticity of population growth rate to fecundity is of the order of $\frac{1}{T}$, and elasticity of growth rate to after-first-year survival rate is nearly $1-\frac{1}{T}$, where T is the population generation time. A consequence of this is that as soon as the generation time is higher than 2, elasticity relative to survival becomes higher than relative to fecundity. This is notably the case for all species that start breeding at age two or later. Therefore population outcomes, especially the population growth rate, are more sensitive to survival than to reproduction[6]. Higher

[6] Management of long-lived species that aim to achieve a target growth rate should therefore focus on adjusting survival rather than on adjusting fecundity. Also, if a change in growth rate is observed, it is more reasonable to suspect a change in survival as the underlying cause than a change in reproduction.

elasticity to survival means density dependence in survival can cause chaotic changes more than density dependence in reproduction can. This agrees with the hypothesis that density dependent population regulation through reproduction is evolutionarily safer than through survival

The existence of a higher elasticity to growth rate does not mean that the presence of density dependence in survival is more important than in reproduction in a realistic model. In fact, a review of mammal population models shows it is more common to have models with density dependence in reproduction than in survival. What could be the reason for this apparent discrepancy? Sensitivity analysis is a prospective analysis (Horvitz et al. 1997). Elasticity analysis says what could happen if DDS (or DDR) were present, but it does not say how likely DDS (or DDR) is to be present. Even though DDR is less effective, its presence could be more important in population models if it is more likely to occur than DDS. It may be that DDR is more likely to be present in populations as it is believed to be evolutionarily safer.

Is there a benefit to the population for having less effective DDR compared to more effective DDS? In other words, as a response to a declining population, is it more beneficial to the population to invest its limited resources on improving reproduction or improving survival? The existence of a negative correlation between survival and reproduction is evident among different populations of the same species (Thomas et al. 2000), and also between different species (Hautekeete et al. 2001), meaning there is a trade off between resource allocations to survival and reproduction. Takada (1995) found that this trade off between resource allocations plays an important role in fitness and evolution. The benefit to the population is usually measured in terms of an evolutionary objective, that is the maximising of the fitness of the population (Ragen and Fowler 1992, Newton 1993, Brommer 2000).

There are various measures of population fitness such as, population size, net productive rate (R_0), population multiplicative rate (λ), and intrinsic growth rate (r), with R_0 and r being the most common. Using R_0 as the measure, Teriokhin (1998) showed that, on the basis of maximising the evolutionary benefits, it is more

beneficial to have density dependent changes in reproduction than in survival when a population is decreasing.

Therefore, even though our results show that projections are more sensitive to density dependent changes in survival, it may be reasonable to pay more attention to density dependence in reproduction in population modelling whenever field data indicates density dependent changes in survival are negligibly small compared to corresponding changes in reproduction[7].

(f) Comments about the method of selecting 'same level of DD'

We changed the DD level simulated by the various functions by changing their parameter b with two points on the DD curve, (i.e., the $(0\ ,\ d)$ and $(N',\ S')$ points in Figure 8.1) having been specified. This makes the parameter a of those functions change with b. In this way we found that the larger the DD level the smaller the corresponding K. Therefore one might think that the DD level is an indicator for K. However, we obtained this result only under the restriction of two points having been specified on the DD curve.

In the case of unstructured models, carrying capacity can be given in terms of the parameters of the DD function. For count-based models those formulas are given in Table 7.1 for the various functions. It is clear from these formulas that if the above restriction is not in place, the same K can be achieved by using various combinations of the function parameters. Different combinations of parameters correspond to different strengths of DD, and so the same K can be achieved by different strengths of DD. Therefore the DD level cannot be an indicator of the carrying capacity. In general, DD level is not an indicator of the carrying capacity; instead it determines how quickly the population returns to carrying capacity when perturbed.

The same carrying capacity does not imply the same level of density dependence. Therefore, we cannot start by specifying K and work backward to find the corresponding function parameters when we want to have the same DD level under

[7] However, we need to be aware of the lessons of Pascual et al. (1997) that the density dependent trends apparent in data may not show the true trend.

each function (as we wanted in this exercise)[8]. First, we do not know the carrying capacity. The intention of this study is to compare the effect of selecting one DD function over another, when the status of the population we intended to model (including the carrying capacity) is unknown. Second, we cannot work backward from K to find function parameters when working with structured demographic models unless the model is very simple, because there is no analytical formula for K for such models (Caswell 2001, p518). That compelled me to use the method explained in section 8.2 to ensure all functions have the 'same DD strength'. As stated in that section, this method does not generate density dependence from different functions with 'exactly the same strength', rather it generates approximately similar strengths. In the absence of other information, my definitions of 'DD level' and 'same DD strength' offer a natural and practical way to compare density dependence.

8.4 Interactive Density Dependent model (IDD model)

8.4.1 Introduction to the IDD models

So far in this chapter I examined the population predictions that could result from population models when various forms of density dependence relationships were used. I assumed separate DD functions can be estimated (or can be specified in some other way) for various vital rates. In this section I examine a way of incorporating DD into a situation where separate DD functions cannot be defined by available input information.

The intention of the previous part of the chapter was to investigate the effect of subjectively selecting one DD function over another. The comparison of DD functions was based on using all functions in a population model and comparing their outcomes. To ensure that the results were comparable I used the same strength of DD with all functions. We observed some differences between model outcomes that depended on functions used. This means that the choice of DD function can influence the population outcomes, so having a function-independent method (or a less function-

[8] Such exercises can be seen in the literature, for example (Runge and Johnson 2002).

dependent method) of modelling DD is preferable. The present section of the chapter examines a way of modelling DD in a relatively function-independent way.

In a demographic modelling exercise if we decide which DD function is to be used then we need to estimate DD curves for each vital rate. This involves estimating parameters for each curve (three parameters for each curve in the cases of the functions selected for this chapter). This raises the question of how we can incorporate density dependence into a demographic model if we do not have enough information in the data to estimate separate DD curves for each vital rate. This section introduces a way to incorporate DD into such a model when an extra piece of information is available, for example when an estimate for the carrying capacity of the population is available or the growth rate at a particular population level is known (or can be specified).

We can state the above aim slightly differently, but in a related way. Although we used one functional form for all the vital rates at a time, it involves using different DD curves for each vital rate. We assumed the DD curves for various vital rates are independent of each other (even though the values of vital rates realised from those curves are determined by the same population density for a given year, the curves themselves are specific and unchanged). The value of each demographic rate corresponds to a given population size, and is determined by the corresponding fixed DD curve regardless of the levels of the other vital rates. How can density dependence be modelled when the DD curve in one vital rate is dependent on the value of another vital rate? Dependence of one vital rate on another is the subject of cross-correlation which we do not aim to model in this thesis as stated in section 4.1.4. Instead, what we consider here is the deterministic dependence of one vital rate on another due to internal factors. A simple example for such a situation is, when the carrying capacity (K) is constant over the years, then if one vital rate becomes highly favourable for some reason then one or more of the other vital rates need to be unfavourable to compensate, so that the population does not overshoot the carrying capacity.

Suppose we feel more comfortable with specifying K than specifying some of the DD parameters. Then this more reliable information can be used with existing methods to model DD if the population model is count-based. That is because an unknown DD

parameter can be expressed in terms of K easily. If the model is demographic this can only be done if only a single vital rate is density dependent. The method introduced in this section allows modelling DD when:

(a) more than one vital rate is density dependent in a demographic model,
(b) density dependence in one vital rate is deterministically influenced by the level of another vital rate,
(c) not enough information is available to calculate separate DD curves for each vital rate.

The approach I use allows the DD curves to make dynamic changes at each time step by interacting with each other, and so I have named it Interactive Density Dependent model (IDD model). To illustrate this method I use the simplest form of demographic population model, a demographic unstructured model with two density dependent vital rates (i.e., survival rate and reproductive rate).

8.4.2 Interactive Density Dependent model (IDD model)

Let us denote the DD functions for survival rate (f_S) and reproductive rate (f_R) by $S_t = f_S(N_t; d_S, a_S, b_S)$ and $R_t = f_R(N_t; d_R, a_R, b_R)$ respectively, where subscripts s and R depict parameters for the f_S and f_R functions respectively, and N_t is the population size at time t. We specify the carrying capacity of the population (K), the values of the survival and reproductive rate at the lowest density (i.e., d_S and d_R), and one other point on each DD curve. Using the notation in section 8.2, I denote the specified point on the survival curve as $(N_S^{'}, S^{'})$, and the point on the reproduction curve as $(N_R^{'}, R^{'})$.

From this information we cannot derive separate DD curves for the two vital rates. That is because we need to specify four parameters (a_S, b_S, a_R, and b_R), whereas we have only three pieces of information: $f_S(N_S^{'}) = S^{'}$, $f_R(N_R^{'}) = R^{'}$, and $\lambda = 1$ at $N = K$. Therefore we cannot use a DDSR population model (as used in section 8.3) with any of the three-parameter DD functions. However, we can use a DDSR model with the IDD model. The basic difference between the IDD model and the ordinary DDSR model used in section 8.3 is that in the IDD model we do not use fixed (specific) DD curves for vital rates. Instead the DD curves in IDD models are

dynamic and interacting; one curve depends on the other and they are re-estimated at each time step. This means that the density dependent survival function at time t is determined by the reproductive rate immediately prior to time t, and the density dependent reproductive function at time t is determined by the survival rate immediately prior to time t. This sequence of re-calculating DD curves for survival and reproduction is shown in Figure 8.7 for prebreeding and postbreeding census models separately.

The population dynamics equation for an unstructured demographic model can be given as $N_{t+1} = \lambda_t N_t$, where $\lambda_t = S_t(1+R_t)$, and S_t and R_t are the density dependent survival and reproductive rates respectively at time t. This is applicable to models with either prebreeding or postbreeding censuses, irrespective of which vital rate(s) are subjected to density dependence, and which DD function is used to represent those relationships. We can find expressions for λ_t under various density dependent functions by substituting relevant expressions for S_t and R_t, and hence the population can be projected.

IDD models are applicable only when more than one vital rate is subjected to density dependence, therefore only the DDSR version of the model is relevant in the present context (there are no DDS or DDR versions of IDD models). Here we assume that each annual cycle is comprised of two discrete events: survival of animals over the year, and their reproduction[9]. The reproductive rate remains constant during the event of survival, and the survival rate remains constant during the event of reproduction. The sequence of these two events within an annual cycle is dependent on whether the model refers to a prebreeding or postbreeding census (described in section 3.3.1). I consider both prebreeding and postbreeding situations in this exercise. Now I illustrate how DD parameters for each vital rate can be calculated interactively in IDD models.

[9] The assumption of discrete events is not correct for populations with birth-flow reproduction (Caswell 2001). Therefore, we have made an implicit assumption here; namely that, the population has a birth pulse reproduction, which is the approximate situation found in seasonally breeding animal populations.

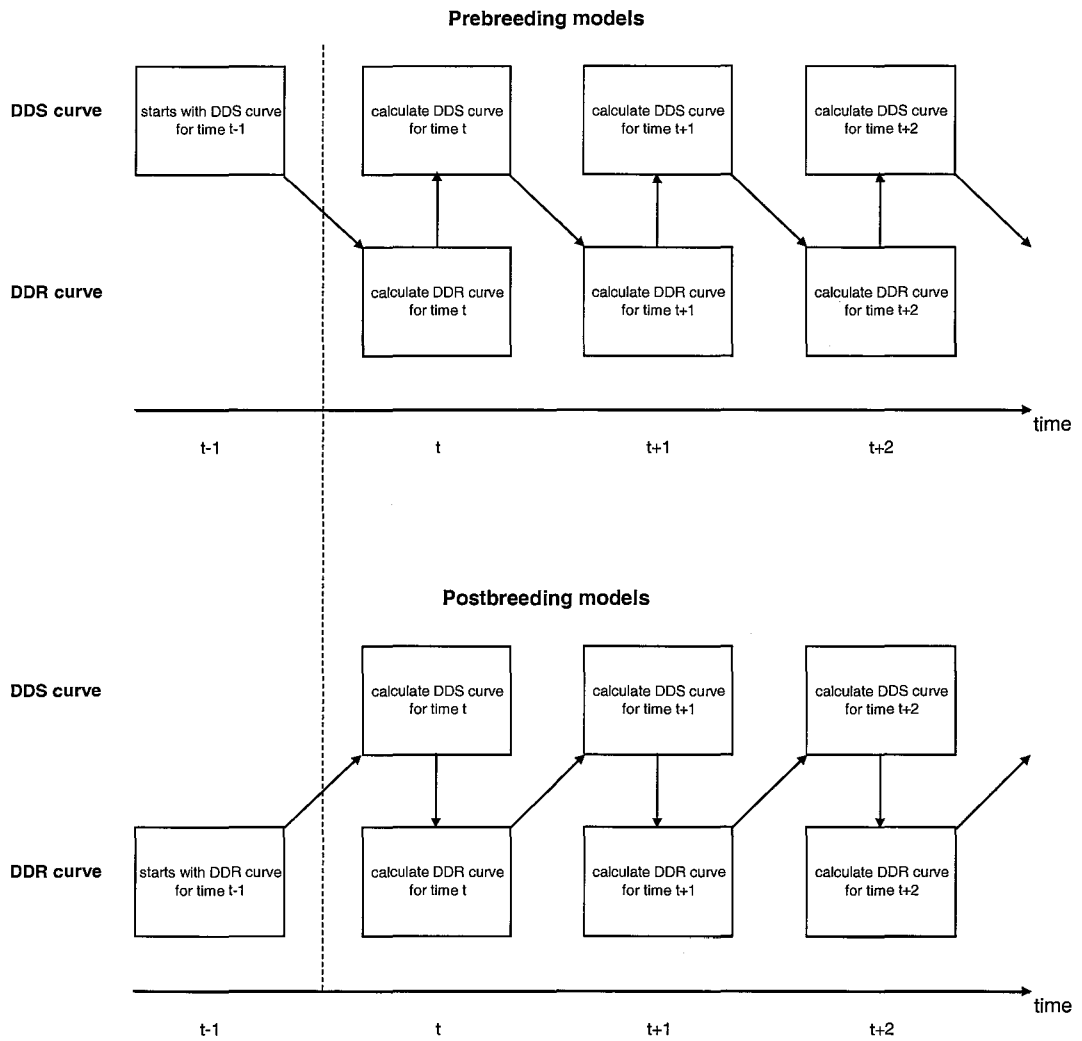


Figure 8. 7: The sequence of re-calculating DDS and DDR curves in IDD models. Arrows show the flow of input information for each calculation.

8.4.3 Calculating parameters for IDD models

Since we cannot specify the four parameters (a_S, b_S, a_R, and b_R) required for separate DD functions for survival and reproduction from the three pieces of information we have in our possession IDD models aim to use one of the pieces of information (i.e., $\lambda = 1$ at $N = K$) to calculate both functions. When calculating the function for one vital rate at time t, the value of the other vital rate is assumed to remain constant at the level immediately prior to time t. In this way, one of the DD functions is re-calculated immediately prior to each event (i.e., there are two re-calculations per year).

I use the θ-logistic function as an example to illustrate the interactive parameter re-calculation at each time step. The function specific details for other DD functions are given in appendix 12. I assume a postbreeding census model where the number of animals in a given year refers to the numbers immediately after breeding. Therefore, individuals have to survive for another year before the next breeding starts; hence the sequence of the two events within the year is first survival then reproduction. In both events, the effective vital rate is determined by the respective time-specific density dependent function. We need to calculate parameters for time-specific DD functions for both vital rates from the three pieces of information stated earlier.

The density dependent survival rate under the θ-logistic function is

$$S_t = d_s\left[1-\left(\frac{N_t}{a_s}\right)^{b_s}\right]$$, where a_S and b_S are unknown parameters.

Using the specified point of $S_t = S'$ at $N_t = N_s^{'}$ on the survival curve and rearranging we get,

$$a_s = N_s^{'}\left(\frac{d_s}{d_s - S'}\right)^{1/b_s} \qquad \textbf{(8.1)}$$

Using the relationship $\lambda_t = S_t(1+R_{t-1})$,

$$\lambda_t = d_s\left[1-\left(\frac{N_t}{a_s}\right)^{b_s}\right](1+R_{t-1}).$$

Note that R_{t-1} is the reproductive rate in the previous annual cycle. This is consistent with the assumption that the two events are discrete. When an event happens in one year, its rate does not change until the same event happens in the following year. Therefore, the reproductive rate in the previous year (R_{t-1}) hasn't changed by the time survival happens in the current year. In general terms, IDD models assume any vital rate applicable at the current time is determined by the level of the other vital rate immediately prior to the current time.

Since $\lambda_t = 1$ at population carrying capacity (K),

$$1 = d_s\left[1-\left(\frac{K}{a_s}\right)^{b_s}\right](1+R_{t-1}).$$

By rearranging this we obtain

$$a_s = K\left[\frac{d_s(1+R_{t-1})}{d_s(1+R_{t-1})-1}\right]^{1/b_s}. \qquad \textbf{(8.2)}$$

Using expressions 8.1 and 8.2 we find the b_s parameter to be

$$b_s = \frac{1}{\ln(N_S^{'})-\ln(K)}\ln\left[\frac{(1+R_{t-1})(d_s-S')}{d_s(1+R_{t-1})-1}\right]. \qquad \textbf{(8.3)}$$

Substituting this into (8.1) gives the parameter a_s. The DD curve defined by these parameters can then be used to determine the survival rate for the year *t*. The next event in the annual cycle is the reproduction. The density dependent reproductive rate under the θ-logistic function is

$$R_t = d_R\left[1-\left(\frac{N_t}{a_R}\right)^{b_R}\right],$$

where parameters a_R and b_R need to be specified in terms of 'known' information. Using the specified point of $R_t = R'$ at $N_t = N_R^{'}$ on the reproductive curve and rearranging we get,

$$a_R = N_R^{'}\left(\frac{d_R}{d_R-R'}\right)^{1/b_R} \qquad \textbf{(8.4)}$$

Using the relationship $\lambda_t = S_t(1+R_t)$,

$$\lambda_t = S_t\left[1+d_R\left\{1-\left(\frac{N_t}{a_R}\right)^{b_R}\right\}\right]$$

Since $\lambda_t = 1$ at $N_t = K$

$$1 = S_t\left[1+d_R\left\{1-\left(\frac{K}{a_R}\right)^{b_R}\right\}\right]$$

By rearranging this we get

$$a_R = K\left[\frac{S_t d_R}{S_t(1+d_R)-1}\right]^{1/b_R} \qquad \textbf{(8.5)}$$

Using expressions 8.4 and 8.5 we find the b_R parameter to be

$$b_R = \frac{1}{\ln(N_R^{'})-\ln(K)}\ln\left[\frac{S_t(d_R-R')}{S_t(1+d_R)-1}\right]. \qquad \textbf{(8.6)}$$

Substituting this into (8.4) gives the parameter a_R. The DD curve defined by these parameters determines the reproductive rate for the current year.

The corresponding formulas for the prebreeding census model can be found in the same way by interchanging the order of the two events within the year. As shown in Appendix 12, the corresponding formulas for the DD reproductive function are

$$b_R = \frac{1}{\ln(N_R^{'}) - \ln(K)} \ln\left[\frac{S_{t-1}(d_R - R')}{S_{t-1}(1+d_R)-1}\right] \quad \text{and} \quad a_R = N_R^{'}\left(\frac{d_R}{d_R - R'}\right)^{1/b_R},$$

and for the DD survival function are

$$b_s = \frac{1}{\ln(N_S^{'}) - \ln(K)} \ln\left[\frac{(1+R_t)(d_s - S')}{d_s(1+R_t)-1}\right] \quad \text{and} \quad a_s = N_s^{'}\left(\frac{d_s}{d_s - S'}\right)^{1/b_s}.$$

Formulas for other DD functions examined in this chapter (θ–Ricker function, MS&S function, and Usher function) are derived in Appendix 12.

In this way, in all IDD models, the density dependent curve for each vital rate is re-specified once at each time step. Even though, for a given vital rate, they were all calculated using a single DD function, this annual re-calculation ensures that the relationships between the realised vital rates and population size do not follow the function that was used as the basis for the calculation (or any other specific DD function).

8.4.4 Simulation

According to the above parameter re-calculation, we need one vital rate from the previous year to calculate parameters for a given year. Prebreeding models require S_{t-1} while postbreeding models require R_{t-1} to estimate parameters for the year *t*. This causes a problem in the first year of a simulation because there is no vital rate available from the previous year. As an approximation we can use one of the known (i.e., specified) values of vital rate (i.e., d_S in place of S_{t-1} in prebreeding models, and d_R in place of R_{t-1} in postbreeding models). This approximation is reasonable if the initial population size is very small (as is the case in the simulation described next). And even if the initial population is not small, the effect of using a different vital rate

for the first year can be assumed to be negligible, especially if the duration of the projection is significantly large[10].

Using the above approximation for the first year, I simulated a hypothetical population under all four DD functions examined in this chapter. The population model used was unstructured, demographic, and can be given by the dynamic equation $N_{t+1} = N_t S_t (1 + R_t)$, where S_t and R_t are the survival and reproductive rate realised from their respective density dependent functions. For easy comparison I used the same input values for all the functions (d_s=0.8, $N_S^{'}$ = 500, S'=0.75, K=1000, d_R=0.4, $N_R^{'}$ = 500, R'=0.35). With these values, the simulated populations are growing. Therefore I started with a smallest possible population size of N_0 = 1, and projected the population until it reached a stationary level. That allows us to see the maximum possible range of population sizes (i.e., from smallest to K). (A trajectory of a population starting with a single individual is biologically meaningful if the population model refers only to females as is the case in the majority of marine mammal models).

In IDD models, both DD curves are re-calculated at each time step. Because of this, the resultant density dependent relationship (i.e., the relationship between the realised vital rates and corresponding population sizes) cannot be expected to be the same as that of the function used to estimate them. By having a linear relationship, the logistic function provides an excellent example to see any deviation of the shape of the relationship between realised vital rates and population density from the shape of the function used in the simulation. Therefore, I simulated the population under the logistic function as well. Unlike the other functions considered in this chapter, the logistic function has only two parameters, and so it requires less input information. Therefore the parameters for this function were specified at each time step using only d_S, d_R, and K (see details in appendix 12).

[10] Also, for the first year, we can use S' in place of S_{t-1} in the prebreeding models, and R' in place of R_{t-1} in the postbreeding models. This is more reasonable when the initial population is closer to $N_S^{'}$ in prebreeding models, or $N_R^{'}$ in postbreeding models. Some declining populations fall in to this group. However, I observed that the resultant trajectories are almost insensitive to this choice.

All the above simulations were for IDD-DDSR models where both vital rates were density dependent, so parameters for those curves had to be re-calculated at each time step. I also simulated DDS and DDR models under all four of the three-parameter DD functions (i.e., excluding the logistic function). In these models one of the vital rates is density independent, and so a fixed DD curve can be estimated for the other vital rate. These are in fact no longer IDD models as there is no pair of interacting vital rates, and no parameter re-calculation at each time step, instead they use a fixed DD curve throughout the simulation. The purpose of this simulation was to see the similarity of their outcomes with those of their corresponding IDD counterparts in IDD-DDSR models.

8.4.5 Simulation results

(a) Trajectories under different functions

Under each DD function, trajectories were indistinguishable between postbreeding and prebreeding models (see discussion on this in section 8.4.6.f), hence I present only the postbreeding trajectories here. The trajectories shown in Figure 8.8 shows that, with the input information used in the simulation, smaller populations increase and asymptotically reach K under all functions. This result essentially shows that DD can be modelled under any of these DD functions even if the input information is insufficient to derive individual DD curves separately for each vital rate.

At the start of each simulation we specified a DD function that determines the relationship between vital rates and the corresponding population density. Due to the re-calculation of function parameters at each time step, the relationships between realised vital rates and corresponding population densities under IDD-DDSR models are not expected to follow the above specified DD function. However, these realised relationships can also be treated as DD functions as per the definition of DD function given in section 6.2. Therefore, I distinguish these two forms of relationships by using the term 'specified DD relationship' to refer to the DD function specified for the vital rate at the start of the simulation, and 'realised DD relationship' to refer to the empirical relationship between the realised vital rate and the corresponding population density.

As shown in Figure 8.8, the population trajectories under the θ-logistic, θ-Ricker, and MS&S functions were indistinguishable, while the trajectory under the Usher function is quite different. Also the realised (empirical) DD relationships are indistinguishable under the first three functions, but are quite different under the Usher function.

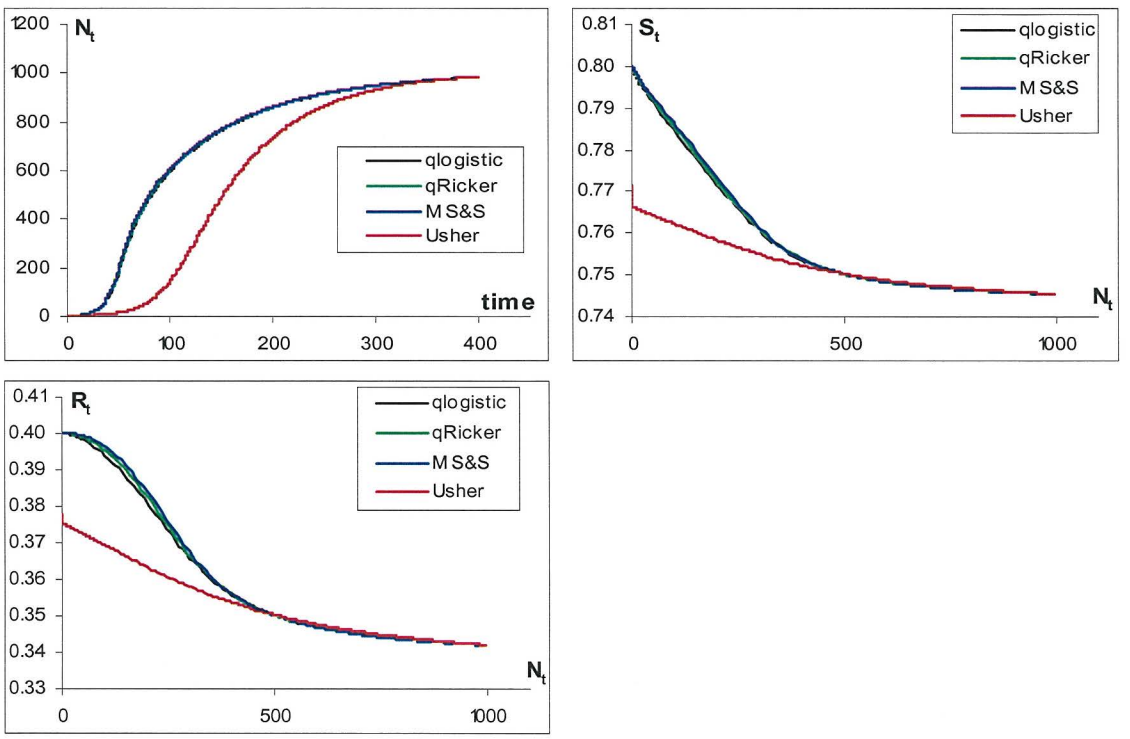


Figure 8.8: An example of the population trajectories from each DD function under the IDD-DDSR model. The input parameters used are d_s=0.8, $N_S^{'}$= 500, S'=0.75, K=1000, d_R=0.4, $N_R^{'}$= 500, R'=0.35. The top left panel shows the population trajectories (N_t) for a population starting with one individual (N_0=1). The trajectories under the θ-logistic, θ-Ricker, and MS&S functions are very similar and not clearly distinguishable, while the trajectory under the Usher function is different from the others. The other two panels show the relationship of realised values of survival rate and reproductive rate to the corresponding population sizes.

Figure 8.8 also shows that vital rates realised under all the DD functions (including the Usher function) become similar when the population size exceeded about K/2. However, the trajectory under the Usher function remains different for a longer period because it takes longer to build up the initially slowly growing population. This can be seen more clearly from Figure 8.9 which tracks the level of realised vital rates against time for each function. As expected by negative density dependence, vital rates become smaller with increasing time (i.e., with increasing population). The θ-

logistic, θ-Ricker, and MS&S functions have identical relationships, but differ from that of the Usher function. In initial years (i.e., at lower densities), vital rates are much lower under the Usher function than the other functions. The opposite is true for later years, and finally they all become similar as the population approaches K.

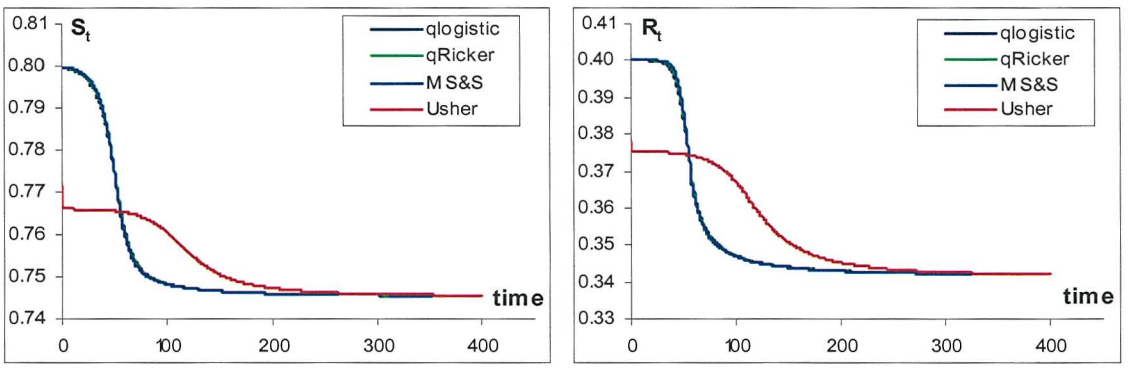


Figure 8.9: Variation of realised vital rates with time. The relationships are indistinguishable in the θ-logistic, θ-Ricker, and MS&S functions, but are different in the Usher function. They all become similar at later years when the population size stabilises at a stationary level.

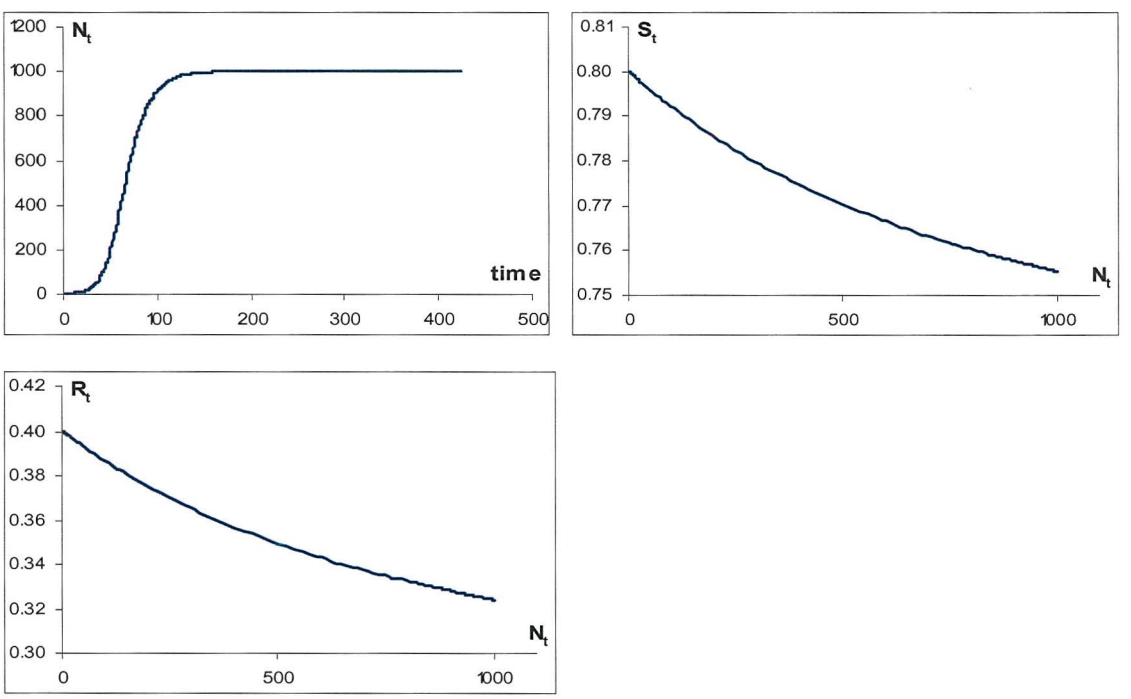


Figure 8.10: An example of a population trajectory from the logistic function under the IDD-DDSR model. The input parameters used are d_s=0.8, d_R=0.4, and K=1000. The left panel shows the population trajectory (N_t) for a population starting with one individual (N_0=1). The other two panels show the relationship of realised values of survival rate and reproductive rate to the corresponding population sizes.

The population trajectories resulting from the logistic function are not comparable to those resulting from the other functions because less input information is used in the logistic function, and hence they are shown separately in Figure 8.10. As expected, this Figure shows that the realised DD relationships are not linear even though they were realised under the linearly shaped logistic functions.

(b) DDS and DDR models

DDS and DDR models represent the density dependence through a single vital rate, assuming that the other vital rate is density independent. Therefore these models do not require all the inputs required by DDSR models. More specifically, DDS models do not require inputs that carry information about density dependence in reproduction (i.e., d_R , R', and $N_R^{'}$); instead they require a density independent reproductive rate. Therefore I used a reproductive rate of 0.35 (which is approximately the mean of realised reproductive rates in DDSR models over the projection period). Similarly, DDR models do not require d_S, S', and $N_S^{'}$; instead I used a density independent survival rate of 0.75 (which is approximately the mean survival rate realised in DDSR models over the projection period). Apart from that, the input parameters used in the DDS and DDR models were the same as in DDSR models. Because of differences in the input information used in the DDS, DDR, and DDSR models, the predictions from those three models are not exactly comparable. However, model projections from DDS and DDR models were observed to be consistent with those from DDSR models. Specifically, the θ-logistic, θ-Ricker, and MS&S functions produce indistinguishable results in terms of both population trajectories, and the realised density dependent relationships (Figure 8.11). Also the outcomes of the Usher function are different to those of the other functions in a consistent way to DDSR models.

Unlike in DDSR models, we need to estimate only one DD curve in a DDS or DDR model. Input information is sufficient to estimate that curve, therefore those models use a fixed DD curve throughout the projection period (i.e. they do not belong to the group of IDD models). For that reason the realised DD relationships shown in two bottom panels of Figure 8.11 become exactly the same as specified DD functions.

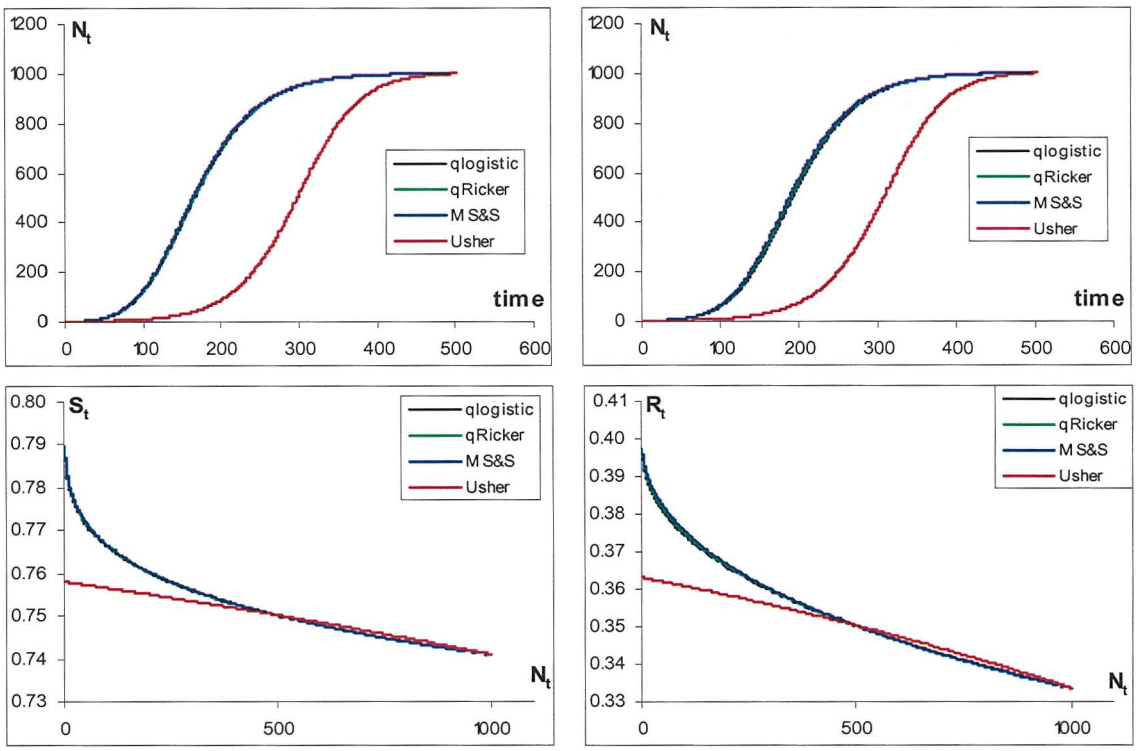


Figure 8.11: An example of population trajectories from DD functions under the DDS and DDR models. The top left panel shows the population trajectories from the DDS model with the input parameters d_s=0.8, $N_S^{'}$ = 500, S'=0.75, K=1000, and R=0.35. The realised survival rates from this DDS model are shown in the bottom left panel. The top right panel shows the population trajectories from the DDR model with input parameters d_R=0.4, $N_R^{'}$ = 500, R'=0.35, K=1000, and S=0.75. The bottom right panel shows the realised reproductive rates from this DDR model. All simulations started with one individual (N_0=1). The trajectories under the θ-logistic, θ-Ricker, and MS&S functions are very similar and are not clearly distinguishable. This is true for the both DDS and DDR models. The trajectory under the Usher function is different from the others.

8.4.6 Discussion of IDD models

(a) The advantages and applicability of IDD models

Modelling density dependence is simpler if separate DD curves can be specified for each vital rate. This requires choosing a DD function for each vital rate, estimating its parameters, and using estimated DD curve to determine the time-specific level of the vital rate at each time step of the projection. I used that approach in section 8.3 when comparing the effect of various DD functions on model predictions with the exception that I specified DD parameters rather than estimating them from data. I have demonstrated that, within certain limitations stated later, the IDD model can be used

to model DD even in situations where the available information is insufficient to estimate the individual DD curves.

In order to estimate a DD function, information on vital rate and corresponding population density covering a wide range of population sizes are required. The non-availability of such data is a hindrance even to the detection the existence of density dependence in populations, yet alone to the estimation of its functional form. Suppose that a second vital rate has also varied during this estimation period. Then the estimated DD function from a univariate analysis is valid only if the second vital rate follows the same path. Since data availability becomes an obstacle to the estimation of the DD function even in univariate cases, it is seldom possible to use a multivariate approach to estimate a DD function by considering the changes in other vital rates. Therefore the validity of any simulation based on the estimated DD curve is questionable if the variation in the second vital rate deviates from its previous path.

In IDD models, the resultant relationships between vital rates and population density are different from the relationships specified by the chosen DD functions (i.e. the realised DD relationships are different from specified DD relationships). Even though the specified DD function is used throughout the projection period, the parameters of that function are re-calculated at each time step, and so there is no single DD curve that its corresponding vital rate follows throughout. This is seen clearly with the logistic function as the resultant vital rates showed a non-linear relationship to the population density.

One might see this as an undesirable feature of this method, but it does have an advantage when we have no reason to believe that the DD relationship follows a specific function (i.e., when we are unsure which DD function to choose). In section 8.3 we observed a considerable difference in population outcomes between DD functions, even when the same strength of DD was simulated under each function. But as seen from Figure 8.8, the results of IDD-DDSR models are less likely to depend on the chosen DD function. Therefore, in IDD models, the burden of selecting one DD function over another can be taken lightly. This relative robustness of population outcome to chosen DD function is achieved through the re-calculation

process, which at each time step attempts to direct the population trajectory towards K irrespective of the actual DD function used.

Unlike in the simulations discussed in section 8.3, in the present simulation we do not assume the vital rates to have the same strength of DD under all functions even though we used the same input information. That is because, in the case of a given vital rate, the input information explicitly specifies two points on the DD curve, and a third point is derived using a function-specific formula, hence it is function-dependent. For example, in the case of survival rate, curves under different functions have two points in common, $(0, d_S)$ and $(N_S^{'}, S^{'})$. The other input information used to determine each DD curve is that K should be the same under all functions. Note that this does not guarantee that DD curves under different functions meet at a third common point. Therefore, there are no three points on DD curves that are common to all functions, and thus there is no reason to believe that these curves refer to the same level of DD in the sense of the 'same level of DD' defined in section 8.2. Therefore one could expect to see different trajectories under each function. However, trajectories under the θ-logistic, θ-Ricker, and MS&S functions were found to be indistinguishable. Accordingly, the realised DD relationships were also indistinguishable in these three functions. The only exception observed was the Usher function.

(b) An explanation for different results under the Usher function

Even though the outcomes of IDD models are fairly robust to the choice of DD function, the Usher function was found to produce a considerably different outcome to others. The reason for this discrepancy is obvious from the markedly different shape of this function at low densities. At very low densities the Usher function produces smaller vital rates than do the other functions. For example, survival rate approaches $\frac{d_S}{1+\exp(-a_S)}$ under the Usher function when N approaches zero, whereas it is d_S from the other functions. The two bottom panels of Figure 8.11 can be used to see the magnitude of this difference, which show the realised DD relationships from the DDS and DDR models. Because they are not IDD-DDSR type models, those relationships should be the same as the specified DD relationships for their respective

vital rates. Therefore, those realised DD relationships are same as the fixed DD relationships specified by the input information. The magnitude of the difference in functional shape in the Usher function is quite clear from this Figure.

The realisation of lower vital rates at lower densities under the Usher function is consistent with trajectories taking a longer time to reach K. Having considered the effect of using the quite differently-shaped Usher function on the model predictions as an extreme case, we can say that the IDD model predictions are fairly robust to the choice of DD function. This is an advantage of IDD models.

Because no DD curves are re-calculated over the period in DDS and DDR models they are not really members of IDD models. DDS, DDR, and DDSR models require different input information, and so the results are not necessarily comparable. However, Figures 8.8 and 8.11 show that the population outcomes follow the same general pattern for the three versions (i.e., they are qualitatively similar even though quantitatively different).

(c) Usefulness and applicability of the IDD model

In IDD models all population trajectories are tending towards the specified K. (In DDS and DDR models, which are not IDD models, this is achieved by using K as one of the inputs required to specify parameters for the DD curve which the model is built upon. In DDSR models this happens through the DD curve re-calculation process at each time step). This seems unrealistic because it is unlikely that all populations ultimately grow (or decline) towards K. In fact the purpose of most modelling exercises is to determine the ultimate fate of the population. Therefore there is a clear need of relaxation from that inflexibility.

This inflexibility can be removed from the model if we can replace the causal input information, specifically the input information of $\lambda = 1$ at $N = K$. Depending on available information about the population, we can replace this input information by another; for example, by the growth rate at a specific population size (i.e., something like $\lambda = \lambda^*$ at $N = N^*$). Then at each step of re-calculation, the population growth rate will be redirected towards that point in growth curve. In DDS and DDR models,

where no re-calculation is involved, the specified DD curve guarantees the resultant growth curves will go through that input point.

The IDD models as they were presented here, used the same DD function for both vital rates. However, this is not a requirement. It is possible to use a function for recruitment that is different from the one used for survival, and the estimation procedure given in appendix 12 can still be used.

(d) Limitations in IDD models

There are at least two limitations in the applicability of IDD models.

1. First, they are not relevant to count-based population models where density dependence in individual vital rates is not considered. They cannot be generalised to structured models because the dynamic equations of those models cannot be solved for the carrying capacity unless the model is very simple, (Caswell 2001, p518). Therefore, the usefulness of IDD models is limited mainly to demographic unstructured models.
2. Second, at a given time this model assumes one of the vital rates is constant, and attempts to adjust the DD curve of the other vital rate such that the resultant growth rate becomes unity at the population carrying capacity. If the first vital rate is very small, then the second needs to be very large to fulfil this requirement. Since the levels of both vital rates at the lowest density are specified (d_S for survival rate, and d_R for reproductive rate) the second vital rate cannot be increased beyond these limits (unless it is assumed that the density dependent relationship is positive). Therefore, there exists a minimum threshold in the vital rates below which this method cannot be used. It is possible for one of the vital rates to become very small for several reasons; e.g., the density dependent process can result in small vital rates when N is very large, or environmental conditions can make vital rates very small even if N is small. Therefore, it is useful to know this minimum threshold. Appendix 13 outlines how those thresholds can be found. The appendix assumes the input information used in the model is the same as was explained in section 8.4.2 (e.g., without the suggested input of $\lambda = \lambda^*$ at $N = N^*$ in section 8.4.6.c).

However, it can still be adapted to suit the actual input information used in the model.

(e) Population size (N_t) in density dependent demographic models

In this exercise we treated the annual cycle as being composed of two discrete events; the survival of animals over the year, and their reproduction. The rate at which these events are occurring at a given time is determined by the relevant respective density dependent curve and the population size at the time of the event. Here the phrase 'population size at the time of the event' needs some clarification. In both events, we used the population size as at 'the beginning of the current year'. More specifically, the dynamic equation we used was $N_{t+1} = N_t S[N_t](1+R[N_t])$ where N_t is the population size at time t, and R[.] and S[.] are the density dependent functions for reproductive rate and survival rate respectively. (Note the different notation; so far R_t and S_t were used in place of R[.] and S[.]). The quantities inside the square brackets are the population densities applicable to those functions. It is worth noting that in this exercise, the same N_t was used for both events. On the other hand, it may be argued in favour of using the population size 'just prior to the current event'. In that case the population size at the time of second event is different from what it was at the beginning of the year. The dynamic equation then becomes

$N_{t+1} = N_t(1+R[N_t])S[N_t(1+R[N_t])]$ in prebreeding models, and

$N_{t+1} = N_t S[N_t](1+R[N_t S[N_t]])$ in postbreeding models.

Runge and Johnson (2002) used this prebreeding dynamics equation in their population model[11].

I opted to use the population density at the 'beginning of the current year' for both events, because I defined the DD functions as the relationships of the vital rates to the population size at the beginning-of-the-year (not to the population size at an intermediate time within the year). Also in discrete time models, vital rates are defined with respect to the population size at the beginning of the year; the survival rate is the number of survived animals at the end of period as a fraction of number of animals at the beginning of period, and the reproductive rate is the number of recruits

[11] Runge and Johnson (2002) model is not an IDD model.

as a fraction of number of breeders at the beginning of the year. Furthermore, when specifying DD parameters using the equilibrium constraints of $\lambda_t=1$ and $N_t=K$ we implicitly use the definition of the growth rate at time t as the ratio of N_{t+1} to N_t, where N_t is the population size at the beginning of time period. This means that we measure all rates relative to the population size at the beginning of the year[12].

(f) Predictions from prebreeding and postbreeding models

In simulations we observed (under all functions) that the trajectories are similar between prebreeding and postbreeding versions of the model. Since we used the same dynamic equation $N_{t+1} = N_t S[N_t]\{1+R[N_t]\}$ under both versions of the model by swapping the sequence of two events within the year, at first sight it seems that our observation is theoretically justifiable. However, the DD relationships used for a given vital rate are not the same in the prebreeding and postbreeding models even if they were based on the same function. This is because the re-calculation of DD functions at every event takes account into the population changes that occurred at the previous event. Therefore the components S[.] and R[.] in the dynamic equation are different in the two models. For example, the population dynamic equation under the θlogistic function is $N_{t+1} = N_t\left[1+d_R\left\{1-\left(\frac{N_t}{a_{R,1}}\right)^{b_{R,1}}\right\}\right]d_S\left\{1-\left(\frac{N_t}{a_{S,2}}\right)^{b_{S,2}}\right\}$ for the prebreeding model, while it is $N_{t+1} = N_t\left[1+d_R\left\{1-\left(\frac{N_t}{a_{R,2}}\right)^{b_{R,2}}\right\}\right]d_S\left\{1-\left(\frac{N_t}{a_{S,1}}\right)^{b_{S,1}}\right\}$ for the postbreeding model, where the second subscript in the density dependent parameters refers to the place of the event in the sequence of events. Formulae for those density dependent parameters are given in appendix 12. Therefore, the two dynamic equations are not mathematically the same, and they do not analytically explain the observed similarity in predictions of two types of models.

[12] As an experiment, I changed the population dynamic equation in prebreeding model to $N_{t+1} = N_t(1+R[N_t])S[N_t(1+R[N_t])]$, and in postbreeding model to $N_{t+1} = N_t S[N_t](1+R[N_t S[N_t]])$. The resultant trajectories were very similar to those described in this exercise. But the relationships of the resultant vital rates to the corresponding population densities were unacceptable shaped.

However, the similarity of trajectories between prebreeding and postbreeding models can intuitively be explained in the following way. At every event, one of the vital rates (say the survival rate) is adjusted such that the population trajectory becomes stabilised at K if the other vital rate (i.e., the reproduction rate) remains unchanged at the level immediately prior to the present event. At the next event, reproduction rate is modified to direct the trajectory to the same K, assuming the survival rate remains unchanged. This cycle repeats itself throughout the projection period. Although the actual functions used in those adjustments are not the same between prebreeding and postbreeding models, they both direct the trajectory in the same direction at every event. Since this re-direction occurs frequently (twice a year) the resultant trajectories cannot be very different, even if they are not identical. That could explain why the trajectories of the prebreeding and postbreeding models are indistinguishable. Therefore, even though the dynamic equations used in prebreeding and postbreeding versions are mathematically different, they do the same thing to the population.

Survival and reproduction are not instantaneous events within a year as assumed by this model, instead they are more likely to be continuous processes. Therefore the interaction between them is also a continuous-time process. These continuous processes are approximated by discrete events in discrete-time models. The competing risk model of Siler (1979), which is further described in Elandt-Johnson and Johnson (1980), discuss models where more than one sources of mortality are competing each other. In his dynamical model, Lebreton (2005) approximated these continuous events by a product of individual discrete events. He showed that, the effect of two competing continuous sources of density dependent mortality can be approximated by the product of two survival rates due to the individual sources of mortality calculated at the beginning of the year. For example, if the two sources of mortality are natural mortality and harvest mortality, the overall survival rate (S) can be approximated by $S \approx S_0(1-h)$, where S_0 is the natural survival rate in the absent of harvest mortality, and (1-h) is the harvest survival rate in the absence of natural mortality, both calculated on the basis of the beginning-of-the-year population size. The approximation is closer when each individual survival rate is large. (The approximation is exact when one of the survival rates is one, i.e., when that source of mortality is absent).

Both the competing risk model and Lebreton's approximation deal with components of mortality only, not with any other sources of population variation. Considering various forms of mortalities as sources of population variation, a similar analogy may be applicable when mortality and reproduction are the sources of population variation. Taking this analogy, one can hypothesize that when there are density dependent survival rate and density dependent reproductive rate acting simultaneously (dynamically over the year in continuous time) the resultant effect on the population is approximately determined by those two individual rates as determined by at the beginning-of-the-year population size. This is consistent with my choice of selecting the dynamic equation as $N_{t+1} = N_t S[N_t](1 + R[N_t])$ where both survival rate and reproductive rate are determined by their respective DD function based on the population size at the beginning of the year.

8.5 Conclusion

In the first part of this chapter we investigated the effect of the choice of the DD function on the model predictions. There we compared the predictions from a population model that used various DD functions. Results from different functions are not comparable if they do not correspond to the same level of DD, and in a strict sense, different functions cannot represent the same level of DD. We defined the 'same level of DD' in a way that would ensure that the different functions represent the DD at approximately the same level.

Population outcomes are found to be dependent on the DD function used, even if all functions include the 'same strength' of density dependence. Even minor differences between DD functions were shown to have large effects on population predictions. Resultant trajectories could go to different levels of K, at different speeds, both in growing and declining populations. Therefore the choice of DD function has a considerable influence on population predictions.

The effect of strength of density dependence on population predictions was investigated. Perturbed populations were observed to return to K more quickly when

the strength of the underlying DD mechanism stronger. Therefore the strength of the DD can be seen as a 'resistance to change' or an 'inclination to return to equilibrium when perturbed'. However, we cannot rule out the possibility of populations being subject to limit cycles or chaos when the density dependence is very strong, although we did not observe this in this study.

Population outcomes appear more sensitive to density dependent changes in survival rate than in reproductive rate at all levels of density dependence. This observation is consistent with the generally accepted view that elasticity of population growth with respect to survival is larger than it is relative to reproduction for long-living animals.

In the second part of the chapter I presented the *Interactive Density Dependent model* which can be used to model density dependence in some situations in more than one vital rate. The IDD model is usable when the DD relationship in one vital rate is affected by the level of another vital rate. It is also usable when there is not sufficient information in the data to estimate individual DD functions for each vital rate separately, but when we instead have an estimate of the population growth rate at a specific level of density (or when comfortable with specifying such a growth rate).

A major advantage of IDD models is that their population predictions are relatively insensitive to the choice of DD function. Consequently, the realised relationships between vital rates and density are not the same as that of the function used to build the model. The realised relationship is relatively insignificant to the function used. This is useful in cases where there is no reason to believe one DD function is better than another.

However, there are limitations in the use of IDD models. First, they are not relevant to count-based models or to any other model where DD is present only in a single vital rate, and they cannot be used with structured models unless the structure is very simple. Therefore, their application is mainly limited to demographic unstructured models. Second, IDD models cannot be used with negative DD if the vital rates fall to very small levels. And third, the IDD model concept is applicable only for birth pulse populations where birth and death processes can assumed to be discrete events.

Chapter 9 Summary and Further Research

Globally, a vast number of animal species are subjected to the influence of human activities. In many such situations the underlying 'natural' dynamics of the population is being modified even if the populations are not harvested or otherwise manipulated directly. The management of such populations relies on successfully understanding their dynamics, and field experiments and population modelling play key roles in this understanding. Every population dynamics model attempts to represent nature in a simple but realistic manner. Not all population models represent all the characteristics of populations to the same extent or in the same manner. Therefore different models can produce different outputs (i.e., possible fates of the population) and can lead to different management decisions. Inappropriate decisions can lead endangered or threatened populations into unrecoverable states. Therefore population characteristics need to be represented in models as realistically as is practical.

This study focussed on incorporating population characteristics, and in particular environmental stochasticity and density dependence, into population dynamics models. The goal of this study was to review the present ways of achieving this, and to recommend improvements. In this chapter I summarise the main findings of the previous chapters.

Any population model, irrespective of how carefully constructed it, is always an approximate depiction of nature. In the majority of cases for which predictions are required, it is not possible to model every element that can be comprehensively justified on the basis of a proven mechanism. Rather, the process of model construction is an attempt to arrive at a parsimonious and robust characterisation of the system under study. Model predictions may display extreme sensitivity to

apparently minor changes in model specifications, and/or great sensitivity to imprecision in input parameters (Wood and Thomas 1999). Therefore newly built models should be subjected to model assessment (e.g., verification, validation, uncertainly analysis, sensitivity analysis).

One of the fundamental problems conservation biologists face when they provide advice to managers is how to characterise and account for the uncertainties which result from measurement errors and model mis-specification. Model assessment is especially critical when apparently realistic models vary in their management implications (Ragen and Fowler 1992). For example, the risk of population decline has been found to be very sensitive to the distribution of individuals among age-classes (Burgman et al. 1995), groupings of data (Eberhardt 1981), and to the precision of vital rates estimates (McCarthy et al. 1995). Failure to adequately account for this uncertainty can have drastic consequences (e.g., Ludwig et al. 1993, Wood and Thomas 1999). Recognition of this is apparent from the literature of all areas of modelling in last decade (Harwood 2000). More attention has been given in the literature to parameter uncertainty than to model uncertainty, although Sainsbury (1988) and Bartholow (1996) are some exceptions. Even though the uncertainty affects the outcomes of a population model, it does not invalidate the use of an uncertain model or uncertain inputs in an assessment like population viability analysis (Starfield 1997, Newman et al. 2006). The correct representation of uncertainty, literally as a probability distribution of outcomes, is exactly what is needed to inform management (Goodman 2002).

9.1 Environmental stochasticity

Ignoring environmental stochasticity is likely to overestimate the precision of population predictions (Engen et al. 2001), and population persistence (Soulé 1987, Caswell 2001), while underestimating the risk of extinction (Burgman et al. 1993), even if demographic variation and uncertainty about the demographic rates have been taken into account. Therefore any model that does not take environmental stochasticity into account is unlikely to predict the population status realistically. The

first species specific population model proposed for the NZSL management (i.e., Hilborn et al. 1997) did not have these stochastic components. Managing a population, especially a threatened or endangered population, with a deterministic model (or even with a model with demographic stochasticity) is very risky as they are known to underestimate the risk. Acceptance of this among the modelling community is evident from the fact that population dynamics models in recent literature are almost entirely stochastic.

The subsequent species specific models (i.e., Hilborn and Wade 1999, Breen et al. 2003) proposed for this population uses the lognormalpower distribution to represent the environmental stochasticity. To my knowledge this is the first appearance of this distribution in the area of modelling stochasticity. This raised a question 'does it matter which distribution is used?' This question motivated me to investigate possible ways of determining the suitability of a distribution, which led to the work presented in chapters 4 and 5.

Although I presented the NZSL population as a motivating example to discuss generic issues in modelling, the simulation results obtained in the thesis cannot be deemed to represent the status of the NZSL due to several reasons. First, I haven't assessed any of my models used in simulations against known information of the NZSL as per section 3.5. Second, I deliberately did not include some important population characteristics in the simulations (i.e., stochasticity in simulations in chapter 8, density dependence in simulations in chapter 5, bycatch in any of the chapters). It is generally accepted that extinction probabilities reported from a density independent model are generally overestimations while those from deterministic models are generally underestimations (Morris and Doak 2002, p101). Third, the simulations do not include dispersal and no assessment was done to see the effect of that. Fourth, use of parameter values as described in section 5.2 can result in using improbable parameter combinations. Therefore the simulation results cannot be interpreted as predictions for the NZSL population.

In this thesis I also reviewed the various approaches in use to represent environmental stochasticity in population models, and then focussed on the most common approach where environmental variability is represented by treating demographic rates as iid

random variates realised from statistical distributions. The thesis focussed on the properties of those distributions that are relevant in evaluating their suitability. On that basis I first developed a criterion to compare distributions based on:

- shape of the distribution,
- ability to simulate variability as intended,
- ability to specify parameters for the distribution using available information, including reliability of approximation methods such as Delta method,
- reliability of parameter specification,
- ability to generate vital rates from the distribution,
- ability to use all sensible combinations of mean and variability of the vital rate.

Then the criterion was demonstrated by assessing the suitability of a sample of selected distributions.

In order to represent the stochastic variation in models, variation due to stochasticity needs to be known. Available information on this is usually subjected to uncertainty. Several approaches have been proposed as steps towards in separating uncertainty from stochasticity in order to account them separately in models (e.g., Burnham et al. 1987, Gould and Nichols 1998, Wade 1999, Akcakaya 2002, Barker et al. 2002). One has to employ such an approach to separate stochastic variability from total observed variability before using them in a stochastic modelling. Failing to do so will overestimate the effect of stochasticity. This is also another reason that simulations in chapter 5 should not be taken as predictions of the NZSL population's fate. However, they still serve the purpose of the thesis, which is to compare the distributions.

Chapters 4 and 5 focussed on the ability of different distributions to express stochastic variability as intended. It is possible to argue that an exact representation of stochastic variability is not very useful because a precise estimate of stochastic variance is unlikely to exist. However, the study is in no way subject to the assumption that the variability observed in field data represents the stochastic variability free of uncertainty. What it does mean is that the study presents a way to distinguish distributions that can represent variability as intended from those that cannot. Undoubtedly a distribution that can represent this variability precisely is better suited than any other distribution that cannot, irrespective of whether we have a precise variance estimate or not.

The normal distribution is probably the most commonly used distribution to represent the stochastic variation. There are advantages of using it; it is simple to use, information that is usually available (i.e. mean and standard deviation of stochastic variation) can be used directly as parameters for the distribution, it is possible to generate random variates easily and accurately in almost any modelling software, it is possible to simulate correlated vital rates (e.g., Burgman et al. 1993, p280), and analytical solutions are easy to obtain (e.g., if the variability is normal in each age-group then the total variability is also normal). However, truncation is often required to confine simulated variates to the theoretically possible range. Problems with this truncation are listed in section 4.2.1.

Some of those problems can be avoided by directly generating random variates from a truncated normal distribution. Even though a general method for generating truncated variates from any continuous distribution has been available for last three decades (e.g., Fishman 1973, p228) its use is problematic in practice, and to my knowledge no one has used this method in population modelling. One might suggest handling this problem by starting with a different mean and standard deviation for the original normal distribution (say, μ and σ) so that the truncated distribution will have the required mean and standard deviation (m and s). This method requires specifying μ and σ in terms of required m and s, which is not easy. Cohen (1949, 1950) showed a method of estimating parameters for the truncated normal distribution, and Cohen (1951) generalised it to a series of other distributions. However this method needs a set of sample data (i.e., observations) from that distribution (knowing m and s is insufficient), and the method is not straightforward. To see the magnitude of the possible effect of truncation, I used parameter values used in the NZSL example to generate variates from truncated normal distribution and estimated that the mean and variance of the variates generated can be different from what were intended by an amount of between 12% and 15%. Caswell (2001, p412) gives an extreme example where mean and variance of data generated from truncated distributions differ from the intended mean and variance by 25% and 35% respectively. Apart from this numerical difficulty, the truncated normal distribution can take only a specific shape, so it can be used only as far as that specific shape is applicable (e.g., it cannot produce

a U shape). Despite these limitations the truncated normal distribution is still in use (Penteriani et al. 2005). It is conceivable that in some situations the use of truncated distributions is biologically justifiable to represent the environmental stochasticity if good enough data exist to estimate their parameters. However, the availability of such data is uncommon.

The choice of the distribution to represent the environmental variation seems subjective, often based on previous practice, convenience or experiences with other species, and it is not justified in the majority of models. Modelers rarely check whether the variation introduced by the chosen distribution represents the intended variation. The reduction in standard deviation caused by truncation will translate to an underestimation of the possible variation in population outcomes, leading to an underestimation of extinction risk in population viability analyses. However, as noted by Todd and Ng (2001), such effects are rarely documented. It is not clear whether the modeling community is aware of these problems (i.e., inability to achieve the required mean and standard deviation, and inflexibility in the shape of the distributions) or not, because it is difficult to find any discussion of this issue.

Another widely used distribution, especially to represent the skewed variability often observed in reproductive rates and population size, is the lognormal distribution. Standard algorithms to generate variates from the lognormal distribution are available (e.g., Burgman et al. 1993, p279, Wade 1998, Wade and DeMaster 1999). These algorithms require the mean and variance of the log of the vital rate (μ and σ^2) to be specified in terms of the mean and standard deviation of the vital rate (m and s), which can be done analytically, unlike in some other distributions. However, since the right tail usually goes beyond the range of the vital rate, modellers tend to use it as a truncated distribution (e.g., Burgman and Lamont 1992), and so it still shares the problems associated with the truncated normal distributions.

One of the obvious ways of getting around with the problems associated with truncation is to transform variates in a suitable manner. This provides the basis for the other three distributions used in this thesis. The inverse logit transformation to normal variates leads to the logitnormal distribution. Since there is no analytical way of

specifying the parameters of this distribution (Aitchison and Shen 1980, Frederic and Lad 2005), parameter specification is only approximate. The probit transformation produces the probitnormal distribution. Todd and Ng (2001) recommended this distribution for survival rates because of its ability to generate correlated variates, and its flexibility in shape. Use of the lognormalpower transformation produces the lognormalpower distribution which was used in the latest NZSL model (Breen and Kim 2005b).

Before using any distribution to represent stochastic variation, its parameters need to be specified in terms of known information about the variability. This is problematic for some distributions (see table 4.1), and the most common approach to avoid this difficulty in practice is to use an approximation. For example, in modelling the NZSL population, Hilborn and Wade (1999) and Breen et al (2003) used the delta method approximation to specify the parameters for the lognormalpower distribution. The present study shows that this approximation does not represent the variability in data with sufficient accuracy (see Figure 4.4), especially when the mean is away from 0.5 or the variance is away from zero. The magnitude of its possible effect on the model outcome can be seen from the elasticity, which measures the possible percentage change in the growth rate caused by one percent change in vital rate. The elasticity of the growth rate to the vital rates is found to be large (section 5.5). This elasticity can be considered as an indirect measure of the influence on the model outcome of the bias caused by representing (mean) stochasticity by the distribution in question. The large elasticity observed in Figure 5.5 implies that the population growth rate estimated for the NZSL population using the Breen et al (2003) model could have been biased. Although it is impossible to comment on the possible direction of this bias, it may be relevant to recall that Breen et al (2003) predicted a maximum possible growth rate of 3% for this population although it is generally believed to be around 8% to 12% for similar species (Wade 1998). (This is not intended to suggest a magnitude for the bias caused by the delta method approximation. Any bias in a growth rate predicted by a population model is a combined result due to all sources of biases. For example, in section 5.3 I demonstrated the possibility of overestimating the NZSL growth rate by using the lognormalpower distribution in the Breen et al, 2003, model).

The lognormal distribution is commonly used to represent positively skewed stochastic variations such as in reproductive rate and population size (Nakaoka 1997, Higgins et al. 2000, Hilborn et al. 2000, Caswell 2001). In theory, this distribution can be reparameterised to have a negatively skewed shape (Johnson et al. 1994b, p207). The distributions of reproductive and survival rates of NZSL population are either estimated or believed to be negatively skew, with the possible exception of the survival rate for the pups (Chilvers 2005). Therefore it is unlikely that this distribution is suitable for modelling this population unless used it as a reparameterised version. In case of the NZSL population, the lognormal distribution has not been used to represent the environmental stochasticity (even though it has been used to represent parameter uncertainty in the PBR formula).

The lognormalpower distribution has only recently been used in representing environmental stochasticity. Hilborn and Wade (1999) proposed this distribution to represent the environmental variability of survival rate of the NZSL population. Although this particular population model was not used for managing the NZSL, Breen (2003) used the same distribution to develop a slightly different model for that population. This new model is presently under consideration for managing the NZSL, and its use of this lognormalpower distribution motivated the present investigation on the suitability of various distributions in representing environmental stochasticity. I found several problems with this distribution, namely, the possibility of having an undesirable shape, difficulty in specifying parameters, inappropriateness of the delta method approximation, and the growth rate being sensitive to bias in parameter specification.

The shapes of the beta, logitnormal and probitnormal distributions are very similar for most combinations of m and s used in my simulations. Although the shape of the lognormalpower distribution (the distribution used for the NZSL model) is similar to other distributions when s is small, it has a prominent peak near zero at moderate to large levels of s. This peak is observable for some combinations of m and s even when m is far from the boundaries, and when other distributions take a shape that is very similar to the uniform distribution. Also the lognormalpower distribution is bimodal when others are unimodel, having a peak near the lower boundary even at

smaller levels of *s* such as 0.1 (see Figure 5.7). Therefore we should be cautious when using such a shaped distribution to represent environmental variation.

The lognormalpower distribution was observed to produce population predictions that were different from other distributions. When the mean and variability (*m* and *s*) are large, the growth rate predicted by this distribution is larger than what is predicted by other distributions (i.e., very close to what is predicted by the deterministic model as seen from figure 5.3A and B). The reason for this is that the survival rates generated from this distribution become larger and closer to the upper boundary than those generated from other distributions at this combination of *m* and *s*. In the NZSL population adults constitutes a large proportion (Chilvers et al. 2005), while a large *m* is appropriate for adult NZSL (Wilkinson et al. 2005). This implies this distribution is likely to overestimate the growth rate of NZSL, and consequently tends to underestimate the extinction risk in highly variable environments as observed in Figure 5.4. Although my simulations in motivating example do not really reflect the nature of the NZSL population, we can hypothesize that the fate of the NZSL population predicted by the proposed management model may have been biased due to this effect.

Using the PBR formula and the NZSL population size estimation model (i.e., Gales and Fletcher 1999) Breen et al (2003) showed that the MALFiRM is proportional to the estimated population size. This implies that whenever growth rate is overestimated MALFiRM is also overestimated. Therefore, the use of the lognormalpower distribution in the proposed NZSL model tends to overestimate the possible MALFiRM.

It was observed that elasticity of the growth rate is predicted to be larger when the variability follows the lognormalpower distribution than when it follows any other distribution. This is consistent with the shapes of the distributions (e.g., Figure 5.7) as the shape of the lognormalpower distribution is more sensitive to changes in *s*. In summary, the distribution recommended in the NZSL model (i.e., the lognormalpower distribution) predicts a larger growth rate, lower extinction risk, and larger elasticity at higher levels of means and standard deviations. The interrelationships of these results are compatible, and consistent with the shapes of the corresponding pdf curves.

Table 4.3 summarises the properties of the distributions used in this thesis. For a number of reasons the beta distribution was found to be more suitable than the other distributions examined. Shape parameters for this distribution can be obtained exactly for any given sensible (as defined by appendix 1) combination of m and s (Table 4.1 and Section 4.3.3), so the variates generated using these shape parameters should have the required mean and standard deviation. Numerical methods are required to specify parameters of the other distributions examined, and so the parameter specification is not exact. Any combination of mean and variance that could theoretically exist in the possible (finite) range of the vital rate can be modelled by the beta distribution (as explained in section 4.3.3), hence it provides an automatic check against attempting to incorporate nonsensical mean-variance combinations (i.e., combinations that are theoretically impossible to exist within the range of stochastic variation) into models. This distribution allows the flexibility of fixing bounds at any range by selecting values for the a and b parameters. It can take a wide range of shapes including unimodal, J-shaped and U-shaped depending on the values of the shape parameters, which in turn depend on the mean and standard deviation (Figure 4.1). It is unimodally shaped over most (biologically realistic) combinations of mean and variance. Since the shape parameters are simple functions of m and s (section 4.2.3), it is easy to determine the shape of the distribution for any given sensible combination of mean and standard deviation. If the shape determined by the m and s combination is not the one intended, it implies either that an incompatible mean and standard deviation combination has been used or that the selected distribution is unsuitable. The shape of the distribution can be found from the input information, and so its shape can be easily checked against the intended shape (Fig 4.6). As detailed in section 4.3.7, the beta distribution is the only distribution (of those examined) where this comparison can be done analytically and easily. Implementation of the population dynamics model under this distribution is faster because it is not dependent on the convergence of a numerical procedure (section 5.6.3).

Both the logitnormal and probitnormal distributions behave in a similar way to the beta distribution in many respects. The main differences are that their parameter specification is neither analytic nor exact, and that their shapes cannot be determined in advance from the mean and standard deviation. The standard techniques that are

available to generate correlated vital rates from the normal distribution (e.g., Burgman et al. 1993) can be adapted to generate correlated vital rates from distributions that are based on the normal distribution (i.e., three of the four distributions considered in this thesis). For example, Todd and Ng (2001) detailed how those techniques can be adapted to generate cross-correlated survival rates from the probitnormal distribution. This seems like an advantage of using the lognormalpower distribution in the NZSL model even though, the model as is proposed, does not have correlated vital rates. None of these methods can be used with the beta distribution. Doak et al. (1994) used an indirect approach in generating correlated beta variates by assuming the variation in the correlated vital rates was driven by a common environmental factor, and by simplifying the correlation structure among vital rates to become individual correlations with that common factor. Gross et al. (1998) generated correlated beta variates by first generating standard normal variates with the required correlation structure, and then transforming to beta variates with the required mean and standard deviation. However, this transformation is not easy, and likely to alter the correlation coefficients severely for some combinations of means, variances and correlations (Morris and Doak 2002). To my knowledge, no method to overcome these shortcomings has been developed yet. Therefore, it is not known how to use the beta distribution (or any other distribution based on the beta distribution) to generate correlated vital rates.

The population predictions in chapter 5 lead to the conclusion that the choice of distribution does affect the population predictions even if the distributions appear appropriate. In particular, using the lognormalpower distribution could lead to quite different results. The effect of the choice of distribution is greater if the level of stochastic variability is greater or if the mean vital rate is close to one of the boundaries of its possible range. The growth rate becomes smaller under all distributions when stochasticity intensifies (Figure 5.3B). This observation is consistent with the formula for stochastic growth rate given by Tuljapurkar (1997).

The simulation in section 5.2 estimated the elasticity of the growth rate to be in the range of about 0.69 to 0.76 (averaging at about 0.73), meaning a 1% bias in adult survival rate translates to about a 0.73% bias in stochastic growth rate (Figure 5.5). Although I accept that my simulations do not necessarily reflect the status of the

NZSL population, such a high level of elasticity is consistent with the general results of Lebreton and Clobert (1993) for long-lived species. Simulations show that a change in mean survival has less effect on elasticity when the mean survival is large. Elasticity is found to be larger when the standard deviation is larger. These observations are not surprising because a stronger variability has a larger effect on growth rate if the mean level of the survival remains unchanged. The elasticity does not depend on the distribution when the level of stochasticity is small. It differs between distributions at large levels of standard deviations, with the lognormalpower distribution producing the largest elasticity. Therefore an unbiased representation of survival rate is more important for this distribution, and so approximation procedures such as the delta method are particularly unsuitable for this distribution. This shows a problem with the distribution used in the proposed NZSL management model.

In the simulations used to evaluate stochastic distributions I used a wide range of levels for the input parameters, and quasiextinction risks were calculated using different definitions of extinction. When using different combinations of parameter values it is possible to get unrealistic combinations too. I did not attempt to eliminate such combinations. In addition to the reasons given earlier, this is another reason that my simulations do not necessarily represent the fate of the NZSL population. However, the objective here was to compare the effect of using different distributions, and not to predict the fate of a given population.

Even though some observations exist to the contrary (e.g., Hess 1996), it is generally believed that models that fail to recognise dispersal properties lead to over-estimation of population extinction risk (Hill et al. 2002). However density dependence in a single population can be considered as an implicit surrogate for metapopulation structure. Starfield (1997) contrasts two views of modelling; a model as a representation of 'truth', and the model as a problem solving tool, and argues in favour of using simpler models when there is not enough data to support complex models. In that sense single population models, such as Breen et al (2003) for the NZSL which does not include dispersal, represent a suitable approach for practical management problems. This model uses a θ-logistic DD function and the lognormalpower stochastic distribution. My research did not find any serious problem

with using the θ-logistic function, but the lognormalpower distribution was found to be unsuitable.

The approach I have used in this thesis is comparable to parametric bootstrap because the variation of the vital rate is assumed to follow a specific statistical distribution, parameters for that distribution are specified, and a value of the vital rate is sampled from that distribution at each time step of the population projection. However, this is not the only possible approach. Bayesian methods provide another approach, where, although it is necessary to specify the distribution that describes the stochastic variation, the difficulty of explicitly estimating (or specifying) parameters for that distribution does not arise. In fact this approach has already been used with NZSL population models, (e.g., Hilborn and Wade 1999, Breen et al. 2003, Breen and Kim 2005a, 2005b).

9.2 Density dependence

Of primary concern in modelling density dependence are the choice of an appropriate DD function and estimation of the parameters for the selected DD function. Even if vital rates data are available over a range of densities, they can often be fitted to several differently-shaped functional forms equally well. Such fitted functions can take quite different shapes beyond the data range, and this can have important impacts on the fate of the population (Runge and Johnson 2002). This is largely due to the fact that the range of densities covered by the data is often small. Therefore, the statistical fit of a function to data is not a suitable basis for selecting one DD function over another. For example, no one believes that good enough data are currently available to identify the forms of density dependent relationships associated with the NZSL population. None of the models proposed for this population have been able to justify the choice of DD relationship used in the model. In those situations, even though some data exists, they are of little help in selecting a function. In such situations modellers are often compelled to use their own subjective judgments in deciding which function is best suited in describing the relationship. It is always useful to have an objective criterion to select a function to minimise the possible misspecification of

the functional form. In this thesis I have focused on the issue of selecting a suitable functional form and the consequences of possible misidentification. I developed a criterion that can be used to distinguish between appropriate and inappropriate functional forms when there is insufficient information in the data to estimate the relationship. This criterion uses the available biological knowledge of the species to assist with the selection of a function.

I developed an ecologically motivated criterion that can be an aid in objectively selecting a function for representing density dependence. In doing this, I first examined the desirable properties of DD functions in the contexts of both count-based and demographic models. Then I explored possible methods that can be used in testing a given function for each of those properties. In this way I developed a set of criteria to match the properties of DD functions against population characteristics. Using common functional forms, in Chapter 7 I demonstrated how that criteria can be used to assess the suitability of a given function to a population at hand. Then, in Chapter 8 I evaluated the effect of DD functions on model predictions. I then examined the possibility of modelling density dependence in a relatively function-insensitive way, and using less input information. I considered the situation in which density dependence is known to exist in more than one vital rate, but where there is not enough information in the data to estimate parameters for the DD functions for each vital rate separately. The resulting IDD model is applicable to situations where the strength of the density dependence in one vital rate is deterministically influenced by the level of DD in other vital rates.

Density independent models assume that the dynamic process is independent of the changes in population size, implying that any changes in the dynamic process can be attributed to other factors such as environmental variation. As a result density independent models will eventually predict higher sensitivity to environmental changes, and higher risk of extinction due to environmental changes (Morris and Doak 2002, p122). Therefore predictions from density independent population models could be more conservative than those that would be obtained from density dependent models. Since the opposite is true if positive DD is present, the presence of density dependence can cause density independent PVAs to either overestimate or underestimate the true viability of a population, depending on how growth rate

responds to density. Consequently, representing DD in population models in its 'real form' is important for reliable predictions.

There is no consensus on either the definition of density or the definition of density dependence. For the majority of models 'density' could be a suitably weighted combination of sub-populations (Caswell 1989, p231). It is important to understand whether density dependence is restricted to a specific age/stage classes. For example, harvesting density independent classes will not activate the density dependent population regulatory mechanisms (DeMaster 1981). Similarly, density indices derived using components of populations that do not contribute towards the density dependent mechanism could result in the model leading to erratic management decisions (Ragen and Fowler 1992). In many wild bird and mammal populations, where adults are long lived and juveniles compose a small fraction of the population, it can be reasonably assumed, at least approximately, that all density dependence is exerted by the adult population (Lande et al. 2003, p70). Use of non-pup population as the density of the NZSL in the simulations in Chapter 8 is consistent with this assertion.

Often density dependence is represented in population models by taking vital rates as functions of density. Although a statistical fit to data seems an objective criterion to select a suitable functional form, such an approach is known to be often impractical (section 6.1.4). However, once the functional form is decided, the parameters for the function are usually specified using the vital rates estimated from field studies, expert opinion, or from similar species. When vital rate estimates are obtained from field surveys it is a good idea to explore and adjust them for any identifiable bias before using in DD modelling. For example, in an ongoing study of NZSL population, animals that were marked at birth during the last several years are now being observed as breeders. Those data are being used to estimate the survival rates and reproductive rates for adults. Since it is unlikely that any of them has reached the age of senescence, observed breeding adults could be considered as a sample biased towards younger breeders, and therefore the estimated vital rates could be considered overestimates. On the other hand, because marked animals in established mark-recapture samples tend to be slightly older than the animals in general (Manly 1970, p17), the survival and reproduction estimates from those studies are likely to be

underestimated. This could be the situation with survival estimates obtained from recapturing breeding NZSL females that were branded as breeders years ago. In the simulations presented in this thesis I used vital rates estimated from more than one study without considering such possible sources of bias because the simulations were not intended to necessarily reflect the 'true' status of the NZSL.

In this thesis I developed a set of criteria to select a suitable functional form (or to eliminate unsuitable functional forms) for the population in hand by matching functional properties to population characteristics. When dealing with a population with unknown characteristics, these criteria can be used to select a function that is flexible enough to be consistent with a wide range of possible characteristics. The criterion is based on four properties of populations that have been identified previously: the shape of the function (Bellows 1981), the forms of competition that can be represented by the function (Varley et al. 1973), the ability of the function to represent known stability properties of the population (Royama 1977), and the level of the population (relative to K) when the growth becomes maximum (Pitcher and Hart 1982). The methods introduced to match populations characteristics to functional properties are summarised in Table 6.2. In this work I presented the concepts 'asymmetric growth' in the context of 'population growth' and 'stability' in the context of count-based models. However, as demonstrated in section 7.2.2, both these properties can be generalised to other vital rates and to unstructured demographic models if the DD is represented only through a single vital rate. For example, when the survival rate is represented by a DD function in a demographic model, at what level of population will the highest number of survivors be expected? Similarly, at what population level will the greatest number of newborns be achieved? These are analogous to the population level corresponding to maximum growth in count-based models. Therefore, if the term 'growth' is defined appropriately, this property is relevant to demographic rates too. The point is that the applicability of these two concepts is not confined to count-based models, even though they were originally presented in that context.

The effect of environmental autocorrelation on model predictions is influenced by the nature of the density dependence. When (positive) autocorrelation is present, the effect of environmental variation will be larger, hence density independent models

predict a higher extinction probability and a shorter time to reach the extinction. In density dependent models, the extinction risk can either increase or decrease by autocorrelation, depending on the values of other parameters in the model and the range over which the strength of autocorrelation is varied. The key determinant for density dependent models is whether the slope of the curve plotting N_{t+1} against N_t at the equilibrium point is positive or negative (Ripa and Heino 1999). If the slope is positive then positive autocorrelation will increase the extinction risk. If the slope is negative then increasing positive autocorrelation can decrease extinction risk over some range of values (Morris and Doak 2002, p135). This shows the importance of representing density dependence with an appropriately shaped function.

The process of matching functional properties to population characteristics was demonstrated using a series of DD functions for which the shape varies from linear to sigmoidal. Non-linearity in density dependent relationships is being observed with more and more populations, especially with marine mammal populations (Ragen and Fowler 1992). Therefore, although the logistic function was perhaps the most well known density dependent model in the past, it is unlikely such a linear function will be suitable for a marine mammal species where the maximum net productivity level (MNPL) is generally believed to be large. This might be the reason that this function not been suggested for the NZSL as part of any of the models proposed for the population.

The θ-logistic function, which is a non-linear generalization of the logistic function, is used in several population models proposed for the NZSL (e.g., Hilborn et al. 2000, Breen et al. 2003). Because this function can be used to represent maximum growth near carrying capacity it might be a potential candidate for the NZSL population. On the other hand, because the maximum population growth cannot occur at a population level below K/e under this function (where K is the carrying capacity and e is the base of natural log), it cannot be used in a count-based model to describe the density dependence of a population where the growth rate is large enough to produce maximum growth at a smaller population level. This agrees with Gilpin et al (1976), Pomerantz et al (1980), and Thomas et al (1980). Even though it does not make biological sense to have maximum growth at a density near zero, it is quite plausible

that populations exist that produce maximum growth at levels much smaller than *K/e* (Gilpin et al. 1976).

The Haldane function predicts maximum growth at a population size of $K(1-b)^{1/b}$, which does not exist if $b>1$ while it can be somewhere between zero and K/e when b is between zero and one (shown in section 7.6.3). Therefore, this function can describe maximum population growth only at population levels below *K/e*. For this reason, this model is not suitable for any species for which maximum population growth occurs at a larger population level. Although we do not know the NZSL population level that produces maximum growth, it is believed to be near *K* for large mammal species (Cooke 1994). It is interesting to note that the θ-logistic function predicts maximum growth at densities above *K/e* while this function predicts it at levels below *K/e*.

Under the B&H function, maximum population growth can occur only at population levels below K/2 (section 7.7.3). For this reason, this model cannot represent the dynamics of populations where the maximum growth occurs near K. This is consistent with the fact that this function has not been used in any of the models proposed for the NZSL population. This could also be a reason why this function is so common in fish population models where it is not uncommon for maximum productivity to be generated at densities of less than half the carrying capacity.

The Ricker function, which is also quite common in fish population models, cannot produce maximum growth at a population size below $\frac{K}{e+\ln(d)}$ or above $\frac{K}{2+\ln(d)}$ (appendix 8). These boundaries can be approximated by *K/e* and *K/2* when the parameter d is close to unity (assumed to be a fairly realistic situation in count-based models for most long-lived species). Since this function cannot exhibit maximum growth at population sizes above *K/2*, it does not seem suitable candidate for count-based modelling of long-lived slow-growing animal populations such as the NZSL. To my knowledge no one has suggested this function for the NZSL.

I used form-of-competition as a population characteristics. However, as stated in section 6.2.2, I should emphasize that populations cannot be divided into two distinct groups corresponding to contest and scramble, because any population shows both these characteristics to some extent. For this reason any method of labelling a population (or DD function) as contest or scramble is based on a certain 'cut-off' level. Different methods can label the same function differently because they do not necessarily use the same cut-off. For example, according to method 3 in section 6.2.2.3 (i.e., last row in table 6.2), only contest competition can be represented by the Hassell function, while according to method 2 (i.e., 4th row in table 6.2) it can represent both forms of competitions depending on the value of the parameter b (section 7.9.2). Bellows (1981) agrees with the first of these results while Hassell et al. (1976, p473) and Alsop (1998) agree with the second result. I have given another such example in section 7.10.2.

The form of the competition under the θ-Ricker function is determined by the magnitude of $b\ln(d)\left(\frac{N}{K}\right)^b$ at large N (section 7.5.2). Although this can increase without limit with increasing N, providing evidence of scramble competition (section 6.2.2.2), N is not much greater than K under most realistic conditions, so the this expression not much larger than $b\ln(d)$. In count-based models for long-lived slow-growing species the parameter d is usually near unity. Therefore, for species such as NZSL, this function is most likely to represent contest competition unless b is very large. Accordingly, if biological knowledge about the form of competition exists for the NZSL population, then the estimated value of the parameter b can be used to determine the suitability of this function for this population.

Under the B&H function, any count- based model predicts a stable population when $d>1$ (section 7.7.2). This result is a special case of the stability conditions obtained for more general functions, i.e., the MS&S function and the Hassell function (see sections 7.8 and 7.9). Of course, for any viable population, $d > 1$ in count-based models, because no population can avoid extinction if $d < 1$. Therefore, in agreement with Gurney and Nisbet (1998, p127), this function represents stable dynamics for all viable populations. This means that if the population at hand is known to be

associated with unstable, chaotic or limit cycles then this function cannot be a suitable candidate in modelling that population. The result of all viable populations being stable under this function is unusual considering that no population is stable when d is very large under any of the other functions examined.

Although I acknowledge the importance of considering a possible time lag in DD modelling, I did not aim to discuss it in this thesis. The biological basis for including such a lag is that in some populations resource limitations imposed on juveniles result in less reproductive adults a few years later. These functions can be modified to include a time lag. For example, Getz (1996) modified the MS&S function to $f(N_t) = d\left\{1 + (aN_{t-1})^b\right\}^{-1}$ to include a lag of one year. It can be generalised to include a lag of several years by using a linear combination of population sizes in previous years as $f(N_t) = d\left\{1 + (a_1 N_{t-1} + a_2 N_{t-2} + \ldots + a_p N_{t-p})^b\right\}^{-1}$.

Some of these functions are used in practice as linearly transformed versions of the forms given in this thesis. For example Runge (2002) used the logistic function, Chivers (1999) used the θ-logistic function, and May et al (1974) used the Usher function in linearly transformed forms. The transformed versions are useful in representing any desired range of the vital rate. However, the transformed and untransformed versions do not necessarily share the same properties. For example, transformation changes the form of competition represented by the Usher function (section 7.11). This result is important because modellers appear to use such transformations simply to cover a different range of the vital rate without realising the possible consequences. Therefore before using such a transformation, it is important to realize that these transformations could cause changes to some of the properties of the original function. The mapping of function properties to population characteristics needs to be done using the actual form of the function used in the population model (after any linear transformations), rather than relying on the summary of results given for untransformed versions in Table 7.1.

Not all functions are completely independent; some being special cases of the others. It is obvious that the logistic and Ricker functions are special cases of the θ–logistic

and θ–Ricker functions respectively, and that the B&H function is a special case of both the MS&S function and the Hassell function. One may think that these special cases are not important here because they are already covered by more general functions. However, if the properties of a special case function are consistent with the characteristics of the population then the simpler function is preferable as fewer parameters need to be specified (or to be estimated from data).

Also, some of these functions are approximations to others. For example, both the logistic and B&H functions are Taylor series approximations to the Ricker function. Similarly both the θ–logistic and MS&S functions are Taylor series approximations of the θ–Ricker function. One function being an approximation of another neither implies that their properties are similar nor that they are based on similar assumptions about the underlying dynamics. As seen in chapter 7, these approximations change some of the main properties of the functions. Most of these approximations are valid only when d is small, i.e. in the case of count-based models when the growth rate is small (Eberhardt 1977, Pitcher and Hart 1982, p188).

Since the suitability of a function is population-specific, I did not recommend one function over any other, but rather presented an approach that can be used to match population characteristics to functional properties. In this thesis I demonstrated this process by evaluating the properties of some commonly used functions, and then the importance of selecting a 'suitable' function (or possible consequences of selecting an 'unsuitable' function) was assessed using a simulation approach. Similar questions such as 'the effect of DD functions on the population predictions' have been addressed in the literature. For example, Runge and Johnson (2002) demonstrated that the model structure had a significant effect on population predictions. Their work differs from mine in two ways. First, they used an unstructured model, and the way that they selected parameter values for DD functions cannot be generalised to structured models. Second, they selected parameters for different functions so that the population reaches the same carrying capacity under all functions. This approach does not ensure that all the functions represent the same strength of DD, so the results are not exactly comparable. Pascual et al. (1997) examined the same question using a

structured model. But all the functions included in their study were simple two-parameter functions with limited flexibility.

In reality different vital rates may be affected differently by the density of different stages in structured models (Caswell 2001, p512), so the same DD function or the same measure of density may not be suitable for all vital rates. In my simulations in section 8.3 I used the same DD function and same measure of density for all vital rates because of the aim of this exercise is to examine the effect of the DD function on the outputs of the population model. This examination would have been quite complicated if different vital rates had followed different DD functions. For the same reason, I used the same strength of DD for all vital rates in each trajectory. This facilitated the interpretation of the effect of strength on population outcomes. However, in reality, DD curves in survival and reproduction need not follow the same function. In addition it is possible to have a high level of density dependence in the survival rate but a low level in the reproductive rate, and vice versa. I haven't explored those situations because of the aim of this exercise is not to study the fate of a population but to explore the effect of the DD function on possible projections. For this reason, although the simulations serve the purpose of the study, simulation results cannot be interpreted as necessarily relevant to the NZSL population.

In these simulations the resultant trajectories were compared between different functions using the predicted stationary population level (K) and the time to reach K. Different performance indicators have been used in the literature for similar purposes. For example, Neubert and Caswell (2000) used population stability (convergence, chaotic behaviour, bifurcation, etc) to compare the effect of DD on various vital rates. Such indicators have relatively little use with long-lived slow-growing animal populations. The annual population growth rate is another commonly used performance indicator, but it was not used in this study because it is dependent on the chosen projection period in density-dependent models.

Simulations show that the DD functions seem very similar within the range of the population, especially if the DD level is small (Figure 8.4). However the minor differences that exist between functions are magnified considerably into quite large differences in population trajectories, suggesting that the population trajectories are

highly sensitive to the functional form of the density dependence (figure 8.2). This is an interesting result in density dependent models, even though it is obvious in density independent models, where even a minor difference between trajectories can widen considerably after enough time has lapsed.

Of the four functions used in the simulation, the θ-logistic is the only one that does not produce a sigmoidal shape, hence one might expect to see different trajectories using this function. However, the simulations do not show this to any great extent. This demonstrates the difference between DD functions and the dynamics of the populations responding to those functions. As seen from Figure 8.4, in that particular example non-sigmoid and sigmoid functions start diverging at population levels above 12000, but none of the simulated populations grew to that level. Therefore, any differences in functional properties beyond the realistic population ranges have only a limited practical value. However, this statement should not be interpreted as saying that the NZSL population responds to θ-logistic function and other DD functions in a similar way (remember that the θ-logistic function is used in the latest model proposed for the NZSL population). This is because, although I use the context of the NZSL population to investigate DD functions my simulations do not reflect the status of that population for several reasons given earlier.

The DDS, DDR, and DDSR models differ from one another according to which vital rate(s) are made density dependent. The effect of density dependence on the various rates can be examined by comparing the elasticity of model outcomes to changes in those vital rates. I haven't calculated those measures in the present study. The method used in section 8.2 ensures that the DD levels are the 'same' for different DD functions within a vital rate, but not between vital rates. Hence, the strengths of different vital rates are therefore not necessarily the same. Consequently, the trajectories from the DDS, DDR, and DDSR models are not strictly comparable, so the above observations from the trajectories should be treated with caution.

Subject to this caveat, the simulations show that density dependent survival has more effect than density dependent reproduction. Introducing density dependent reproduction to a model with density dependent survival has only a small additional

effect. This additional effect is larger at lower DD levels than at higher DD levels. This means that if the DD in survival is already sufficient enough to regulate the population then the additional DD imposed by reproduction has only a marginal effect.

Heppell et al (2000) showed that fast-growing mammal species (such as rodents and small carnivores) generally have relatively high elasticity of growth rate to reproduction and lower elasticity of growth rate to survival. This agrees with Neubert and Caswell (2000) who found that for fast-growing animals, density dependence in survival cannot control the population growth when the reproduction is high, and density dependent reproduction is more likely to cause chaotic dynamics than density dependence in other vital rates. On the other hand, our observation of populations being more sensitive to density-dependent changes in survival is consistent with the results reported by Pascual et al. (1997) and Yearsley et al. (2003) for long-lived species. For such slow-growing species, many authors have also noted a strong sensitivity of other population outcomes (e.g., growth rate) to survival rate and weaker sensitivity to fecundity (e.g., Weimerskirch et al. 1997, Crooks et al. 1998, Russell 1999, Heppell et al. 2000, Slooten et al. 2000). Lebreton and Clobert (1993) quantified this result for long-lived species by showing that the elasticity of population growth rate to fecundity is of the order of $1/T$, and elasticity of growth rate to after-first-year survival rate is nearly $\left(1-1/T\right)$, where T is the population generation time. A consequence of this result is that if the generation time is greater than 2, elasticity relative to survival becomes higher than it is relative to fecundity. Notably, this is the case for all species that start breeding at age two or later. Therefore population outcomes for these species, and especially their population growth rate, are more sensitive to survival than to reproduction. Higher elasticity to survival means density dependence in survival can cause more chaotic changes than is caused by density dependence in reproduction. This agrees with the hypothesis that density dependent population regulation through reproduction is evolutionarily safer than through survival. It also implies that management of long-lived species that aim to achieve a target growth rate should focus on adjusting survival rate rather than on

adjusting fecundity. Also, if a change in growth rate is observed, it is more likely that a change in survival is the underlying cause than a change in reproduction.

The existence of a higher elasticity of growth rate with respect to survival does not mean that density dependent survival is more important than density dependent reproduction in a realistic model. In fact, density dependent reproduction is more common than density dependent survival in mammal population models. The reason for this apparent discrepancy is that elasticity analysis says what could happen if DD were present in survival (or in reproduction), but it does not say how likely it is to be present (Horvitz et al. 1997). Even though DDR has less effect, its presence could be more important in population models if it is more likely to occur than DDS. The existence of a negative correlation between survival and reproduction is evident among different populations of the same species (Thomas et al. 2000), and also between different species (Hautekeete et al. 2001), implying that there is a trade off between resource allocations to survival and reproduction. Takada (1995) found that this trade off plays an important role in fitness and evolution, which is usually measured in terms of an evolutionary objective (Ragen and Fowler 1992, Newton 1993, Brommer 2000). Teriokhin (1998) showed that, on the basis of maximising the evolutionary benefits, it is more beneficial to have density dependent changes in reproduction than in survival when a population is decreasing. Therefore, even though the simulation results show that projections are more sensitive to density dependent changes in survival, it may be reasonable to pay more attention to density dependence in reproduction in population modelling whenever field data indicates density dependent changes in survival are negligibly small compared to corresponding changes in reproduction. However, we need to be aware of the lessons of Pascual et al. (1997) that the density dependent trends apparent in data may be quite different from the true relationship.

The simulations show that a population model can predict different levels of carrying capacity (K) for a population if they are run with different DD functions (Figures 8.2, 8.5, and 8.6). The carrying capacity is the maximum level of population that can be supported by a given habitat or area without damage to the environment (Hine and Martin 2004), and therefore is a constant for a given population (in a given environment). If so how can we explain trajectories of the same population reaching

different levels of K under different functions when the environment remains the same? Suppose all DD functions were parameterized in terms of K (i.e., if K is specified explicitly in all the functions). Then the magnitudes of the density dependent vital rates are determined by K, and all of the trajectories can be expected to reached the specified K. Such an approach was used by Runge and Johnson (2002) but it cannot be generalised to structured models. Furthermore, the carrying capacity is usually unknown and cannot be specified explicitly for most populations, meaning that its magnitude is determined by the vital rates, which are specified by the DD function as in this study. Therefore the resultant value of K is influenced by the choice of the function. In other words, vital rates were assigned with different values when a different DD function was selected, resulting in a change in resource requirement per individual, and thus changing K.

9.3 IDD model

In this thesis I examined the population predictions that could result from population models when various forms of density dependence relationships were used. In doing so I assumed that separate DD functions can be estimated (or can be specified in some other way) for the vital rates. This raises the question of how we can incorporate density dependence into a demographic model if we do not have enough information in the data to estimate separate DD curves for each vital rate. I therefore presented the IDD model, which might be useful in such situations if we are comfortable with specifying population growth rate at a specific level of density. It can also be used to model density dependence when the DD relationship for one vital rate is influenced by the density dependence in another vital rate. Here the term 'influence' means deterministic dependence, as opposed to the stochastic dependence, implicit in the concept of cross correlation. A simple example for such a situation is when the maximum number of individuals (i.e., either the carrying capacity or a similar measure) that can be supported by the environment is constant over time. Suppose one vital rate becomes highly favourable for some reason. Then resource limitations may bring one or more of the other vital rates to an unfavourable level. A major advantage

in IDD models is that population predictions obtained from them are relatively insensitive to the choice of DD function.

In order to estimate a DD function for a single vital rate, information on the vital rate and population density are required over a wide range of population sizes. The non-availability of such data is a hindrance even to detecting the existence of density dependence in some populations, let alone to estimating its functional form. Suppose that a second vital rate has also varied during this estimation period. A DD function estimated from a univariate analysis becomes invalid as soon as this second vital rate deviates from its previous path. Since data availability becomes an obstacle to the estimation of the DD function even in univariate cases, it is seldom possible to use a multivariate approach to estimate a DD function by considering the changes in other vital rates. Therefore the validity of any simulation based on an estimated DD curve is likely to be questionable if the variation in the second vital rate deviates from its previous levels. A possible approach to this problem is modelling density dependence in both vital rates together. The IDD model is a step in that direction. They are not relevant to count-based population models where density dependence in individual demographic rates is not considered.

In section 8.3 we observed a considerable difference in population outcomes between different DD functions, even when the same strength of DD was simulated under each function. But as seen from Figure 8.8, the results of IDD-DDSR models are less likely to depend on the chosen DD function. Therefore, in IDD models, the burden of selecting one DD function over another can be taken lightly. This relative robustness of population outcome to chosen DD function is achieved through the re-calculation process, which at each time step attempts to direct the population trajectory towards a specific population level specified in the model (towards K in the simulations used in this thesis) irrespective of the actual DD function used.

IDD models, as introduced here, require K to be specified, and all population trajectories tend towards that specified level of K. This seems unrealistic because it is unlikely that all populations ultimately grow (or decline) towards K. In fact the purpose of most modelling exercises is to determine the ultimate fate of the population. This inflexibility can be removed by replacing the $\lambda = 1$ at $N = K$

statement in IDD models by the growth rate at a specific population size (i.e., something like $\lambda = \lambda^*$ at $N = N^*$). Then at each re-calculation step, the population growth rate will be redirected towards that point in the growth curve. Also, in the demonstration of the IDD model I used the same DD function for both vital rates. However, this is not strictly necessary. It is possible to use two different functional forms for reproduction and survival, and the estimation procedure given in Appendix 12 can still be used. However, there are at least two limitations to the applicability of IDD models. First, it is not immediately clear how they can be generalised to structured models because the dynamic equations of those models cannot be solved for the carrying capacity (or N^*) unless the model is very simple (Caswell 2001, p518). Second, there exists a minimum threshold in the vital rates below which this method cannot be used. It is possible for one of the vital rates to become smaller than this threshold for several reasons (e.g., due to density dependence when N is very large, or due to environmental variation when N is small).

9.4 Summary

In this research I have recommended some objective criteria that can be used in selecting a distribution for stochasticity, and in choosing a function for density dependence. The findings of this research will make the incorporation of those characteristics into population models more realistic. Even though the findings were presented in the context of modelling the long-lived vertebrate population NZSL, they are widely applicable as the methods and findings are useful anywhere in the area of population biology. Of the two questions 'how can we decide which distribution is better' and 'which distribution is better' the former has more practical value than the latter. This study attempted to answer the first question by taking the second question to illustrate the answer. To my knowledge no prior efforts have been devoted to explicitly assess any of these two questions.

The lognormalpower distribution was found to perform poorly. It can have undesirable shapes, and population models can produce quite different predictions when this distribution is used compared to using other distributions. The beta

distribution has several advantages over the other distributions considered. Namely, the parameters of the distribution can be specified analytically from the mean and standard deviation, parameter specification is exact, the generated random variates will have the intended statistical properties, can be used with vital rates that are distributed over any interval, can be used with all combinations of mean and standard deviation (that can be defined on the interval of interest), and model implementation is not dependent on the convergence of a numerical procedure. Its shape can be determined in advance from the mean and standard deviation. This helps avoid use of unrealistic combinations of mean and standard deviation when other available information suggests certain shapes are impossible. For example, if the available information suggests a unimodel shape is most likely for the population under consideration, then measures can be taken to avoid combinations of the mean and standard deviation that lead to other shapes. The only known drawback of the beta distribution is that it is not known how this distribution can be used to represent correlated vital rates.

Relaxation of the assumptions and limitations used in the thesis may suggest some paths for future research. Specifically, in modelling environmental stochasticity, I considered vital rates to be independently and identically distributed variates, and ignored cross-correlation and auto-correlation. It is widely accepted that models with correlated variates can represent the environmental stochasticity more realistically. Even though I identified several advantages of the beta distribution, there is no known way of generating correlated vital rates from this distribution. By choosing an appropriate transformation, and adapting the methods of Burgman et al (1993) and Todd and Ng (2001) to suit the chosen transformation, correlated vital rates can be generated from distributions if they are primarily based on the normal distribution. The logitnormal, probitnormal, and lognormalpower distributions are all based on the normal distribution. The suitability of the logitnormal and probitnormal distributions for representing correlated environmental stochasticity can be evaluated following the same general procedures used in this thesis.

In studying density dependence I did not consider the possibility of auto-correlation or time-lag. The growth rate or vital rates of a population do not necessarily respond to changes in density instantaneously. Neither do the properties of density-dependent

functions nor the population predictions necessarily remain the same if correlation and time lags are present. However, the general method used to investigate DD functions still remains valid, and these extra factors could be incorporated in the same way to future studies.

In this thesis I evaluated methods of modelling density dependence and environmental stochasticity independently of one another. However, in nature, various population effects occur simultaneously, and cannot always be distinguished from each other. Nevertheless, considering them independently helps us to understand the way in which they manifest themselves, and aids in interpretation. Such results are consistent with the aims of this thesis. However, it is not appropriate to take the model predictions obtained here to be predictions of the fate of an actual population. In studies with such an intention, both effects should be investigated simultaneously in a single model. In fact, other characteristics of populations (e.g., senescence), or features of data (e.g., uncertainty) could also be included in such models.

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APPENDIX 1

Maximum possible variance that can exist in a finite interval under any distribution

For any random variable, irrespective of which distribution it follows, the greatest variance ($s^2_{\max}$) occurs when some realizations are located at the lower boundary (a) of its range and all remaining realizations are at the upper boundary (b). Here, the use of the word 'boundary' implies that the variable has a finite range. The variable behaves like a discrete variable at this extreme situation, irrespective of whether the variable itself is discrete or continuous. If p_a is the fraction of realizations at the lower boundary, then the rest of the values (i.e., a fraction of $1-p_a$) are at the upper boundary. These fractions are dependent on the relative distances to a and b from the mean (m).

In this extreme situation we can consider the random variable as discrete, hence

$$m = (ap_a) + \{b(1-p_a)\} \quad ====> \quad p_a = \frac{b-m}{b-a}$$

$$s^2_{\max} = (a-m)^2 p_a + (b-m)^2 (1-p_a)$$

Since this is the maximum possible variance that can exist on this interval with given mean m,

$s^2 \le (a-m)^2 p_a + (b-m)^2 (1-p_a)$, where s is the standard deviation. Hence,

$$s^2 \le (a-m)^2 \left(\frac{b-m}{b-a}\right) + (b-m)^2 \left[1 - \left(\frac{b-m}{b-a}\right)\right].$$

This can be simplified to obtain

$$s^2 \le \frac{a^2b - a^2m - m^2b + am^2 + b^2m - b^2a}{b-a}$$

$$s^2 \le \frac{(bm - ba - m^2 + ma)(b-a)}{b-a}$$

$$s^2 \le (b-m)(m-a) \qquad \textbf{(A.1.1)}$$

Therefore, given the minimum, maximum, and mean for any random variable, the variance that can never be exceeded under any distribution is given by this expression. This s^2 is maximal when $m = \frac{a+b}{2}$. At this value of m this formula reduces to

$$s \leq \frac{(b-a)}{2}, \qquad \textbf{(A.1.2)}$$

offering an expression for the largest possible standard deviation in a given range.

In the case of survival, where $a = 0$ and $b = 1$, the above two formulas reduce to

$$s^2 \leq (1-m)m \quad \text{and} \quad s \leq \frac{1}{2}. \qquad \textbf{(A.1.3)}$$

This yields the plausible range of standard deviations for any given mean survival rate, and the largest possible standard deviation that can ever exist for survival under any distribution. Therefore it does not make sense to use any combination of mean and standard deviation with any distribution that does not conform to the above requirement.

APPENDIX 2

Probability density functions of distributions used for environmental stochasticity

(a) Beta distribution

The beta distribution has the probability density function of

$$f_S(s) = \frac{(s-a)^{\upsilon-1}(b-s)^{\omega-1}}{B(\upsilon,\omega)(b-a)^{\upsilon+\omega-1}}, \qquad \nu,\omega > 0$$

where, a and b are the minimum and maximum of the variable S, ν and ω are the shape parameters of the distribution, and $B(\upsilon,\omega)$ is the beta function (Johnson et al. 1994a). When a = 0, and b = 1 (i.e., when the variable is on unit range) the function reduces to

$$f_S(s) = \frac{(s)^{\upsilon-1}(1-s)^{\omega-1}}{B(\upsilon,\omega)}. \qquad \textbf{(A.2.1)}$$

This is often referred to as the standard beta distribution.

(b) Logitnormal distribution

If $\log\left(\frac{S}{1-S}\right) \sim N(\mu,\sigma^2)$ then the variable S is said to be logitnormally distributed. Therefore, the variate S has the probability density function of

$$f_S(s) = \left|\frac{1}{s(1-s)}\right| f_{\log it(S)}(\log it(s)),$$

where $\left|\frac{1}{s(1-s)}\right|$ is the Jacobian of the *inverse logit* transformation, and $f_{\log it(S)}(\log it(s))$ is the probability density function of logit(s) where $\log it(S) \sim N(\mu,\sigma^2)$. Therefore,

$$f_S(s) = \left|\frac{1}{s(1-s)}\right| \frac{1}{\sigma\sqrt{2\pi}} \exp\left[\frac{-\{\log it(s)-\mu\}^2}{2\sigma^2}\right]. \qquad \textbf{(A.2.2)}$$

(c) Probitnormal distribution

Variate S is said to be probitnormally distributed if $S \sim \Phi(x)$ where $X \sim N(\mu, \sigma^2)$ or equivalently $\Phi^{-1}(S) \sim N(\mu, \sigma^2)$, where Φ refers to the cumulative distribution function of standard normal. The probability function of S is then given by

$$f_S(s) = f_X\left(\Phi^{-1}(s)\right)\left|\frac{d}{ds}\left(\Phi^{-1}(s)\right)\right|.$$

This can be written as

$$f_S(s) = \phi\left(\frac{\Phi^{-1}(s) - \mu}{\sigma}\right)\frac{1}{\sigma.\phi(\Phi^{-1}(s))} \qquad \textbf{(A.2.3)}$$

where ϕ and Φ^{-1} refer to the density function of the standard normal, and the inverse function of the cumulative distribution function of the standard normal respectively.

An equivalent yet simpler version can be obtained by using the relationship of

$f_S(s) = f_X(x)\left|\frac{dx}{ds}\right|$ where $s = \Phi(x) = \int_{-\infty}^{x} \frac{1}{\sqrt{2\pi}} \exp\left(\frac{-z^2}{2}\right) dz$ and $X \sim N(\mu, \sigma^2)$.

Using Leibniz's rule of integration (Trench 1978, p589), it can be seen that

$$\left|\frac{ds}{dx}\right| = \frac{1}{\sqrt{2\pi}} \exp\left(\frac{-x^2}{2}\right).$$

Therefore,

$$f_S(s) = f_X(x)\sqrt{2\pi}\exp\left(\frac{x^2}{2}\right), \qquad \textbf{(A.2.4)}$$

where $X \sim N(\mu, \sigma^2)$, $S = \Phi(x)$, and $f_X(x)$ is the probability density function of x.

Equation A.2.4 version is easier to evaluate in Matlab® than A.2.3, and is used in producing Figures 4.1, 4.5, 5.7, and 5.9. Both A.2.3 and A.2.4 can be used in Excel®.

(d) Lognormalpower distribution

A variate S is lognormalpower distributed if $S = A^{\exp(x)}$, $X \sim N(0,\sigma^2)$, or equivalently if $\ln\left(\frac{\ln(S)}{\ln(A)}\right) \sim N(0,\sigma^2)$. Therefore, the probability density function of S is given by

$$f_S(s) = \left|\frac{1}{s\ln(s)}\right| * f_X\left(\ln\{\ln(s)/\ln(A)\}\right)$$

where, $f_X\left(\ln\{\ln(s)/\ln(A)\}\right)$ is the probability density function of $\ln\left(\frac{\ln(S)}{\ln(A)}\right)$, and $\left|\frac{1}{s\ln(s)}\right|$ is the Jacobian of the lognormalpower transformation. Therefore,

$$f_S(s) = \left|\frac{1}{s\ln(s)}\right| \frac{1}{\sigma\sqrt{2\pi}} \exp\left[-\left\{\ln\left(\frac{\ln(s)}{\ln(A)}\right)\right\}^2 \Big/ 2\sigma^2\right]. \qquad \textbf{(A.2.5)}$$

APPENDIX 3

Obtaining parameters for distributions in terms of their mean and standard deviation

The parameters of the beta distribution can be specified analytically in terms of the mean and standard deviation of the stochastic variation, but the parameters of other three distributions (logitnormal, lognormalpower, or probitnormal) cannot. Even though the delta method (Stuart and Ord 1994) can be used to obtain approximate parameter values, the level of approximation is found to be unacceptable (section 4.3.6). Therefore a numerical estimation method needs to be employed to obtain parameter values for these three distributions. This appendix shows the method used in the thesis to obtain parameter values.

This is not a 'parameter estimation' in the sense of usual meaning of the term which refers to estimating values of parameters from raw data, but rather a specifying parameters in terms of mean and standard deviation of variability. Since mean and standard deviation are estimated from raw data in practice, this becomes an indirect way of seeing the possibility of estimating parameters of the distributions from field data.

(a) Beta distribution

For standard beta distribution (i.e. when minimum is zero and maximum is one), two shape parameters ν and ω are related to the mean (m) and standard deviation (s) of the distribution by (Evans et al. 1993),

$$m = \frac{\nu}{\nu + \omega} \quad \text{and} \quad s^2 = \frac{\nu\omega}{(\nu+\omega)^2(\nu+\omega+1)}.$$

Because a beta distributed variable (x) over the range a to b is related to the standard beta variable (x_s) as $x_s = \dfrac{x-a}{b-a}$ (Evans et al. 1993), the above relationships can be generalized to have a minimum of a and maximum of b by writing

$$m = a + \left(\frac{\nu}{\nu+\omega}\right)(b-a) \quad \text{and} \quad s^2 = \frac{(\nu\omega)(b-a)^2}{(\nu+\omega)^2(\nu+\omega+1)}. \qquad \textbf{(A.3.1)}$$

These two equations can be used to obtain the shape parameters:

$$v=\left(\frac{m-a}{b-a}\right)\left(\frac{(b-m)(m-a)}{s^2}-1\right) \quad \text{and} \quad \omega=\left(\frac{b-m}{b-a}\right)\left(\frac{(b-m)(m-a)}{s^2}-1\right). \quad \textbf{(A.3.2)}$$

When a = 0 and b =1 (i.e., the case of survival rate), the above equations are simplified to

$$v=(m)\left(\frac{(1-m)m}{s^2}-1\right) \quad \text{and} \quad \omega=(1-m)\left(\frac{(1-m)m}{s^2}-1\right). \quad \textbf{(A.3.3)}$$

(b) Logitnormal distribution

If the variable S is logitnormally distributed over the unit interval then its mean is given by

$E(S)=\int_0^1 s.f(s).ds$, where *f(s)* is the probability density function of S. Using the formula for *f(s)* from appendix 2,

$$E(S)=\int_0^1\left|\frac{s}{s(1-s)}\right|\frac{1}{\sigma\sqrt{2\pi}}\exp\left[\frac{-\{\log it(s)-\mu\}^2}{2\sigma^2}\right].ds$$

$$=\int_{-\infty}^{+\infty}\phi(z)\frac{\exp(z\sigma+\mu)}{1+\exp(z\sigma+\mu)}dz, \quad \text{where} \quad z=\frac{\log it(s)-\mu}{\sigma} \quad \text{and}$$

$\phi(z)$ = standard normal probability density function of z.

Similarly, $E(S^2)=\int_{-\infty}^{+\infty}\phi(z)\left(\frac{\exp(z\sigma+\mu)}{1+\exp(z\sigma+\mu)}\right)^2.dz$

Therefore the variance of S is given by $V(S)=E(S^2)-[E(S)]^2$.

Since the required mean (m) and required standard deviation (s) are specified, numerical integration and optimisation techniques can be used to find values for μ and σ that result in E(S) and V(S) at required level. For example, the *quadl* integration function and the *fminsearch* optimisation function in Matlab® can be used to find values of μ and σ that minimise the sum of squares of the differences given by $\{m-E(S)\}^2+\{s-\sqrt{V(S)}\}^2$.

(c) ProbitNormal distribution

If the variable S is probitnormally distributed over the unit interval then its mean is given by

$$E(S) = \int_0^1 s.f(s).ds \ ,$$

where *f(s)* is the probability density function of S. Using the formula for *f(s)* from appendix 2,

$$E(S) = \int_0^1 \phi\left(\frac{\Phi^{-1}(s) - \mu}{\sigma}\right)\frac{s}{\sigma.\phi(\Phi^{-1}(s))}.ds$$

$$= \int_{-\infty}^{+\infty} \phi(z).\Phi(\sigma z + \mu).dz \text{ , where } z = \frac{\Phi^{-1}(s) - \mu}{\sigma}.$$

Similarly, $E(S^2) = \int_{-\infty}^{+\infty} \phi(z).\Phi^2(\sigma z + \mu).dz$.

Therefore the variance of S is given by $V(S) = E(S^2) - [E(S)]^2$.

Once the mean (m) and variance (s^2) are specified, these equations for *E(S)* and *V(S)* can be solved for μ and σ using numerical methods. Here we find the values of μ and σ that minimise the sum of the squares of difference given by $\{m - E(S)\}^2 + \left\{s - \sqrt{V(S)}\right\}^2$. It is easier to perform this minimization in Matlab® using the built in *quald* and *fminsearch* functions, if the above expressions for *E(S)* and *E(S²)* are written as

$$E(S) = \int_{-\infty}^{+\infty} \frac{1}{\sqrt{2\pi}}\exp(-0.5z^2)\left(\frac{1}{2}\right)\left(1 + erf\left(\frac{\mu + \sigma z}{\sqrt{2}}\right)\right).dz \text{ , and}$$

$$E(S^2) = \int_{-\infty}^{+\infty} \frac{1}{\sqrt{2\pi}}\exp(-0.5z^2)\left[\left(\frac{1}{2}\right)\left(1 + erf\left(\frac{\mu + \sigma z}{\sqrt{2}}\right)\right)\right]^2.dz \text{ , where}$$

erf(x) is the error function of *x*.

(d) LogNormalPower distribution

If the variable S is lognormalpower distributed over the unit interval then its mean is given by

$$E(S) = \int_0^1 s.f(s).ds \ ,$$

where *f(s)* is the probability density function of S. Using the formula for *f(s)* from appendix 2,

$$E(S) = \int_0^1 \frac{1}{|\ln(s)|} \frac{1}{\sigma\sqrt{2\pi}} \exp\left[\left(\frac{-1}{2\sigma^2}\right)\left\{\ln\left(\frac{\ln(s)}{\ln(A)}\right)\right\}^2\right].ds$$

$$= \int_{-\infty}^{+\infty} \frac{1}{\sqrt{2\pi}} \exp\left(\frac{-z^2}{2}\right) A^{\exp(z\sigma)}.dz$$

where $s = A^{\exp(z\sigma)}$, or equivalently $z = \frac{1}{\sigma}\ln\left(\frac{\ln(s)}{\ln(A)}\right)$.

Similarly, $$E(S^2) = \int_0^1 \frac{s}{|\ln(s)|} \frac{1}{\sigma\sqrt{2\pi}} \exp\left[\left(\frac{-1}{2\sigma^2}\right)\left\{\ln\left(\frac{\ln(s)}{\ln(A)}\right)\right\}^2\right].ds$$

$$= \int_{-\infty}^{+\infty} \frac{1}{\sqrt{2\pi}} \exp\left(\frac{-z^2}{2}\right)\left(A^{\exp(z\sigma)}\right)^2.dz$$

Therefore, $V(S) = E(S^2) - [E(S)]^2$.

Like the logitnormal distribution, once mean and variance are specified, numerical integration and optimization techniques can be used to find values for A and σ. Here we can find values of A and σ that minimise the sum of squares given by $\{m - E(S)\}^2 + \{s - \sqrt{V(S)}\}^2$.

APPENDIX 4A

Comparison of intended and realised variability from beta distribution.

The intended variability is given in the first three columns. Mean values were selected to be spaced between 0.5 and 1.0, which is the most likely range for a large mammal's survival rate. Standard deviations were selected to cover most of the realistic range for each mean value. θ is the concentration parameter defined in section 5.6.9. The fourth and fifth columns give the shape parameters specified by intended mean and variance. The next 4 columns present the variability of the variates generated by two different beta generators when specified values are used for parameters.

Intended variability			parameters specified		Variability of variates generated by Csbetarnd		variability of variates generated by Betadat	
mean	s	θ	v	ω	mean	s	mean	s
0.50	0.05	99.00	49.5000	49.5000	0.5000	0.0497	0.5003	0.0497
0.50	0.10	24.00	12.0000	12.0000	0.5018	0.0995	0.5016	0.0995
0.50	0.15	10.11	5.0556	5.0556	0.5002	0.1523	0.4971	0.1509
0.50	0.20	5.25	2.6250	2.6250	0.5014	0.2015	0.5027	0.1993
0.50	0.25	3.00	1.5000	1.5000	0.5089	0.2490	0.5028	0.2465
0.50	0.30	1.78	0.8889	0.8889	0.5146	0.3009	0.5012	0.2999
0.50	0.35	1.04	0.5204	0.5204	0.5006	0.3459	0.4910	0.3547
0.50	0.40	0.56	0.2813	0.2813	0.4883	0.3621	0.4879	0.3984
0.50	0.45	0.23	0.1173	0.1173	0.5030	0.3931	0.5138	0.4491
0.55	0.05	98.00	53.9000	44.1000	0.5507	0.0499	0.5498	0.0499
0.55	0.10	23.75	13.0625	10.6875	0.5511	0.1013	0.5510	0.1017
0.55	0.15	10.00	5.5000	4.5000	0.5517	0.1494	0.5481	0.1479
0.55	0.20	5.19	2.8531	2.3344	0.5486	0.1984	0.5559	0.2018
0.55	0.25	2.96	1.6280	1.3320	0.5547	0.2467	0.5523	0.2530
0.55	0.30	1.75	0.9625	0.7875	0.5488	0.2956	0.5485	0.2971
0.55	0.35	1.02	0.5612	0.4592	0.5126	0.3394	0.5469	0.3524
0.55	0.40	0.55	0.3008	0.2461	0.5257	0.3651	0.5437	0.4023
0.55	0.45	0.22	0.1222	0.1000	0.5070	0.3928	0.5569	0.4453
0.60	0.05	95.00	57.0000	38.0000	0.6023	0.0509	0.6010	0.0492
0.60	0.10	23.00	13.8000	9.2000	0.5986	0.1006	0.5956	0.1018
0.60	0.15	9.67	5.8000	3.8667	0.5964	0.1520	0.6050	0.1473
0.60	0.20	5.00	3.0000	2.0000	0.5949	0.1979	0.6014	0.1981
0.60	0.25	2.84	1.7040	1.1360	0.6063	0.2503	0.6009	0.2517
0.60	0.30	1.67	1.0000	0.6667	0.6001	0.3004	0.5958	0.3035
0.60	0.35	0.96	0.5755	0.3837	0.5656	0.3432	0.6021	0.3509
0.60	0.40	0.50	0.3000	0.2000	0.5281	0.3692	0.5915	0.4025
0.60	0.45	0.19	0.1111	0.0741	0.5349	0.3967	0.5879	0.4515
0.65	0.05	90.00	58.5000	31.5000	0.6505	0.0491	0.6531	0.0494
0.65	0.10	21.75	14.1375	7.6125	0.6508	0.1002	0.6516	0.1023
0.65	0.15	9.11	5.9222	3.1889	0.6465	0.1517	0.6514	0.1492
0.65	0.20	4.69	3.0469	1.6406	0.6525	0.1980	0.6574	0.1945
0.65	0.25	2.64	1.7160	0.9240	0.6460	0.2490	0.6575	0.2451
0.65	0.30	1.53	0.9931	0.5347	0.6321	0.2988	0.6522	0.3007
0.65	0.35	0.86	0.5571	0.3000	0.5902	0.3380	0.6560	0.3468
0.65	0.40	0.42	0.2742	0.1477	0.5453	0.3812	0.6589	0.3974
0.65	0.45	0.12	0.0802	0.0432	0.5329	0.4092	0.6487	0.4498
0.70	0.05	83.00	58.1000	24.9000	0.7005	0.0481	0.7004	0.0497
0.70	0.10	20.00	14.0000	6.0000	0.6972	0.1004	0.6985	0.0985
0.70	0.15	8.33	5.8333	2.5000	0.7021	0.1497	0.7033	0.1508
0.70	0.20	4.25	2.9750	1.2750	0.6995	0.2019	0.6977	0.2016
0.70	0.25	2.36	1.6520	0.7080	0.6920	0.2500	0.6978	0.2541
0.70	0.30	1.33	0.9333	0.4000	0.6524	0.2978	0.6931	0.3041
0.70	0.35	0.71	0.5000	0.2143	0.6123	0.3450	0.6950	0.3548
0.70	0.40	0.31	0.2188	0.0938	0.5539	0.3815	0.6982	0.3991
0.70	0.45	0.04	0.0259	0.0111	0.5105	0.4219	0.7107	0.4460
0.75	0.05	74.00	55.5000	18.5000	0.7505	0.0477	0.7506	0.0503
0.75	0.10	17.75	13.3125	4.4375	0.7505	0.0995	0.7489	0.1008
0.75	0.15	7.33	5.5000	1.8333	0.7501	0.1528	0.7471	0.1500
0.75	0.20	3.69	2.7656	0.9219	0.7412	0.2058	0.7521	0.1956
0.75	0.25	2.00	1.5000	0.5000	0.7094	0.2524	0.7546	0.2447
0.75	0.30	1.08	0.8125	0.2708	0.6753	0.3069	0.7706	0.2904
0.75	0.35	0.53	0.3980	0.1327	0.5982	0.3587	0.7462	0.3519
0.75	0.40	0.17	0.1289	0.0430	0.5329	0.3977	0.7374	0.4081
0.80	0.05	63.00	50.4000	12.6000	0.7989	0.0512	0.8009	0.0495
0.80	0.10	15.00	12.0000	3.0000	0.8018	0.1010	0.7978	0.0995
0.80	0.15	6.11	4.8889	1.2222	0.8005	0.1477	0.7971	0.1514
0.80	0.20	3.00	2.4000	0.6000	0.7798	0.1964	0.8018	0.1984
0.80	0.25	1.56	1.2480	0.3120	0.7338	0.2545	0.8148	0.2420
0.80	0.30	0.78	0.6222	0.1556	0.6588	0.3287	0.8049	0.2965
0.80	0.35	0.31	0.2449	0.0612	0.5892	0.3806	0.8011	0.3478
0.85	0.05	50.00	42.5000	7.5000	0.8520	0.0495	0.8494	0.0497
0.85	0.10	11.75	9.9875	1.7625	0.8507	0.1007	0.8495	0.1023
0.85	0.15	4.67	3.9667	0.7000	0.8304	0.1483	0.8509	0.1499
0.85	0.20	2.19	1.8594	0.3281	0.7913	0.2094	0.8494	0.2002
0.85	0.25	1.04	0.8840	0.1560	0.7310	0.2817	0.8578	0.2450
0.85	0.30	0.42	0.3542	0.0625	0.6336	0.3606	0.8486	0.3029
0.85	0.35	0.04	0.0347	0.0061	0.5039	0.4248	0.8491	0.3526
0.90	0.05	35.00	31.5000	3.5000	0.8976	0.0511	0.8996	0.0501
0.90	0.10	8.00	7.2000	0.8000	0.8754	0.1038	0.8998	0.1026
0.90	0.15	3.00	2.7000	0.3000	0.8402	0.1673	0.8997	0.1499
0.90	0.20	1.25	1.1250	0.1250	0.7820	0.2486	0.8963	0.2019
0.90	0.25	0.44	0.3960	0.0440	0.6552	0.3610	0.8903	0.2676
0.95	0.05	18.00	17.1000	0.9000	0.9266	0.0554	0.9501	0.0497
0.95	0.10	3.75	3.5625	0.1875	0.8869	0.1262	0.9536	0.0909
0.95	0.15	1.11	1.0556	0.0556	0.8076	0.2475	0.9528	0.1412
0.95	0.20	0.19	0.1781	0.0094	0.6103	0.4020	0.9549	0.1905

APPENDIX 4B

Comparison of intended and realised variability from logitnormal distribution.

The intended variability is given in the first three columns. Mean values were selected to be spaced between 0.5 and 1.0, which is the most likely range for large mammal's survival rate. Standard deviations were selected to cover most of the realistic range with each mean value. θ is the concentration parameter defined in section 5.6.9. The fourth and fifth columns give the parameter values specified to represent the intended variability. Since the parameter specification is not exact, I calculated the mean and standard deviation of the distribution defined by the specified parameter values, and they are presented in columns 6 and 7. The last two columns give the variability of variates generated by the distribution.

Intended variability			parameters specified		variability represented by parameters		variability represented by variates	
mean	s	θ	μ	σ	mean	s	mean	s
0.50	0.05	99.0000	0.0000	0.2020	0.5000	0.0500	0.4996	0.0492
0.50	0.10	24.0000	0.0000	0.4165	0.5000	0.1000	0.4997	0.0996
0.50	0.15	10.1111	0.0000	0.6578	0.5000	0.1500	0.4969	0.1539
0.50	0.20	5.2500	0.0000	0.9459	0.5000	0.2000	0.5061	0.1988
0.50	0.25	3.0000	0.0000	1.3126	0.5000	0.2500	0.4933	0.2463
0.50	0.30	1.7778	0.0000	1.8187	0.5000	0.3000	0.4863	0.3010
0.50	0.35	1.0408	0.0000	2.6009	0.5000	0.3500	0.4927	0.3506
0.50	0.40	0.5625	0.0000	4.0625	0.5000	0.4000	0.4908	0.4020
0.50	0.45	0.2346	-0.0022	7.0978	0.4999	0.4422	0.4982	0.4416
0.55	0.05	98.0000	0.2027	0.2041	0.5500	0.0500	0.5518	0.0505
0.55	0.10	23.7500	0.2092	0.4207	0.5500	0.1000	0.5492	0.1006
0.55	0.15	10.0000	0.2209	0.6646	0.5500	0.1500	0.5501	0.1503
0.55	0.20	5.1875	0.2396	0.9560	0.5500	0.2000	0.5476	0.1995
0.55	0.25	2.9600	0.2687	1.3281	0.5500	0.2500	0.5481	0.2483
0.55	0.30	1.7500	0.3157	1.8431	0.5500	0.3000	0.5474	0.3029
0.55	0.35	1.0204	0.3979	2.6442	0.5500	0.3500	0.5466	0.3511
0.55	0.40	0.5469	0.5684	4.1605	0.5500	0.4000	0.5410	0.4014
0.55	0.45	0.2222	0.9014	7.0888	0.5490	0.4398	0.5396	0.4427
0.60	0.05	95.0000	0.4098	0.2104	0.6000	0.0500	0.6004	0.0497
0.60	0.10	23.0000	0.4236	0.4339	0.6000	0.1000	0.6012	0.1003
0.60	0.15	9.6667	0.4483	0.6858	0.6000	0.1500	0.5987	0.1516
0.60	0.20	5.0000	0.4881	0.9880	0.6000	0.2000	0.6023	0.1990
0.60	0.25	2.8400	0.5505	1.3763	0.6000	0.2500	0.6064	0.2441
0.60	0.30	1.6667	0.6519	1.9202	0.6000	0.3000	0.6068	0.2966
0.60	0.35	0.9592	0.8327	2.7841	0.6000	0.3500	0.6014	0.3477
0.60	0.40	0.5000	1.2234	4.4888	0.6000	0.4000	0.6039	0.4004
0.60	0.45	0.1852	1.7912	7.0799	0.5969	0.4332	0.5924	0.4325
0.65	0.05	90.0000	0.6264	0.2220	0.6500	0.0500	0.6494	0.0506
0.65	0.10	21.7500	0.6491	0.4577	0.6500	0.1000	0.6533	0.1014
0.65	0.15	9.1111	0.6904	0.7242	0.6500	0.1500	0.6526	0.1479
0.65	0.20	4.6875	0.7567	1.0460	0.6500	0.2000	0.6522	0.1984
0.65	0.25	2.6400	0.8618	1.4647	0.6500	0.2500	0.6510	0.2494
0.65	0.30	1.5278	1.0360	2.0647	0.6500	0.3000	0.6567	0.2982
0.65	0.35	0.8571	1.3582	3.0566	0.6500	0.3500	0.6523	0.3505
0.65	0.40	0.4219	2.1141	5.1871	0.6500	0.4000	0.6401	0.4041
0.65	0.45	0.1235	2.6618	7.0712	0.6424	0.4226	0.6436	0.4204
0.70	0.05	83.0000	0.8587	0.2404	0.7000	0.0500	0.7004	0.0518
0.70	0.10	20.0000	0.8942	0.4958	0.7000	0.1000	0.6973	0.1000
0.70	0.15	8.3333	0.9583	0.7855	0.7000	0.1500	0.7027	0.1515
0.70	0.20	4.2500	1.0619	1.1392	0.7000	0.2000	0.6955	0.1980
0.70	0.25	2.3600	1.2289	1.6096	0.7000	0.2500	0.6968	0.2499
0.70	0.30	1.3333	1.5151	2.3101	0.7000	0.3000	0.7063	0.2941
0.70	0.35	0.7143	2.0819	3.5542	0.7000	0.3500	0.7130	0.3464
0.70	0.40	0.3125	3.6462	6.7166	0.7000	0.4000	0.7009	0.3991
0.70	0.45	0.0370	3.5040	7.0628	0.6847	0.4088	0.6966	0.4042
0.75	0.05	74.0000	1.1164	0.2691	0.7500	0.0500	0.7506	0.0516
0.75	0.10	17.7500	1.1716	0.5546	0.7500	0.1000	0.7519	0.0984
0.75	0.15	7.3333	1.2708	0.8803	0.7500	0.1500	0.7502	0.1501
0.75	0.20	3.6875	1.4329	1.2856	0.7500	0.2000	0.7477	0.2005
0.75	0.25	2.0000	1.7017	1.8448	0.7500	0.2500	0.7491	0.2505
0.75	0.30	1.0833	2.1915	2.7355	0.7500	0.3000	0.7450	0.3052
0.75	0.35	0.5306	3.2951	4.5477	0.7500	0.3500	0.7541	0.3509
0.75	0.40	0.1719	4.7169	7.0507	0.7416	0.3837	0.7506	0.3786
0.80	0.05	63.0000	1.4155	0.3148	0.8000	0.0500	0.8005	0.0507
0.80	0.10	15.0000	1.5046	0.6475	0.8000	0.1000	0.7997	0.1022
0.80	0.15	6.1111	1.6643	1.0307	0.8000	0.1500	0.8007	0.1476
0.80	0.20	3.0000	1.9304	1.5240	0.8000	0.2000	0.7959	0.2053
0.80	0.25	1.5600	2.3977	2.2524	0.8000	0.2500	0.8004	0.2472
0.80	0.30	0.7778	3.3581	3.5733	0.8000	0.3000	0.8046	0.2946
0.80	0.35	0.3061	6.0953	7.0369	0.7993	0.3490	0.8072	0.3464
0.85	0.05	50.0000	1.7877	0.3932	0.8500	0.0500	0.8501	0.0491
0.85	0.10	11.7500	1.9444	0.8040	0.8500	0.1000	0.8520	0.0970
0.85	0.15	4.6667	2.2248	1.2877	0.8500	0.1500	0.8507	0.1497
0.85	0.20	2.1875	2.7166	1.9562	0.8500	0.2000	0.8520	0.1965
0.85	0.25	1.0400	3.6975	3.0955	0.8500	0.2500	0.8529	0.2464
0.85	0.30	0.4167	6.4396	5.9470	0.8500	0.3000	0.8438	0.3059
0.85	0.35	0.0408	6.9305	7.0285	0.8302	0.3257	0.8274	0.3306
0.90	0.05	35.0000	2.3141	0.5477	0.9000	0.0500	0.8999	0.0517
0.90	0.10	8.0000	2.6377	1.1050	0.9000	0.1000	0.8966	0.0993
0.90	0.15	3.0000	3.2327	1.8088	0.9000	0.1500	0.8944	0.1531
0.90	0.20	1.2500	4.4558	2.9852	0.9000	0.2000	0.9010	0.1981
0.90	0.25	0.4400	8.1706	6.1150	0.9000	0.2500	0.8945	0.2512
0.95	0.05	18.0000	3.3379	0.9577	0.9500	0.0500	0.9500	0.0498
0.95	0.10	3.7500	4.3163	1.9131	0.9500	0.1000	0.9511	0.0936
0.95	0.15	1.1111	6.7245	3.6673	0.9500	0.1500	0.9478	0.1534
0.95	0.20	0.1875	11.3704	6.9841	0.9425	0.1956	0.9449	0.1917

APPENDIX 4C

Comparison of intended and realised variability from probitnormal distribution.

The intended variability is given in the first three columns. Mean values were selected to be spaced between 0.5 and 1.0, which the most likely range for large mamml's survival rate. Standard deviations were selected to cover most of the realistic range with each mean value. θ is the concentration parameter defined in section 5.6.9. The fourth and fifth columns give the parameter values specified to represent the intended variability. Since this specification is approximate, I calculated the mean and standard deviation of the distribution defined by the specified values of parameters, and they are presented in columns 6 and 7. The last two columns give the variability of the variates generated by the distribution.

intended variability			parameters specified		variability represented by parameters		varibility represented by variates	
mean	s	θ	μ	σ	mean	s	mean	s
0.50	0.05	99.0000	0.0000	0.1263	0.5000	0.0500	0.5014	0.0491
0.50	0.10	24.0000	0.0000	0.2589	0.5000	0.1000	0.4989	0.1025
0.50	0.15	10.1111	0.0000	0.4050	0.5000	0.1500	0.4975	0.1500
0.50	0.20	5.2500	0.0000	0.5753	0.5000	0.2000	0.5020	0.1984
0.50	0.25	3.0000	0.0000	0.7873	0.5000	0.2500	0.5050	0.2509
0.50	0.30	1.7778	0.0000	1.0744	0.5000	0.3000	0.5083	0.2994
0.50	0.35	1.0408	0.0000	1.5128	0.5000	0.3500	0.5027	0.3468
0.50	0.40	0.5625	0.0000	2.3289	0.5000	0.4000	0.5154	0.4019
0.50	0.45	0.2346	0.0000	4.6498	0.5000	0.4500	0.4961	0.4516
0.55	0.05	98.0000	0.1267	0.1273	0.5500	0.0500	0.5503	0.0496
0.55	0.10	23.7500	0.1299	0.2609	0.5500	0.1000	0.5516	0.1016
0.55	0.15	10.0000	0.1358	0.4085	0.5500	0.1500	0.5457	0.1506
0.55	0.20	5.1875	0.1453	0.5807	0.5500	0.2000	0.5542	0.2022
0.55	0.25	2.9600	0.1605	0.7955	0.5500	0.2500	0.5499	0.2515
0.55	0.30	1.7500	0.1857	1.0875	0.5500	0.3000	0.5557	0.2976
0.55	0.35	1.0204	0.2304	1.5364	0.5500	0.3500	0.5595	0.3526
0.55	0.40	0.5469	0.3248	2.3832	0.5500	0.4000	0.5472	0.4038
0.55	0.45	0.2222	0.6257	4.8776	0.5500	0.4500	0.5507	0.4515
0.60	0.05	95.0000	0.2555	0.1305	0.6000	0.0500	0.5994	0.0507
0.60	0.10	23.0000	0.2623	0.2675	0.6000	0.1000	0.6003	0.1009
0.60	0.15	9.6667	0.2747	0.4193	0.6000	0.1500	0.6013	0.1496
0.60	0.20	5.0000	0.2951	0.5972	0.6000	0.2000	0.6061	0.2013
0.60	0.25	2.8400	0.3278	0.8210	0.6000	0.2500	0.6015	0.2461
0.60	0.30	1.6667	0.3821	1.1290	0.6000	0.3000	0.6002	0.2989
0.60	0.35	0.9592	0.4808	1.6129	0.6000	0.3500	0.5980	0.3500
0.60	0.40	0.5000	0.6976	2.5655	0.6000	0.4000	0.6116	0.3977
0.60	0.45	0.1852	1.4760	5.7396	0.6000	0.4500	0.5949	0.4502
0.65	0.05	90.0000	0.3889	0.1361	0.6500	0.0500	0.6503	0.0498
0.65	0.10	21.7500	0.4000	0.2795	0.6500	0.1000	0.6500	0.1027
0.65	0.15	9.1111	0.4208	0.4389	0.6500	0.1500	0.6508	0.1500
0.65	0.20	4.6875	0.4549	0.6275	0.6500	0.2000	0.6465	0.2015
0.65	0.25	2.6400	0.5102	0.8679	0.6500	0.2500	0.6327	0.2513
0.65	0.30	1.5278	0.6040	1.2070	0.6500	0.3000	0.6422	0.3025
0.65	0.35	0.8571	0.7808	1.7623	0.6500	0.3500	0.6485	0.3505
0.65	0.40	0.4219	1.2018	2.9543	0.6500	0.4000	0.6582	0.4015
0.65	0.45	0.1235	3.2242	8.3076	0.6500	0.4500	0.6338	0.4548
0.70	0.05	83.0000	0.5299	0.1451	0.7000	0.0500	0.6990	0.0504
0.70	0.10	20.0000	0.5472	0.2984	0.7000	0.1000	0.7017	0.1010
0.70	0.15	8.3333	0.5794	0.4701	0.7000	0.1500	0.6971	0.1492
0.70	0.20	4.2500	0.6330	0.6761	0.7000	0.2000	0.6974	0.1977
0.70	0.25	2.3600	0.7216	0.9451	0.7000	0.2500	0.6999	0.2476
0.70	0.30	1.3333	0.8768	1.3400	0.7000	0.3000	0.7029	0.2947
0.70	0.35	0.7143	1.1899	2.0368	0.7000	0.3500	0.7070	0.3448
0.70	0.40	0.3125	2.0655	3.8097	0.7000	0.4000	0.7030	0.3968
0.70	0.45	0.0370	13.7171	26.1385	0.7000	0.4500	0.6967	0.4519
0.75	0.05	74.0000	0.6829	0.1588	0.7500	0.0500	0.7505	0.0504
0.75	0.10	17.7500	0.7097	0.3274	0.7500	0.1000	0.7493	0.0996
0.75	0.15	7.3333	0.7598	0.5185	0.7500	0.1500	0.7494	0.1481
0.75	0.20	3.6875	0.8442	0.7528	0.7500	0.2000	0.7490	0.1963
0.75	0.25	2.0000	0.9884	1.0711	0.7500	0.2500	0.7465	0.2482
0.75	0.30	1.0833	1.2568	1.5723	0.7500	0.3000	0.7531	0.2988
0.75	0.35	0.5306	1.8715	2.5882	0.7500	0.3500	0.7492	0.3521
0.75	0.40	0.1719	4.4021	6.4494	0.7500	0.4000	0.7459	0.4000
0.80	0.05	63.0000	0.8552	0.1805	0.8000	0.0500	0.8007	0.0496
0.80	0.10	15.0000	0.8983	0.3732	0.8000	0.1000	0.7954	0.1036
0.80	0.15	6.1111	0.9795	0.5955	0.8000	0.1500	0.7991	0.1498
0.80	0.20	3.0000	1.1204	0.8787	0.8000	0.2000	0.7941	0.2034
0.80	0.25	1.5600	1.3747	1.2914	0.8000	0.2500	0.8099	0.2428
0.80	0.30	0.7778	1.9077	2.0341	0.8000	0.3000	0.7957	0.2982
0.80	0.35	0.3061	3.5498	4.0975	0.8000	0.3500	0.8006	0.3478
0.85	0.05	50.0000	1.0605	0.2168	0.8500	0.0500	0.8491	0.0501
0.85	0.10	11.7500	1.1367	0.4502	0.8500	0.1000	0.8471	0.1010
0.85	0.15	4.6667	1.2822	0.7283	0.8500	0.1500	0.8479	0.1485
0.85	0.20	2.1875	1.5482	1.1097	0.8500	0.2000	0.8514	0.1964
0.85	0.25	1.0400	2.0923	1.7536	0.8500	0.2500	0.8498	0.2448
0.85	0.30	0.4167	3.6335	3.3601	0.8500	0.3000	0.8418	0.3061
0.85	0.35	0.0408	27.2675	26.2899	0.8500	0.3500	0.8605	0.3403
0.90	0.05	35.0000	1.3334	0.2875	0.9000	0.0500	0.9021	0.0489
0.90	0.10	8.0000	1.4947	0.6003	0.9000	0.1000	0.8948	0.1063
0.90	0.15	3.0000	1.8153	1.0032	0.9000	0.1500	0.8990	0.1541
0.90	0.20	1.2500	2.4970	1.6723	0.9000	0.2000	0.8993	0.2004
0.90	0.25	0.4400	4.5942	3.4426	0.9000	0.2500	0.8973	0.2529
0.95	0.05	18.0000	1.8224	0.4770	0.9500	0.0500	0.9504	0.0484
0.95	0.10	3.7500	2.3502	1.0206	0.9500	0.1000	0.9503	0.0999
0.95	0.15	1.1111	3.7162	2.0259	0.9500	0.1500	0.9513	0.1504
0.95	0.20	0.1875	12.7310	7.6750	0.9500	0.2000	0.9589	0.1788

APPENDIX 4D

Comparison of intended and realised variability from lognormalpower distribution.

The intended variability is given in the first three columns. Mean values were selected to be spaced between 0.5 and 1.0, which is the most likely range for large mammal's survival rate. Standard deviations were selected to cover most of the realistic range with each mean value. θ is the concentration parameter defined in section 5.6.9. The fourth and fifth columns give the parameter values specified to represent the intended variability. Since this specification is not exact, I calculated the mean and standard deviation of the distribution defined by those parameter values, and they are presented in columns 6 and 7. The last two columns give the variability of data generated by the distribution when the specified parameter values are used.

Intended variability			Specified parameters		variability represented by parameters		variability represented by variates	
mean	s	θ	μ	σ	mean	s	mean	s
0.50	0.05	99.0000	0.5011	0.1453	0.5000	0.0500	0.5016	0.0489
0.50	0.10	24.0000	0.5044	0.2969	0.5000	0.1000	0.4988	0.1007
0.50	0.15	10.1111	0.5098	0.4636	0.5000	0.1500	0.5054	0.1493
0.50	0.20	5.2500	0.5171	0.6589	0.5000	0.2000	0.4925	0.2020
0.50	0.25	3.0000	0.5261	0.9062	0.5000	0.2500	0.4984	0.2417
0.50	0.30	1.7778	0.5365	1.2490	0.5000	0.3000	0.5012	0.3027
0.50	0.35	1.0408	0.5475	1.7840	0.5000	0.3500	0.5019	0.3534
0.50	0.40	0.5625	0.5581	2.7940	0.5000	0.4000	0.4906	0.4017
0.50	0.45	0.2346	0.5667	5.6695	0.5000	0.4500	0.5059	0.4454
0.55	0.05	98.0000	0.5515	0.1530	0.5500	0.0500	0.5502	0.0496
0.55	0.10	23.7500	0.5561	0.3121	0.5500	0.1000	0.5495	0.1009
0.55	0.15	10.0000	0.5638	0.4859	0.5500	0.1500	0.5525	0.1469
0.55	0.20	5.1875	0.5746	0.6882	0.5500	0.2000	0.5538	0.2002
0.55	0.25	2.9600	0.5891	0.9433	0.5500	0.2500	0.5466	0.2481
0.55	0.30	1.7500	0.6082	1.2962	0.5500	0.3000	0.5486	0.3006
0.55	0.35	1.0204	0.6346	1.8484	0.5500	0.3500	0.5712	0.3490
0.55	0.40	0.5469	0.6761	2.9023	0.5500	0.4001	0.5470	0.4002
0.55	0.45	0.2222	0.7690	5.9940	0.5500	0.4500	0.5371	0.4504
0.60	0.05	95.0000	0.6020	0.1640	0.6000	0.0500	0.6002	0.0501
0.60	0.10	23.0000	0.6080	0.3339	0.6000	0.1000	0.6008	0.0990
0.60	0.15	9.6667	0.6179	0.5184	0.6000	0.1500	0.5995	0.1521
0.60	0.20	5.0000	0.6323	0.7328	0.6000	0.2000	0.6069	0.1946
0.60	0.25	2.8400	0.6520	1.0033	0.6000	0.2500	0.6048	0.2516
0.60	0.30	1.6667	0.6791	1.3804	0.6000	0.3000	0.6137	0.2932
0.60	0.35	0.9592	0.7185	1.9805	0.6000	0.3500	0.5858	0.3579
0.60	0.40	0.5000	0.7834	3.1681	0.6000	0.4000	0.6151	0.3978
0.60	0.45	0.1852	0.9131	7.1017	0.6000	0.4500	0.5912	0.4494
0.65	0.05	90.0000	0.6525	0.1793	0.6500	0.0500	0.6514	0.0494
0.65	0.10	21.7500	0.6601	0.3644	0.6500	0.1000	0.6495	0.1007
0.65	0.15	9.1111	0.6726	0.5644	0.6500	0.1500	0.6511	0.1513
0.65	0.20	4.6875	0.6906	0.7969	0.6500	0.2000	0.6491	0.2009
0.65	0.25	2.6400	0.7154	1.0926	0.6500	0.2500	0.6478	0.2494
0.65	0.30	1.5278	0.7499	1.5131	0.6500	0.3000	0.6477	0.2996
0.65	0.35	0.8571	0.8004	2.2070	0.6500	0.3500	0.6454	0.3495
0.65	0.40	0.4219	0.8803	3.6959	0.6500	0.4000	0.6463	0.3985
0.65	0.45	0.1235	0.9898	10.3124	0.6500	0.4500	0.6480	0.4522
0.70	0.05	83.0000	0.7032	0.2010	0.7000	0.0500	0.6985	0.0489
0.70	0.10	20.0000	0.7126	0.4069	0.7000	0.1000	0.7040	0.0982
0.70	0.15	8.3333	0.7280	0.6290	0.7000	0.1500	0.6963	0.1491
0.70	0.20	4.2500	0.7498	0.8884	0.7000	0.2000	0.6983	0.2021
0.70	0.25	2.3600	0.7796	1.2246	0.7000	0.2500	0.6930	0.2520
0.70	0.30	1.3333	0.8209	1.7197	0.7000	0.3000	0.7042	0.2992
0.70	0.35	0.7143	0.8794	2.5950	0.7000	0.3500	0.6962	0.3557
0.70	0.40	0.3125	0.9590	4.8070	0.7000	0.4000	0.6900	0.4034
0.70	0.45	0.0370	0.99999995	30.9117	0.6999	0.4497	0.7001	0.4499
0.75	0.05	74.0000	0.7541	0.2321	0.7500	0.0500	0.7481	0.0510
0.75	0.10	17.7500	0.7658	0.4681	0.7500	0.1000	0.7497	0.1021
0.75	0.15	7.3333	0.7844	0.7215	0.7500	0.1500	0.7486	0.1511
0.75	0.20	3.6875	0.8101	1.0219	0.7500	0.2000	0.7505	0.2024
0.75	0.25	2.0000	0.8446	1.4250	0.7500	0.2500	0.7413	0.2574
0.75	0.30	1.0833	0.8908	2.0587	0.7500	0.3000	0.7484	0.2963
0.75	0.35	0.5306	0.9497	3.3364	0.7500	0.3500	0.7501	0.3508
0.75	0.40	0.1719	0.9978	8.1247	0.7500	0.4000	0.7590	0.3928
0.80	0.05	63.0000	0.8053	0.2795	0.8000	0.0500	0.8010	0.0503
0.80	0.10	15.0000	0.8199	0.5597	0.8000	0.1000	0.8008	0.0978
0.80	0.15	6.1111	0.8420	0.8605	0.8000	0.1500	0.8019	0.1485
0.80	0.20	3.0000	0.8714	1.2281	0.8000	0.2000	0.7956	0.2046
0.80	0.25	1.5600	0.9089	1.7557	0.8000	0.2500	0.8005	0.2460
0.80	0.30	0.7778	0.9543	2.6974	0.8000	0.3000	0.8069	0.2950
0.80	0.35	0.3061	0.9942	5.2756	0.8000	0.3500	0.8001	0.3496
0.85	0.05	50.0000	0.8572	0.3584	0.8500	0.0500	0.8489	0.0517
0.85	0.10	11.7500	0.8754	0.7079	0.8500	0.1000	0.8459	0.1032
0.85	0.15	4.6667	0.9006	1.0869	0.8500	0.1500	0.8532	0.1459
0.85	0.20	2.1875	0.9317	1.5844	0.8500	0.2000	0.8542	0.1959
0.85	0.25	1.0400	0.9667	2.4092	0.8500	0.2500	0.8507	0.2435
0.85	0.30	0.4167	0.9953	4.4287	0.8500	0.3000	0.8529	0.2957
0.85	0.35	0.0408	0.99999999	17.9457	0.8474	0.3465	0.8442	0.3515
0.90	0.05	35.0000	0.9103	0.5102	0.9000	0.0500	0.9019	0.0489
0.90	0.10	8.0000	0.9321	0.9812	0.9000	0.1000	0.8977	0.1076
0.90	0.15	3.0000	0.9575	1.5248	0.9000	0.1500	0.9024	0.1491
0.90	0.20	1.2500	0.9829	2.3916	0.9000	0.2000	0.8951	0.2059
0.90	0.25	0.4400	0.9988	4.6217	0.9000	0.2500	0.8981	0.2531
0.95	0.05	18.0000	0.9653	0.8990	0.9500	0.0500	0.9502	0.0495
0.95	0.10	3.7500	0.9844	1.6720	0.9500	0.1000	0.9502	0.0990
0.95	0.15	1.1111	0.9974	2.9780	0.9500	0.1500	0.9491	0.1490
0.95	0.20	0.1875	0.99999988	9.2735	0.9494	0.1996	0.9471	0.2000

APPENDIX 5

Relationship between mean and standard deviation for differently shaped beta distributions

The shape of a beta distribution is determined by its parameters. The distribution is undefined if any of the shape parameters are negative. It is unimodal shaped when both parameters are higher than one. It becomes U shaped when both parameters are between zero and one, J shaped when one parameter is between zero and one the other is more than one (Evans et al. 1993). These results can be used to derive the relationship between the mean and standard deviation for differently shaped beta distributions.

(a) For any shaped beta distribution

The shape parameters for the beta distribution can be derived using mean and standard deviation as (formula A.3.2 in appendix 3)

$$\upsilon = \left(\frac{m-a}{b-a}\right)\left(\frac{(b-m)(\text{m-a})}{s^2}-1\right), \qquad \textbf{(A.5.1)}$$

$$\omega = \left(\frac{b-m}{b-a}\right)\left(\frac{(b-m)(\text{m-a})}{s^2}-1\right), \qquad \textbf{(A.5.2)}$$

where a, b, m and s are the minimum, maximum, mean, and standard deviation respectively. These formulas imply that the necessary and sufficient condition for beta distribution to be defined (i.e., for both shape parameters to be positive) is being

$$\left(\frac{m-a}{b-a}\right)\left(\frac{(b-m)(\text{m-a})}{s^2}-1\right)>0\ , \qquad \text{and} \qquad \left(\frac{b-m}{b-a}\right)\left(\frac{(b-m)(\text{m-a})}{s^2}-1\right)>0\,.$$

Since the first factor of each of these expressions is always positive, the required condition is that the second factor is positive.

$$\left(\frac{(b-m)(\text{m-a})}{s^2}-1\right)>0$$

This result gives the necessary and sufficient condition for the beta distribution as

$$s^2 < (b-m)(m-a) \qquad \textbf{(A.5.3)}$$

Note that this is the expression developed in appendix 1 for the maximum variability that cannot be exceeded under any distribution. This proves that beta distribution can produce a variability as large as that of any other distribution (an advantage of the

beta distribution). Therefore, if the variability we try to fit to a finite random variable is too large to handle in the beta distribution, it cannot be handled in any other distribution, and the combination of mean and standard deviation being tried is inappropriate. The maximum variance occurs when $m = \frac{a+b}{2}$. In this situation the above formula is reduced to $s < \frac{b-a}{2}$, giving the maximum possible variability in the range between a and b. When $a = 0$ and $b = 1$ this results in $s < \frac{1}{2}$. This is the maximum standard deviation that the survival rate (or any other variable with a unit range) can have under the beta distribution (or any other distribution). Figure A.5.1 summarises these (and other) results.

(b) For unimodally shaped beta distributions

Formulas A.5.1 and A.5.2 imply that the pdf is unimodal (i.e., both shape parameters are larger than one) only when

$$\frac{(m-a)^2(b-m)}{(b+m-2a)} > s^2 \quad \text{and} \quad \frac{(b-m)^2(m-a)}{(2b-m-a)} > s^2.$$

Therefore the necessary and sufficient condition for the unimodality of a beta distribution is

$$s^2 < \min\left(\frac{(m-a)^2(b-m)}{(b+m-2a)}, \frac{(b-m)^2(m-a)}{(2b-m-a)}\right). \qquad \textbf{(A.5.4)}$$

The maximum variance occurs when $m = \frac{a+b}{2}$. In this situation the above condition is reduced to $s^2 < \frac{(b-a)^2}{12}$. When $a = 0$ and $b = 1$ this again reduces to $s < \frac{1}{\sqrt{12}}$. Therefore the standard deviation of any unimodal beta distribution over the unit interval will never exceeds $\frac{1}{\sqrt{12}}$.

Any unimodal distribution over a finite distribution, irrespective of which distribution it follows, asymptotically becomes closer to the uniform distribution with increasing variability. Since the standard deviation of the unit uniform distribution is $\frac{1}{\sqrt{12}}$, the above result implies that the unimodal beta distribution can have a standard deviation as large as any other unimodal distribution when $m = \frac{a+b}{2}$.

(c) For U shaped beta distributions

Using similar steps as in (b) above, the necessary and sufficient condition for U shaped beta distribution can be derived from formulas A.5.1 and A.5.2 as

$$\max\left(\frac{(m-a)^2(b-m)}{(b+m-2a)},\frac{(b-m)^2(m-a)}{(2b-m-a)}\right)<s^2<(b-m)(m-a). \qquad \textbf{(A.5.5)}$$

When a = 0 and b = 1, (i.e., the case of survival rate), the above condition simplifies to

$$\max\left(\frac{m^2(1-m)}{(1+m)},\frac{(1-m)^2m}{(2-m)}\right)<s^2<(1-m)m \qquad \textbf{(A.5.6)}$$

As shown earlier, the highest value that this upper limit can take is 1/2. The lowest value that the lower limit can take is zero which occurs when m = 0 or when m = 1. Therefore, the standard deviation can vary from zero to1/2 for U shaped beta distributions over the unit interval.

(d) For J shaped beta distributions

For a beta distribution to be J shaped, one of the shape parameters needs to be between zero and one while the other is more than one. From equations A.5.1 and A.5.2, this condition can be written as;

$$0<\left(\frac{m-a}{b-a}\right)\left(\frac{(b-m)(\text{m-a})}{s^2}-1\right)<1 \quad \text{and} \quad 1<\left(\frac{b-m}{b-a}\right)\left(\frac{(b-m)(\text{m-a})}{s^2}-1\right)$$

or

$$1<\left(\frac{m-a}{b-a}\right)\left(\frac{(b-m)(\text{m-a})}{s^2}-1\right) \quad \text{and} \quad 0<\left(\frac{b-m}{b-a}\right)\left(\frac{(b-m)(\text{m-a})}{s^2}-1\right)<1.$$

Taking $p=\dfrac{m-a}{b-a}$ and by rearranging, the above requirement can be rephrased as;

$$\frac{p(1-p)^2}{2-p}<\left(\frac{s}{b-a}\right)^2<\frac{p^2(1-p)}{1+p} \quad \text{or} \quad \frac{p^2(1-p)}{1+p}<\left(\frac{s}{b-a}\right)^2<\frac{p(1-p)^2}{2-p}.$$

This implies that the necessary and sufficient condition for beta distribution to be J shaped is:

$$\min\left(\frac{p(1-p)^2}{2-p},\frac{p^2(1-p)}{1+p}\right)<\left(\frac{s}{b-a}\right)^2<\max\left(\frac{p(1-p)^2}{2-p},\frac{p^2(1-p)}{1+p}\right). \qquad \textbf{(A.5.7)}$$

Therefore both the maximum possible standard deviation and the minimum possible standard deviation of a J shaped distribution approach zero when p approaches zero or

one (i.e., when mean approaches either a or b). When $m = \frac{a+b}{2}$ (i.e., when p = 0.5) both the minimum possible and maximum possible standard deviations become $\frac{b-a}{\sqrt{12}}$. This is same as the maximum possible standard deviation of a unimodal distribution (see Figure A.5.1). Unlike in unimodal distributions, the maximum standard deviation does not occur at $m = \frac{a+b}{2}$ in J shaped distributions. At this level of the mean a unimodal distribution becomes U shaped with increasing standard deviation without going through to J shape. At other levels of the mean a unimodal distribution first becomes J shaped and then becomes U shaped with increasing mean.

(e) For symmetric beta distributions

The coefficient of skewness of the beta distribution is given by $\frac{2(\omega - \nu)(\nu + \omega + 1)^{1/2}}{(\nu + \omega + 2)(\nu\omega)^{1/2}}$ (Evans et al. 1993). Therefore, the skewness is zero (i.e., distribution is symmetric) only when $\omega = \nu$. Using formulas A.5.1 and A.5.2, this can be translated to say, the beta distribution is symmetric (given that it is defined) when $m = \frac{a+b}{2}$.

(f) For uniform shaped beta distributions

The beta distribution is uniform shaped when both shape parameters are unity (Evans et al. 1993). Therefore, using the formula A.3.1, the necessary and sufficient condition for beta distribution to be uniform shaped is $s = \sqrt{\frac{b-a}{12}}$ and $m = \frac{a+b}{2}$.

Summary

The relationships between mean and standard deviation for differently shaped beta distributions as derived above are summarized in Figure A.5.1. Specifically, it shows the relationships given in formulas A.5.3 to A.5.7, for beta distributions over the unit interval. The curves in the figure demarcate five distinct regions- each region includes the combinations of mean and standard deviation of a particular shaped distribution (two separate regions belong to the J shape).

As seen from this figure, $s < \frac{1}{\sqrt{12}}$ for any unimodal standard beta distribution, and it could be larger for J shaped and U shaped distributions. For U shaped standard beta distributions $s < \frac{1}{2}$. Therefore if $0 < s < \frac{1}{\sqrt{12}}$ the distribution can take unimodal shape, J shape, or U shape depending on the value of the mean, while if $\frac{1}{\sqrt{12}} < s$ the distribution never be unimodal. When $\frac{1}{\sqrt{12}} < s < \frac{1}{2}$ the distribution is either J shaped or U shaped depending on the value of the mean[1]. These ranges can be converted to any other (non-standard) beta distribution by multiplying them by $(b-a)$, (Johnson et al. 1994a).

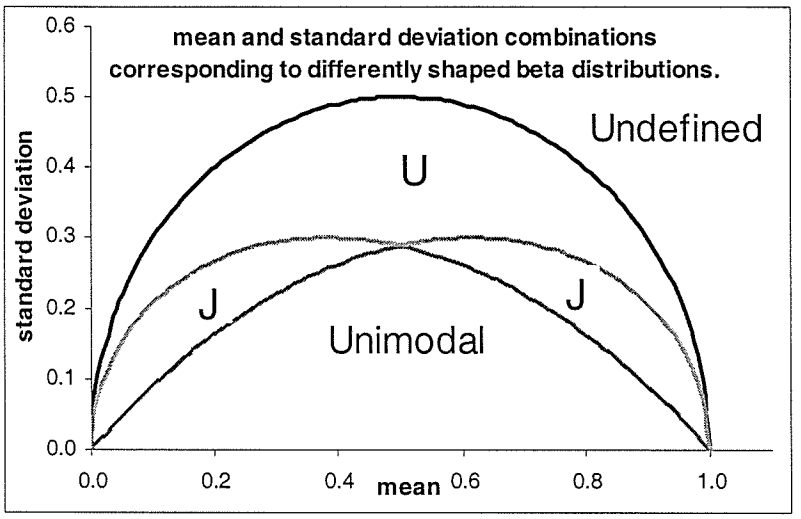


Figure A.5.1 Relationship between the mean and standard deviation for differently shaped standard beta distributions. The beta distribution is not defined for any combination of mean and standard deviation that falls outside the half-circle area. The four sub areas in the half-circle area show the combinations of mean and standard deviations correspond to unimodal, J shaped and U shaped beta distributions as marked, with J shape having two distinct areas.

[1] Pham-Gia (1994, p2179) established some simple bounds for the variance of standard beta distribution. Specifically, it is shown that if standard beta distribution is defined then $s < 1/2$, and if unimodel then $s < 1/\sqrt{12}$. Those findings agree with this appendix. However, it further reports that if beta distribution is U shaped then $s > 1/\sqrt{12}$. This result does not agree with this appendix.

APPENDIX 6A

The interrelationship between two methods used to investigate the stability at equilibrium population density

The first method of investigating stability uses the derivative of N_{t+1} with respect to N_t , while the second method uses derivative of k_t with respect to $\ln(N_t)$, both evaluated at equilibrium population density K. The interrelationship between these two quantities can be shown as follows.

The derivative of K_t with respect to $\ln(N_t)$ can be specified as

$$\frac{dk_t}{d\ln(N_t)} = \frac{d(-\ln[f(N_t)])}{d\ln(N_t)} = \frac{-d\ln[f(N_t)]}{dN_t}\frac{dN_t}{d\ln(N_t)} = -N_t\frac{d\ln[f(N_t)]}{dN_t}$$

$$= -N_t\frac{d\ln[f(N_t)]}{df(N_t)}\frac{df(N_t)}{dN_t} = \frac{-N_t f'(N_t)}{f(N_t)}$$

Using the condition of $f(N_t)=1$ at N=K,

$$\left.\frac{dk_t}{d\ln(N_t)}\right|_K = -Kf'(K)$$

Similarly, the derivative of N_{t+1} with respect to N_t can be specified as

$$\frac{dN_{t+1}}{dN_t} = \frac{d}{dN_t}(f(N_t)N_t) = N_t f'(N_t) + f(N_t)$$

Using the condition of $f(N_t)=1$ when N=K,

$$\left.\frac{dN_{t+1}}{dN_t}\right|_K = Kf'(K)+1$$

Therefore,

$$\left.\frac{dN_{t+1}}{dN_t}\right|_K + \left.\frac{dk_t}{d\ln(N_t)}\right|_K = 1.$$

Therefore, the two quantities used in the two methods adds to unity.

APPENDIX 6B

Stability properties at multiple time steps

We can generalise the definition of one-period stability to p-time period by saying that a population is locally stable at p-time period at the population level of $N_{K,p}$ if

$$N_{t+p} = N_t, \quad \text{and} \quad \left| \left. \frac{dN_{t+p}}{dN_t} \right|_{N_{K,p}} \right| < 1.$$

May (1976) showed that,

$$\left. \frac{dN_{t+2}}{dN_t} \right|_{N_{K,2}} = \left(\left. \frac{dN_{t+1}}{dN_t} \right|_{N_{K,2}} \right)^2.$$

This relationship can be generalised to a p-time period as

$$\left. \frac{dN_{t+p}}{dN_t} \right|_{N_{K,p}} = \left(\left. \frac{dN_{t+1}}{dN_t} \right|_{N_{K,p}} \right)^p.$$

Using this, we can redefine the condition for stability at p-time period as, population is locally stable at p-time period at the population level of $N_{K,p}$ if

$$N_{t+p} = N_t, \quad \text{and} \quad \left| \left(\left. \frac{dN_{t+1}}{dN_t} \right|_{N_{K,p}} \right)^p \right| < 1.$$

Also note that $\left| \left(\left. \frac{dN_{t+1}}{dN_t} \right|_{N_{K,p}} \right)^p \right| < 1$ if and only if $\left| \left(\left. \frac{dN_{t+1}}{dN_t} \right|_{N_{K,p}} \right) \right| < 1$.

Therefore, the stability of the population at time period p at $N_{K,p}$ can be examined analytically by evaluating $\left(\left. \frac{dN_{t+1}}{dN_t} \right|_{N_{K,p}} \right)^p$ and using the criterion for a single-time-period given in Table 6.1. Therefore any population that is stable (monotonic damping or oscillatory damping) at period one is also stable at any higher time periods. Similarly if it is unstable at period one it is unstable at other periods too.

APPENDIX 7

Population stability and asymmetric growth in demographic models

This appendix shows that the representation of population stability and asymmetric growth characteristics by density dependent functions is not confined to count-based models - they are applicable to all unstructured models (i.e., to both count-based models and unstructured demographic models) provided that the entire density dependence acts through a single vital rate. For this illustration I use the logistic function as an example.

First consider the asymmetric growth property in an unstructured demographic model. Suppose if only the survival rate is density dependent, the population multiplication rate at time t is $\lambda_t = S_t(1+R)$, where $S_t = d\left(1-\frac{N_t}{a}\right)$, and R is the density independent reproductive rate. Therefore,

$$\lambda_t = d\left(1-\frac{N_t}{a}\right)(1+R).$$

Note that d and a are different from those in count-based models. Since $\lambda_t = 1$ at the carrying capacity K,

$$1 = d\left(1-\frac{K}{a}\right)(1+R).$$

This gives the carrying capacity, in terms of the model parameters, as

$$K = \frac{a[d(1+R)-1]}{d(1+R)}.$$

Since the population dynamics model becomes

$$N_{t+1} = \lambda_t N_t = d\left(1-\frac{N_t}{a}\right)(1+R)N_t,$$

the population growth is given by $N_t\left[(1+R)d\left(1-\frac{N_t}{a}\right)-1\right]$.

By differentiating this we can find the N at which the growth is maximum; i.e.,

$$N^* = \frac{a[d(1+R)-1]}{2d(1+R)} = \frac{K}{2}.$$

$N^* = \frac{K}{2}$ is what we observed in section 7.2 for the count-based model version of the logistic function, and so the symmetric growth property is represented in this demographic model in the same way as it is in the count-based model.

Now consider the stability property of the same demographic population model under the same DD function:

$$N_{t+1} = d\left(1 - \frac{N_t}{a}\right)(1+R)N_t \cdot$$

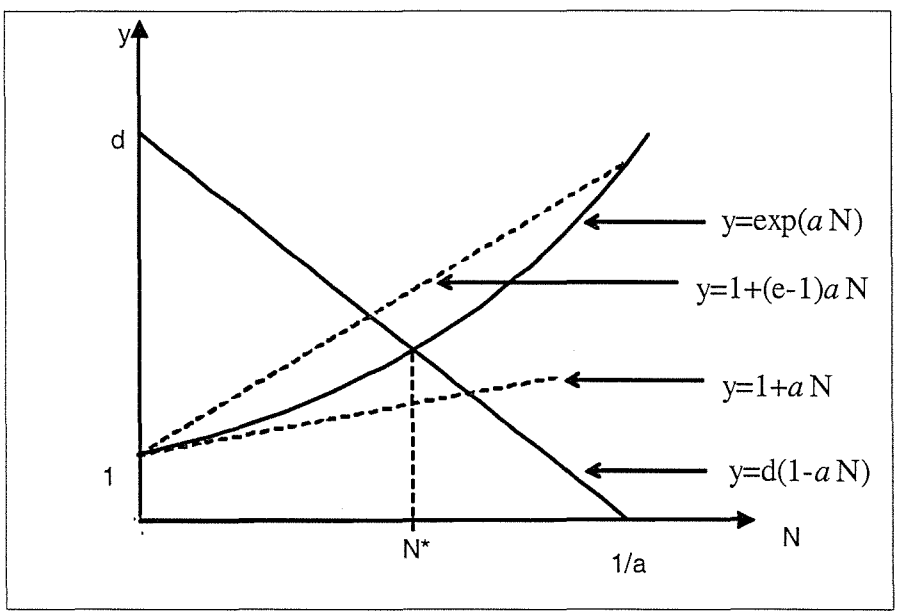
Because $\left.\frac{dN_{t+1}}{dN_t}\right|_K = 2 - d(1+R)$, according to the criterion in Table 6.1, the population is stable at K if and only if $-1 < \{2 - d(1+R)\} < 1$, or equivalently, if $1 < d(1+R) < 3$. The corresponding condition obtained in section 7.2 for the count-based model under the same DD function was $1 < d < 3$. Noting that the parameter d in a count-based model is comparable to $d(1+R)$ in a demographic model, the conditions in the two models are equivalent.

Even though I assumed here the density dependence is represented through survival rate, the same results can be obtained by assuming DD is represented by the reproductive rate. Therefore, the representation of asymmetric growth and stability characteristics by DD functions is not limited to count-based models; it is applicable to all unstructured models where the density dependence is represented by a single vital rate.

APPENDIX 8

Population level corresponds to maximum productivity under the Richer function

It can be seen by taking the first derivative of the expression for the population growth ($N_{t+1} - N_t$) with respect to N_t that the growth becomes maximum at the population size that satisfies the condition $d(1-aN) = e^{aN}$. Since this expression cannot be solved for N analytically, this appendix finds a narrow range that the solution is confined to. If we consider $y = \exp(aN)$ and $y = d(1-aN)$ relationships, the solution (N^*) is the intersection point as shown in the figure.



If the first-order Taylor series approximation for $y = \exp(aN) \approx 1 + aN$ is used, at the intersection point

$$1 + aN = d(1-aN).$$

Therefore, $N = \dfrac{d-1}{a(d+1)} = \dfrac{d-1}{a(d-1+2)}$

$$\approx \frac{\ln(d)}{a\{\ln(d)+2\}}, \text{ if } d \text{ is near one}$$

$$= \frac{K}{2+\ln(d)} \text{ where K is the equilibrium density given by } K = \frac{\ln(d)}{a}.$$

As seen from above sketch this will be an overestimation for N^*.

If we use a straight line from N=0 to $N = 1/a$ to approximate $y = \exp(aN)$, that line is given by $y = 1 + (e-1)aN$. Therefore at the intersection

$$1 + (e-1)aN = d(1 - aN).$$

Therefore, $N = \dfrac{d-1}{a(e+d-1)}$

Under the assumption of $d \approx 1$ (a somewhat reasonable assumption for long-lived species),

$$N \approx \frac{\ln(d)}{a[e+\ln(d)]} = \frac{K}{e+\ln(d)}.$$

This will be an underestimation for N^* as shown in above sketch. Therefore,

$$\frac{K}{e+\ln(d)} < N^* < \frac{K}{2+\ln(d)}.$$

This solution is valid only for populations which $d \approx 1$ only. The interval obtained without this assumption is

$$\frac{d-1}{a(e+d-1)} < N^* < \frac{d-1}{a(d+1)}.$$

None of these statements imply that N^* can vary anywhere between these two boundaries; rather they mean that N* will not go beyond these ranges.

APPENDIX 9

Determine the shape of the MS&S function from its functional form

For MS&S function $\frac{df(N)}{dN} = \frac{-dab(aN)^{b-1}}{\left(1+(aN)^b\right)^2}$, which is negative for all values of N.

$\frac{d^2 f(N)}{dN^2} = a^2 b(aN)^{b-1}\left[\frac{2b(aN)^{b-1}}{[1+(aN)^b]^3} - \frac{(b-1)}{[1+(aN)^b]^2 aN}\right]$. This can shown to be proportional to, and has the same sign as $(aN)^b(b+1)-(b-1)$.

Suppose $0<b<1$, $\lim_{N\to 0}\frac{df(N)}{dN} = -\infty$, $\lim_{N\to\infty}\frac{df(N)}{dN} = 0$, and $(aN)^b(b+1)-(b-1)$ is positive for any value of N. Therefore the function has a monotonically decreasing concave-up shape, and so it does not have a sigmoid shape.

However, if $b>1$, $\lim_{N\to 0}\frac{df(N)}{dN} = 0$, and $\lim_{N\to\infty}\frac{df(N)}{dN} = 0$. In this case, the sign of the expression $(aN)^b(b+1)-(b-1)$ depends on the level of N. It is zero at $N = \frac{1}{a}\left(\frac{b-1}{b+1}\right)^{1/b}$, is positive if N is larger than this value, and negative if N is smaller. Therefore $N = \frac{1}{a}\left(\frac{b-1}{b+1}\right)^{1/b}$ is an inflection point. In this case the function takes a sigmoid shape.

The inflection point can be given in terms of K as $N = K\left(\frac{(b-1)}{(d-1)(b+1)}\right)^{1/b}$.

APPENDIX 10

Form of competition represented by the MS&S function

For the MS&S function, the second derivative of the kvalue with respect to N is $\frac{a^2 b(aN)^{b-2}}{1+(aN)^b}\left((b-1)-\frac{b(aN)^b}{1+(aN)^b}\right)$. Since the term outside the brackets is positive, the sign of this expression is influenced only by the term inside the brackets, which can be simplified to $\frac{(b-1)-(aN)^b}{1+(aN)^b}$. This is positive if and only if $(b-1)>(aN)^b$. This condition cannot be satisfied if $b < 1$, so scramble competition is impossible.

When $b > 1$, this expression cannot be simplified to determine which level of b leads to positive and negative values, because it depends on N. Therefore we do not know which values of b corresponds to contest and scramble competitions. The above expression says, the second derivative is positive if $N < \frac{(b-1)^{1/b}}{a}$, so the scramble competition is represented at those population levels, and contest competition is represented at higher population levels. Thus the type of competition changes as the population grows.

$N = \frac{(b-1)^{1/b}}{a}$ may not exists when $b < 1$, implying that the sign of above second derivative remains unchanged. Therefore only one kind of competition (i.e., only contest from workings in section 7.8.2) can be represented. This could be another reason why Getz (1996) imposed the condition that $b > 1$.

We can compare the population size at which scramble competition turns into contest with the carrying capacity of $K = \frac{(d-1)^{1/b}}{a}$. They are the same if $b = d$. Therefore if $b > d$ scramble competition is active until population passes K. If $b < d$ scramble competition becomes contest at a population level below K.

APPENDIX 11

Obtaining curves with the same level of DD from various functions

This appendix shows how to obtain DD curves for different DD functions with the same level of DD. Different functions are said to have the 'same level of DD' if the vital rate is the same under all functions at three specified levels of N, for example at the lowest population level of $N = 0$, at an observed population level of $N = N'$, and at the largest expected level of population at $N = qN'$, (using the notation defined in the main text, section 8.2).

The functions of interest in section 8.2 of the main text are;

θ–Logistic function $S = d\left\{1-\left(N/a\right)^b\right\}$

θ-Ricker function $S = d\exp(-aN^b)$

MS&S function $S = d\left\{1+(aN)^b\right\}^{-1}$

Usher function $S = d\left\{1+\exp(bN-a)\right\}^{-1}$

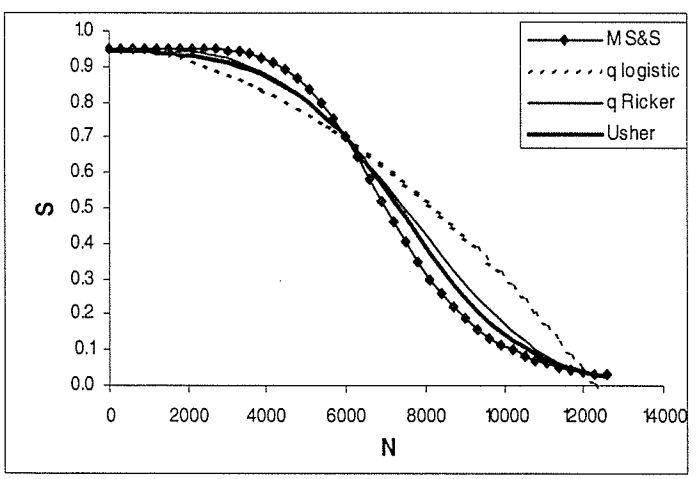


Figure:A.11.1 An example showing four DD curves crossing at three specified points. Here $d = 0.95$, $N' = 6000$, $S' = 0.7$, $q = 2$, and parameter b for MS&S function is 6. All other parameters (for all functions) were calculated from this information.

Let us consider any one single vital rate S (could be any of S_1, S_2, S_3 or R from the population model used in section 8.3 of the main text), and say the DD curve of that rate is fully defined under one of the DD functions. This can be done by specifying

two of its parameters (d and b_1) and a point on its curve (N', S'). What we need is to derive DD curves for all the other functions in such a way that they all have the same level of DD. For that we need to calculate parameters for each of the other functions. The parameter d is common to all, so we only need to find two parameters (say a_2 and b_2) for each function. This appendix shows how those two parameters can be calculated.

The general case

Taking two functions at a time, at any population size (N) where the vital rate is same under both functions,

$$f_1(d, a_1, b_1, N) = f_2(d, a_2, b_2, N)$$

where f_1 and f_2 are the two DD functions, subscript 1 and 2 in parameters denote the first and second function respectively. (In this notation I did not distinguish parameters from random variable N because N is used here as a fixed constant value). We can ignore the parameter d as it is a common multiplier (i.e., both curves pass through (0, d) point). Therefore

$$f_1(a_1, b_1, N) = f_2(a_2, b_2, N)$$

At the population levels of N=N' and N=qN',

$$f_1(a_1, b_1, N') = f_2(a_2, b_2, N'), \qquad \text{and} \qquad f_1(a_1, b_1, qN') = f_2(a_2, b_2, qN').$$

These two equations can be solved for a_2 and b_2 if quantities a_1, b_1, N', and q can be specified. These equations can be solved analytically for most DD functions, including all the functions considered in chapter 8. Numerical methods could be used if no analytical solutions exist for a given pair of functions.

Specific cases for individual functions

Now I show how analytical solutions can be found for the functions used in our case study in section 8.3. In the following explanation, I take the MS&S function to be f_1 to derive parameters for each of the other functions in terms of its parameters. Parameters d and b_1 for the MS&S function, and quantities N' and S', q are assumed to be specified. Then, for the MS&S function, $S' = d\left\{1 + (a_1 N')^{b_1}\right\}^{-1}$, so

$$a_1 = \frac{1}{N'}\left(\frac{d - S'}{S'}\right)^{1/b_1}.$$

All three parameters are now specified for this function, and it remains to derive the parameters for each of the other functions.

(a) Parameters for the θ-logistic function

The aim here is to find the two unknown parameters for the θ-logistic function (a_2 and b_2) assuming the parameters for the MS&S function (d, a_1, and b_1) are known (or have been specified). At any population level (N) where both functions have the same level of vital rate

$$d\left\{1+(a_1N)^{b_1}\right\}^{-1} = d\left\{1-\left(N/a_2\right)^{b_2}\right\}.$$

Therefore, $a_2 = N\left[\dfrac{1+(a_1N)^{b_1}}{(a_1N)^{b_1}}\right]^{1/b_2}$

At $N = N'$ and $N = qN'$ where the vital rate under both functions are the same,

$$a_2 = N'\left[\frac{1+(a_1N')^{b_1}}{(a_1N')^{b_1}}\right]^{1/b_2} \qquad \textbf{(A.11.1)}$$

and

$$a_2 = qN'\left[\frac{1+(a_1qN')^{b_1}}{(a_1qN')^{b_1}}\right]^{1/b_2} \qquad \textbf{(A.11.2)}$$

From these two formulas,

$$b_2 = \left(\frac{1}{\ln(q)}\right)\ln\left[q^{b_1}\left(\frac{1+(a_1N')^{b_1}}{1+(a_1qN')^{b_1}}\right)\right] \qquad \textbf{(A.11.3)}$$

By substituting this to equation A.11.1 we can obtain parameter a_2.

(b) Parameters for the θ-Ricker function

Here we find the two unknown parameters for the θ-Ricker function (a_2 and b_2) assuming parameters for MS&S function (d, a_1, and a_2) are specified. At any point where both functions have the same level of S

$$d\left\{1+(a_1N)^{b_1}\right\}^{-1} = d\exp(-a_2N^{b_2}).$$

Therefore

$$a_2 = \frac{\ln\left\{1+(a_1N)^{b_1}\right\}}{N^{b_2}}$$

At the population levels of $N = N'$ and $N = qN'$

$$a_2 = \frac{\ln\left\{1+(a_1 N')^{b1}\right\}}{N'^{b_2}} \qquad \textbf{(A.11.4)}$$

and

$$a_2 = \frac{\ln\left\{1+(a_1 qN')^{b_1}\right\}}{(qN')^{b_2}} . \qquad \textbf{(A.11.5)}$$

From these two expressions

$$b_2 = \left(\frac{1}{\ln(q)}\right)\ln\left[\frac{\ln\left\{1+(a_1 qN')^{b_1}\right\}}{\ln\left\{1+(a_1 N')^{b_1}\right\}}\right] . \qquad \textbf{(A.11.6)}$$

By substituting this into equation A.11.4 we get a formula for a_2.

(c) Parameters for the Usher function

We now find parameters for the Usher function (a_2 and b_2) assuming that parameters for the MS&S function (d, a_1, and b_1) can be specified. At any population level where both functions have the same level of vital rate

$$d\left\{1+(a_1 N)^{b_1}\right\}^{-1} = d\left\{1+\exp(b_2 N - a_2)\right\}^{-1} .$$

Therefore,

$$a_2 = b_2 N - b_1 \ln(a_1 N)$$

Hence, at the points when $N=N'$and $N=qN'$ where the vital rate is the same under both functions,

$$a_2 = b_2 N' - b_1 \ln(a_1 N') \text{ , and}$$

$$a_2 = b_2 qN' - b_1 \ln(a_1 qN')$$

From these two expressions we get

$$b_2 = \frac{b_1 \ln(q)}{N'(q-1)} \qquad \textbf{(A.11.7)}$$

and so

$$a_2 = b_1\left(\frac{\ln(q)}{q-1} - \ln(a_1 N')\right) \qquad \textbf{(A.11.8)}$$

APPENDIX 12

Estimating the parameters for DD functions at each time step for IDD models

For the θ-logistic, θ-Ricker, MS&S and Usher functions

We assume that the following information is known or has been specified.

1. The value of the vital rate at the lowest density (d_S for survival, d_R for reproduction),
2. The value of vital rate at a known density (S=S' at $N = N_S^{'}$ for survival, R=R' at N= $N_R^{'}$ for reproduction,
3. The carrying capacity of the population (K).

The parameters that have to be calculated from the above information are:

Two DD parameters for the DD curve of each vital rate (a_S and b_S for survival curve and a_R and b_R for reproductive curve). These curves are time-specific, as are the parameters.

For the logistic function

The information that is assumed to be known or to have been specified:

1. The value of vital rate at the lowest density (d_S for survival, d_R for reproduction),
2. The carrying capacity of the population (K).

The parameters that have to be calculated are:

One DD parameter for the DD relationship of each vital rate (a_S for survival rate and a_R for reproductive rate). These relationships are time specific, so as the parameters.

(A) θ-logistic function

(i) Prebreeding census model

In prebreeding models reproduction occurs prior to survival. Under the θ-logistic function, the density dependent reproductive rate is given by

$$R_t = d_R\left[1-\left(\frac{N_t}{a_R}\right)^{b_R}\right],$$

where a_R and b_R are the unknown parameters for the function. Using the specified point of $R_t = R'$ at $N_t = N_R^{'}$ on the reproductive curve and rearranging we obtain

$$a_R = N_R^{'}\left(\frac{d_R}{d_R - R'}\right)^{1/b_R} . \qquad \textbf{(A.12.1)}$$

Using the relationship $\lambda_t = S_{t-1}(1+R_t)$,

$$\lambda_t = S_{t-1}\left[1+d_R\left\{1-\left(\frac{N_t}{a_R}\right)^{b_R}\right\}\right]$$

Note that the survival rate of S_{t-1} is used here, not S_t, because under IDD models any vital rate applicable at time t is determined by the level of the other vital rates immediately prior to time t.

Since $\lambda_t = 1$ at $N_t = K$

$$1 = S_{t-1}\left[1+d_R\left\{1-\left(\frac{K}{a_R}\right)^{b_R}\right\}\right],$$

and by rearranging this we find a_R to be

$$a_R = K\left[\frac{S_{t-1}d_R}{S_{t-1}(1+d_R)-1}\right]^{1/b_R} . \qquad \textbf{(A.12.2)}$$

Using A.12.1 and A.12.2 we can now obtain the parameter b_R , which is given by

$$b_R = \frac{1}{\ln(N_R^{'})-\ln(K)}\ln\left[\frac{S_{t-1}(d_R - R')}{S_{t-1}(1+d_R)-1}\right], \qquad \textbf{(A.12.3)}$$

and substituting this in to A.12.1 gives the parameter a_R. The DD curve defined by these parameters determines the reproductive rate (R_t) for the current year.

The survival event occurs after reproduction in prebreeding models. The density dependent survival under the θ–logistic function follows the relationship

$$S_t = d_s\left[1-\left(\frac{N_t}{a_s}\right)^{b_s}\right],$$

where the parameters a_S and b_S need to be written in terms of the specified information. Using the specified point of $S_t = S'$ at $N_t = N_s^{'}$ on the survival curve, and rearranging we get

$$a_s = N_s^{'}\left(\frac{d_s}{d_s - S'}\right)^{1/b_s} \quad . \qquad \textbf{(A.12.4)}$$

Using the relationship $\lambda_t = S_t(1+R_t)$,

$$\lambda_t = d_s\left[1-\left(\frac{N_t}{a_s}\right)^{b_s}\right](1+R_t)\cdot$$

Since $\lambda_t = 1$ at $N_t = K$

$$1 = d_s\left[1-\left(\frac{K}{a_s}\right)^{b_s}\right](1+R_t), \text{ so}$$

$$a_s = K\left[\frac{d_s(1+R_t)}{d_s(1+R_t)-1}\right]^{1/b_s} \qquad \textbf{(A.12.5)}$$

Using A.12.4 and A.12.5 we can now write the parameter b_s as

$$b_s = \frac{1}{\ln(N_S^{'})-\ln(K)}\ln\left[\frac{(1+R_t)(d_s - S')}{d_s(1+R_t)-1}\right] \qquad \textbf{(A.12.6)}$$

Substituting this into A.12.4 gives the parameter a_s. The DD curve defined by these parameters determines the survival rate (S_t) for the year.

(ii) Postbreeding census model

The derivation of the formulae for parameters of the postbreeding census model follows the same steps. As shown in the main text (section 8.4.3) these formulae are

$$b_s = \frac{1}{\ln(N_S^{'})-\ln(K)}\ln\left[\frac{(1+R_{t-1})(d_s - S')}{d_s(1+R_{t-1})-1}\right], \qquad a_s = N_s^{'}\left(\frac{d_s}{d_s - S'}\right)^{1/b_s}$$

$$b_R = \frac{1}{\ln(N_R^{'})-\ln(K)}\ln\left[\frac{S_t(d_R - R')}{S_t(1+d_R)-1}\right], \qquad a_R = N_R^{'}\left(\frac{d_R}{d_R - R'}\right)^{1/b_R} .$$

(B) θ-Ricker function

(i) Prebreeding census model

Under the θ-Ricker function, the density dependent reproductive rate is given by

$$R_t = d_R \exp(-a_R N_t^{b_R}),$$

where a_R and b_R are the unknown parameters of the function. Using the fixed point of $R_t = R'$ at $N_t = N_R^{'}$ and rearranging we find a_R to be

$$a_R = \frac{1}{(N_R^{'})^{b_R}} \ln\left(\frac{d_R}{R^{'}}\right) . \qquad \textbf{(A.12.7)}$$

Using the relationship $\lambda_t = S_{t-1}(1+R_t)$,

$$\lambda_t = S_{t-1}\{1+d_R \exp(-a_R N_t^{b_R})\}.$$

Note that, S_{t-1} is used here (not S_t) as it is the relevant survival rate.

Since $\lambda_t = 1$ at $N_t = K$

$$1 = S_{t-1}\{1+d_R \exp(-a_R K_t^{b_R})\}, \text{ so}$$

$$a_R = \frac{1}{K^{b_R}} \ln\left(\frac{S_{t-1} d_R}{1-S_{t-1}}\right) . \qquad \textbf{(A.12.8)}$$

Using A.12.7 and A.12.8 we can write the parameter b_R as

$$b_R = \frac{1}{\ln(K)-\ln(N_R^{'})} \ln\left[\frac{\ln(S_{t-1}d_R)-\ln(1-S_{t-1})}{\ln(d_R)-\ln(R^{'})}\right], \qquad \textbf{(A.12.9)}$$

And substituting this in to A.12.7 gives the parameter a_R. The DD curve defined by these parameters determines the reproductive rate (R_t) for the year.

In prebreeding models, survival occurs after reproduction. The density dependent survival under the θ–Ricker function follows

$$S_t = d_s \exp(-a_S N_t^{b_S}),$$

where parameters a_S and b_S need to be calculated. Using the fixed point of $S_t = S'$ at $N_t = N_S^{'}$ on the survival curve, and rearranging we get

$$a_S = \frac{1}{(N_S^{'})^{b_S}} \ln\left(\frac{d_S}{S^{'}}\right). \qquad \textbf{(A.12.10)}$$

Using the relationship $\lambda_t = S_t(1+R_t)$,

$$\lambda_t = (1+R_t) d_S \exp(-a_S N_t^{b_S}),$$

and since $\lambda_t = 1$ *at* $N_t = K$,

$$1 = (1+R_t) d_S \exp(-a_S K^{b_S}),$$

By rearranging this we get

$$a_S = \frac{1}{K^{b_S}} \ln\{d_S(1+R_t)\}. \qquad \textbf{(A.12.11)}$$

Equations A.12.10 and A.12.11 imply that

$$b_S = \frac{1}{\ln(K) - \ln(N_S^{'})} \ln\left[\frac{\ln[d_S(1+R_t)]}{\ln(d_S) - \ln(S^{'})}\right], \qquad \textbf{(A.12.12)}$$

and substituting this in to A.12.10 gives the parameter a_S. The DD curve defined by these parameters determines the survival rate (S_t) for the year.

(ii) Postbreeding census model

The derivation of the formulae for the parameters of the postbreeding census model follows the same steps. These formulae are

$$b_S = \frac{1}{\ln(K) - \ln(N_S^{'})} \ln\left[\frac{\ln\{d_S(1+R_{t-1})\}}{\ln(d_S) - \ln(S^{'})}\right], \qquad a_S = \frac{1}{(N_S^{'})^{b_S}} \ln\left(\frac{d_S}{S^{'}}\right),$$

$$b_R = \frac{1}{\ln(K) - \ln(N_R^{'})} \ln\left[\frac{\ln(S_t d_R) - \ln(1-S_t)}{\ln(d_R) - \ln(R^{'})}\right], \qquad a_R = \frac{1}{(N_R^{'})^{b_R}} \ln\left(\frac{d_R}{R^{'}}\right).$$

(C) MS&S function

(i) Prebreeding census model

Under the MS&S function the density dependent reproductive rate is given by

$$R_t = d_R\left\{1 + (a_R N_t)^{b_R}\right\}^{-1},$$

where a_R and b_R are the parameters intended to be derived. Using the fixed point of $R_t = R'$ at $N_t = N_R^{'}$, and rearranging we obtain

$$a_R = \frac{1}{N_R^{'}}\left(\frac{d_R - R^{'}}{R^{'}}\right)^{1/b_R} \qquad \textbf{(A.12.13)}$$

Using the relationship $\lambda_t = S_{t-1}(1+R_t)$,

$$\lambda_t = S_{t-1}\left[1 + d_R\left\{1 + (a_R N_t)^{b_R}\right\}^{-1}\right].$$

Note that the relevant survival rate is the survival rate at the end of the previous annual cycle (i.e., S_{t-1}), not S_t.

Now since $\lambda_t = 1$ at $N_t = K$,

$$1 = S_{t-1}\left[1 + d_R\left\{1 + (a_R K)^{b_R}\right\}^{-1}\right]$$

By rearranging this we find

$$a_R = \frac{1}{K}\left(\frac{S_{t-1}(1+d_R)-1}{1-S_{t-1}}\right)^{1/b_R}. \qquad \textbf{(A.12.14)}$$

Using A.12.13 and A.12.14 we can write the parameter b_R as

$$b_R = \frac{1}{\ln(K)-\ln(N_R^{'})}\ln\left[\frac{\{S_{t-1}(1+d_R)-1\}R^{'}}{(1-S_{t-1})(d_R-R^{'})}\right]. \qquad \textbf{(A.12.15)}$$

and substituting this in to A.12.13 gives the parameter a_R. The DD curve defined by these parameters determines the reproductive rate (R_t) for the year.

In prebreeding models survival occurs after the reproduction. The density dependent survival under the MS&S function follows

$$S_t = d_S\left\{1+(a_S N_t)^{b_S}\right\}^{-1},$$

where parameters a_S and b_S need to be calculated. Using the specified point of $S_t = S'$ at $N_t = N_s^{'}$, and rearranging we get

$$a_S = \frac{1}{(N_S^{'})}\left(\frac{d_S - S^{'}}{S^{'}}\right)^{1/b_S}. \qquad \textbf{(A.12.16)}$$

Using the relationship $\lambda_t = S_t(1+R_t)$,

$$\lambda_t = (1+R_t)d_S\left\{1+(a_S N_t)^{b_S}\right\}^{-1}.$$

Since $\lambda_t = 1$ at $N_t = K$,

$$1 = (1+R_t)d_S\left\{1+(a_S K)^{b_S}\right\}^{-1}.$$

By rearranging this we get

$$a_S = \frac{1}{K}\{d_S(1+R_t)-1\}^{1/b_S}, \qquad \textbf{(A.12.17)}$$

and using A.12.16 and A.12.17 we can write the parameter b_s as

$$b_S = \frac{1}{\ln(K)-\ln(N_S^{'})}\ln\left[\frac{\{d_S(1+R_t)-1\}S^{'}}{(d_S-S^{'})}\right]. \qquad \textbf{(A.12.18)}$$

Substituting this into A.12.16 gives the parameter a_s. The DD curve defined by these parameters determines the survival rate (S_t) for the year.

(ii) Postbreeding census model

The derivation of the formulae for the parameters of the postbreeding census model follows the same steps. These formulae are

$$b_S = \frac{1}{\ln(K) - \ln(N_S^{'})} \ln\left[\frac{\{d_S(1+R_{t-1})-1\}S^{'}}{(d_S - S^{'})}\right], \qquad a_S = \frac{1}{(N_S^{'})}\left(\frac{d_S - S^{'}}{S^{'}}\right)^{1/b_S},$$

$$b_R = \frac{1}{\ln(K) - \ln(N_R^{'})} \ln\left[\frac{\{S_t(1+d_R)-1\}R^{'}}{(1-S_t)(d_R - R^{'})}\right], \qquad a_R = \frac{1}{N_R^{'}}\left(\frac{d_R - R^{'}}{R^{'}}\right)^{1/b_R}.$$

(D) Usher function

(i) Prebreeding census model

Under the Usher function the density dependent reproductive rate is given by

$$R_t = d_R\{1+\exp(b_R N_t - a_R)\}^{-1},$$

where a_R and b_R are the parameters intended to be specified. Using the fixed point of $R_t = R'$ at $N_t = N_R^{'}$ we get

$$R^{'} = d_R\{1+\exp(b_R N_R^{'} - a_R)\}^{-1}.$$

By rearranging this we get

$$a_R = b_R N_R^{'} - \ln\left(\frac{d_R - R^{'}}{R^{'}}\right) \qquad \textbf{(A.12.19)}$$

Using the relationship $\lambda_t = S_{t-1}(1+R_t)$,

$$\lambda_t = S_{t-1}\left[1+d_R\{1+\exp(b_R N_t - a_R)\}^{-1}\right].$$

The relevant survival rate is the survival rate at the end of previous annual cycle (i.e., S_{t-1}), not S_t.

Now since $\lambda_t = 1$ at $N_t = K$

$$1 = S_{t-1}\left[1+d_R\{1+\exp(b_R K - a_R)\}^{-1}\right].$$

By rearranging this we find

$$a_R = b_R K - \ln\left(\frac{S_{t-1}(1+d_R)-1}{1-S_{t-1}}\right), \qquad \textbf{(A.12.20)}$$

and using A.12.19 and A.12.20 we can write the parameter b_R as

$$b_R = \frac{1}{K - N_R^{'}}\left[\ln\left(\frac{S_{t-1}(1+d_R)-1}{1-S_{t-1}}\right) - \ln\left(\frac{d_R - R^{'}}{R^{'}}\right)\right]. \qquad \textbf{(A.12.21)}$$

Substituting this into A.12.19 gives the parameter a_R. The DD curve defined by these parameters determines the reproductive rate (R_t) for the year. In prebreeding models survival occurs after reproduction. The density dependent survival under the Usher function follows

$$S_t = d_S\{1+\exp(b_S N_t - a_S)\}^{-1},$$

where parameters a_S and b_S need to be calculated. Using the fixed point of $S_t = S'$ at $N_t = N_S^{'}$ on the survival curve we find

$$S^{'} = d_S\left\{1+\exp(b_S N_S^{'} - a_S)\right\}^{-1}.$$

By rearranging this we get

$$a_S = b_S N_S^{'} - \ln\left(\frac{d_S - S^{'}}{S^{'}}\right). \qquad \textbf{(A.12.22)}$$

Using the relationship $\lambda_t = S_t(1+R_t)$,

$$\lambda_t = (1+R_t)d_S\{1+\exp(b_S N_t - a_S)\}^{-1}.$$

Therefore, since $\lambda_t = 1$ at $N_t = K$,

$$1 = (1+R_t)d_S\{1+\exp(b_S K - a_S)\}^{-1},$$

Rearranging this we get

$$a_S = b_S K - \ln\{d_S(1+R_t)-1\}, \qquad \textbf{(A.12.23)}$$

and using A.12.22 and A.12.23 we can write the b_S as

$$b_S = \frac{1}{K - N_S^{'}}\left[\ln\{d_S(1+R_t)-1\} - \ln\left(\frac{d_S - S^{'}}{S^{'}}\right)\right]. \qquad \textbf{(A.12.24)}$$

Substituting this into A.12.22 gives the parameter a_S. The DD curve defined by these parameters determines the survival rate (S_t) for the year.

(ii) Postbreeding census model

The derivation of the formulae for the parameters of the postbreeding census model follows the same steps. These formulae are

$$b_S = \frac{1}{K - N_S^{'}}\left[\ln\{d_S(1+R_{t-1})-1\} - \ln\left(\frac{d_S - S^{'}}{S^{'}}\right)\right], \quad a_S = b_S N_S^{'} - \ln\left(\frac{d_S - S^{'}}{S^{'}}\right),$$

$$b_R = \frac{1}{K - N_R^{'}}\left[\ln\left(\frac{S_t(1+d_R)-1}{1-S_t}\right) - \ln\left(\frac{d_R - R^{'}}{R^{'}}\right)\right], \qquad a_R = b_R N_R^{'} - \ln\left(\frac{d_R - R^{'}}{R^{'}}\right).$$

(E) Logistic function

Unlike the other functions considered in this appendix, the logistic function is a two-parameter function having a linear relationship between the vital rate and population density. As a consequence we can model it with less input information than required by the other functions. Therefore we assume that only the values of the vital rate at the lowest density (d_S for survival, d_R for reproduction), and the carrying capacity of the population (K) are specified. In this function we only have to calculate one parameter for the DD relationship of each vital rate (a_S for survival rate and a_R for reproductive rate). As with the other functions, these values are time-specific because the DD relationships vary with time.

(i) Prebreeding census model

In prebreeding models we consider the reproduction prior to survival. Under the logistic function the density dependent reproductive rate is given by

$$R_t = d_R\left(1 - \frac{N_t}{a_R}\right),$$ where a_R is the unknown parameter for the function.

Using the relationship $\lambda_t = S_{t-1}(1+R_t)$,

$$\lambda_t = S_{t-1}\left[1 + d_R\left(1 - \frac{N_t}{a_R}\right)\right].$$

The applicable survival rate in year t is S_{t-1}, because in IDD models, any vital rate applicable at the present time is determined by the level of other vital rates immediately prior to the present.

Since $\lambda_t = 1$ at $N_t = K$,

$$1 = S_{t-1}\left[1 + d_R\left(1 - \frac{K}{a_R}\right)\right], \qquad \text{so}$$

$$a_R = \frac{d_R S_{t-1} K}{S_{t-1}(1+d_R) - 1}. \qquad \textbf{(A.12.25)}$$

The DD relationship defined by parameters d_R and a_R determines the reproductive rate (R_t) for the year. Survival occurs after the reproduction. The density dependent survival under the logistic function follows the relationship,

$S_t = d_S\left(1-\frac{N_t}{a_S}\right)$, where the parameter a_S need to be specified in terms of known information. Using the relationship $\lambda_t = S_t(1+R_t)$,

$$\lambda_t = d_s\left(1-\frac{N_t}{a_s}\right)(1+R_t).$$

Since $\lambda_t = 1$ at $N_t = K$

$$1 = d_s\left(1-\frac{K}{a_s}\right)(1+R_t),$$

Which means that

$$a_s = \frac{d_S(1+R_t)K}{d_S(1+R_t)-1}. \qquad \textbf{(A.12.26)}$$

The DD curve defined by d_S and a_S parameters determines the survival rate (S_t) for the year.

(ii) Postbreeding census model

The same steps can be followed to calculate the DD parameters for postbreeding models. The resultant formulae are

$$a_R = \frac{d_R S_t K}{S_t(1+d_R)-1} \qquad \text{and} \qquad a_s = \frac{d_S(1+R_{t-1})K}{d_S(1+R_{t-1})-1}.$$

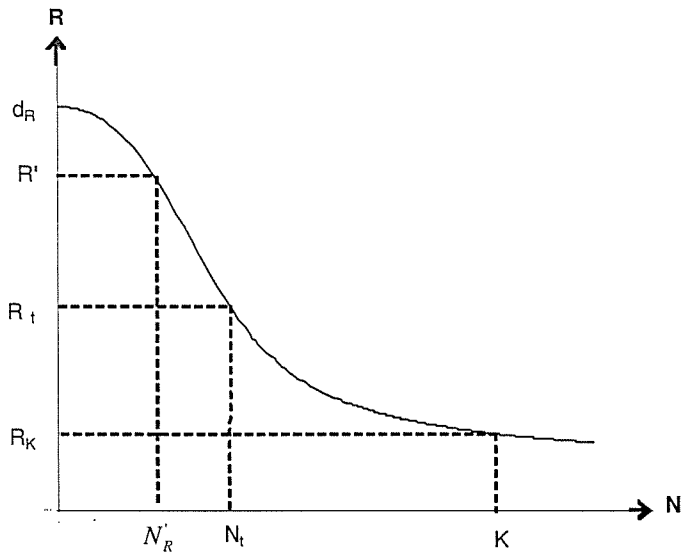
APPENDIX 13

Smallest levels of vital rates that can be used with IDD models

The IDD models consider an annual cycle as being comprised of two discrete, non-overlapping events; survival over the cycle, and reproduction during the cycle. The rate at which each event is occurring is determined by its own time-dependent DD curve. The shape of the DD curve of any vital rate at time t is influenced by the level of the other vital rate at its last occurrence. At each event the model attempts to adjust the DD curve such that the resultant growth rate becomes unity at carrying capacity. For example, in prebreeding models, the exact shape of the reproduction DD curve is determined by S_{t-1}. Suppose for some reason if S_{t-1} falls to a small level then R_t needs to be large to fulfil the above requirement. If S_{t-1} is very small, R_t could even be larger than d_R. Similarly, in the second half of the prebreeding cycle, the survival function is determined by R_t. If R_t is very small then S_t can become very large, even larger than d_S. Values of R_t larger than d_R, or of S_t larger than d_S, are impossible in negative density dependence. Therefore in these situations the IDD cannot be used with any negative DD function. Specifically, it cannot be used if either of the vital rates have fallen below some minimum threshold. In this appendix I show how to derive these minimum thresholds.

(A) Prebreeding models

In prebreeding models, reproduction occurs prior to survival. Two points on the reproduction curve are specified by the input information, therefore the exact shape of the reproduction curve that passes through these two points is determined by the function selected to represent the DD and the strength of the DD. The strength of the DD is determined to ensure that $S_{t-1}(1+R_K)=1$, where R_K is the reproductive rate at carrying capacity under the resultant reproduction curve.



Now, $S_{t-1}(1+R_K)=1$, so $R_K=\frac{1}{S_{t-1}}-1$. Further, $R_K<d_R$ for any negative DD function. Therefore $\frac{1}{S_{t-1}}-1<d_R$, or equivalently $S_{t-1}>\frac{1}{1+d_R}$, which implies that, if the previous year survival rate was smaller than $\frac{1}{1+d_R}$ then the reproductive rate cannot increase enough to keep the population growing towards the carrying capacity as required by the IDD model.

Similarly, two points on the survival curve are given by the input information, and the exact shape of the survival curve that passes through these points is determined by the selected DD function and R_t. That is done by ensuring that $S_K(1+R_t)=1$, where S_K is the survival rate at carrying capacity under the resultant survival DD curve.

Since $S_K(1+R_t)=1$, $S_K=\frac{1}{1+R_t}$. For any negative DD function $S_K<d_S$. Therefore, $\frac{1}{1+R_t}<d_S$, or equivalently, $R_t>\frac{1}{d_S}-1$. This implies that, if the reproductive rate was smaller than $\left(\frac{1}{d_S}-1\right)$ then the survival rate cannot increase enough to compensate for the drop, and a population level of K cannot be reached as expected by the model.

(B) Postbreeding models

In postbreeding models survival occurs prior to reproduction. Since two points on the survival curve are determined by the input information, the exact shape of the survival curve that passes through these two points is determined by the selected DD function and the reproductive rate resulted in previous year (R_{t-1}). That is done by ensuring that $S_K(1+R_{t-1})=1$, where S_K is the survival rate at carrying capacity under the resultant survival curve.

$S_K(1+R_{t-1})=1$. So $S_K=\frac{1}{1+R_{t-1}}$. Hence, for any negative DD function $S_K<d_S$. Therefore, $\frac{1}{1+R_{t-1}}<d_S$, or equivalently, $R_{t-1}>\frac{1}{d_S}-1$. This implies that if the previous year reproductive rate was smaller than $\left(\frac{1}{d_S}-1\right)$ then a survival DD function as required by the IDD model cannot exist.

Similarly, the shape of the reproductive curve that passes through the two specified points is determined by the selected DD function and the S_t by ensuring that $S_t(1+R_K)=1$, where R_K is the reproductive rate at carrying capacity under the resultant reproductive curve.

$S_t(1+R_K)=1$, so $R_K=\frac{1}{S_t}-1$. For any negative DD function $R_K<d_R$. Therefore, $\frac{1}{S_t}-1<d_R$, or equivalently, $S_t>\frac{1}{1+d_R}$. This implies that if the survival rate was smaller than $\left(\frac{1}{1+d_R}\right)$ then a reproductive DD function as required by this method cannot exist.

Therefore, in both prebreeding and postbreeding situations, IDD models cannot be used along with negative DD functions if the reproductive rate drops below $\left(\frac{1}{d_S}-1\right)$ or the survival rate drops below $\frac{1}{1+d_R}$.

Notes:

(1) Here the intention was simply to show that this method does not work in some situations. The critical factor that determines the applicability of this method is whether the levels of vital rates exceed a certain minimum level. We derived this minimum level in order to adhere to the negative density dependence condition

only (i.e., $S_K < d_S$, and $R_K < d_R$ only). Instead, we could have used stricter conditions of $S_K < S'$ and $R_K < R'$ when $N_S^{'}$ and $N_R^{'}$ are smaller than K. Then they can be transformed to function-specific conditions by using the functional relationship between vital rate and density. Therefore, the minimum levels derived above can only be considered as absolute minima that should never be reached, rather than function-specific minima. Also, having been confined to the thresholds shown above does not guarantee that this method can be used with any given DD function. Depending on the function, and on the exact input information used, the minimum level that the vital rate should not reach can be larger.

(2) The descriptions in this appendix assumed the input information used in the model is the same as that which was used in the model description in section 8.4.2. It is possible that some IDD models could be based on different input information, as is shown in section 8.4.6.c. In those situations new thresholds can be recalculated using the same approach used here.

(3) Vital rates could be small in populations that are already small and declining towards extinction. When one vital rate is small, this method as describe above, tends to produce a DD curve for the other vital rate with larger values, which does not correspond to a declining population. In fact, in this situation, one of the inputs (i.e. $\lambda_t = 1$ at $N_t = K$) is inappropriate for a declining population, because the population cannot grow towards K. A different input information, such as $\lambda_t = \lambda^*$ at $N_t = N^*$ could be more appropriate in this situation (as explained in section 8.4.6.c).