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Mathematical models for the frequency-dependent transmission of cultural traits

Caroline E. Walters

A Thesis presented for the degree of
Doctor of Philosophy



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March 2014

Dedicated to

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Abstract

Cultural evolutionary theory is concerned with the social transmission of behaviours, beliefs or ideas that constitute culture. In humans, transmission of culture may be from one generation to the next or between individuals of the same generation. This thesis contains three models for the transmission of cultural traits, subject to frequency-dependent social learning. All models are formulated as a system of differential equations that cannot be solved analytically. By finding the equilibria of the systems and analysing their stability, the long-term behaviour of the systems may be determined.

A mathematical model for the spread of drinking behaviour is presented, with a focus on total recovery. The equilibria of the system are found and a local stability analysis is performed. The system is found to have a parameter-dependent threshold at which the two equilibria switch stability. This indicates a change in the long-term system behaviour. Consequently, whether drinking behaviour dies out or becomes endemic may be predicted from the values of the model parameters. The rate at which individuals take up drinking behaviour is found to have the greatest effect on whether it becomes endemic.

A model for both the linear and nonlinear frequency-dependent transmission of a cultural trait, with potential applications to binge drinking behaviour, is then investigated. The system equilibria cannot be found explicitly in terms of the model parameters. However, by considering different cases corresponding to regions of

parameter space, qualitative differences in the long-term behaviour of the system are determined. By comparing the linear and nonlinear frequency-dependent models, the effect of conformity is determined for different regions of parameter space.

Finally, a reaction-diffusion model for two competing languages, u and v , with a focus on language coexistence is presented. Language u is assumed to confer a status advantage to its speakers, thus switching languages is one-directional from v to u . Four constant system equilibria are found and global instability and stability thresholds are found for each solution. The coexistence of languages u and v is found to be globally stable, subject to certain parameter constraints and a sufficiently small initial population of speakers.

Declaration

The work in this thesis is based on research carried out in the Departments of Anthropology and Mathematical Sciences at the University of Durham, England. No part of this thesis has been submitted elsewhere for any other degree or qualification and it is all my own work, unless referenced to the contrary in the text. In particular, section 1.5 is based on joint work published in Kendal and Walters (in press), chapter 2 is a reproduction of the joint work Walters et al. (2012), and chapter 3 is a reproduction of the joint work Walters and Kendal (2013), for which I performed all of the mathematical analysis. Chapter 4 has been submitted for publication in *Meccanica*, for which I am the sole author.

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Chapter 1

Introduction

1.1 Thesis overview

Presented in this thesis are mathematical models for the spread of culture within human populations via social learning mechanisms. Using techniques developed in mathematical biology, the models represent the frequency-dependent transmission of cultural traits. Whilst all models are, to some extent, generally applicable to a variety of behaviours and situations, chapters 2 and 3 focus on drinking behaviour and chapter 4 on language competition. The motivation behind developing models for alcohol problems stems from the increasing concern regarding the health and economic effects of problem drinking, whether in the form of alcohol addiction or binge drinking behaviour. To reduce the prevalence of the behaviour (and thus reduce the negative associated costs) it is necessary to understand what influences drinking behaviour and how this may be changed. Mathematical modelling alone does not provide the answer to this, but it provides a mechanism for analysing trends and predicting future outcomes.

The language model extends existing work aimed at determining whether competing languages can coexist over time. Many of the world's minority languages are in decline, so predictions from mathematical models may prove vital to developing appropriate strategies for the preservation of these languages. We develop a model for two competing languages, where one is a minority language, and analyse the global stability and instability properties of all of the constant system equilibria.

This reveals conditions under which the survival of both languages is stable.

Each research chapter has its own distinct novel aspect, however all are conceptual contributions to the field of cultural evolution. In chapter 2, this is the inclusion of a term in the alcohol model which permits individuals to fully recover from an alcohol problem. The model is an extension of work by Mulone and Straughan (2011) and was developed in light of recent theories regarding the nature of recovery. In chapter 3, the novel aspect is the incorporation of a conformist social learning bias, from the cultural evolution literature, into an SIS (Susceptible-Infected-Susceptible) model framework. The model reduces to a single polynomial equation, allowing for information to be gained through utilising methods from calculus. This is an approach not previously documented in the literature, thus the model and accompanying analysis are new contributions. The global stability analysis method in chapter 4 is well-documented in the fluid dynamics literature, however here it is applied to a novel situation: the coexistence of languages.

Following a brief introduction to each of the research chapters, the proceeding sections of this chapter introduce the main topics of the thesis. A review of cultural evolutionary theory is presented, with a focus on social learning transmission biases. A critical analysis of mathematical modelling methods follows, leading to a discussion of the use of this approach, and others, in cultural evolution research. The remaining sections, 1.5 and 1.6, review existing work which provides an introduction to the modelling approaches utilised in chapters 2 to 4. Specifically, section 1.5 contains a review of an ordinary differential equation (ODE) model for drinking behaviour by Sanchez et al. (2007). The extension of such models to systems of partial differential equations (PDEs) is discussed in section 1.6.

As the motivation for this thesis was to develop mathematical modelling techniques applicable to cultural evolution, the merits and pitfalls of the theory itself are not debated. For an insight into where cultural evolutionary theory lies within the broader context of human evolution, Laland and Brown (2011) provides an introduction. The mathematical models presented in the thesis are examples of dynamical systems and the analytic methods have been widely applied in the fields of epidemiology and fluid mechanics (see Hethcote (2000); Straughan (2004)). Some of the

material reviewed later in this chapter centres on behaviour transmission, developed from models for the spread of infectious diseases. For an introduction to dynamical systems, see any undergraduate textbook on the subject, such as Arrowsmith and Place (1990); Hale and Koçak (1991); Tu (1992).

Chapter 2 consists of a model for the linear frequency-dependent social transmission of problem drinking behaviour, extending previous work in this area by Mulone and Straughan (2011). Binge drinking in the UK is an increasing problem, resulting in negative health, social and economic effects. Mathematical modelling allows for future predictions to be made and may provide valuable information regarding how to approach solving the problem of binge drinking in the UK. We develop a 3-equation model for alcohol problems, specifically binge drinking, which allows for total recovery. Individuals are split into those that are susceptible to developing an alcohol problem, those with an alcohol problem, and those in treatment. We find that the model has two equilibrium points: one without alcohol problems and one where alcohol problems are endemic in the population. We compare our results with those of an existing model that does not allow for total recovery. We show that without total recovery, the threshold for alcohol problems to become endemic in the population is lowered. The endemic equilibrium solution is also affected, with an increased proportion of the population in the treatment class and a decreased proportion in the susceptible class. Including total recovery does not determine whether the proportion of individuals with alcohol problems increases or decreases, however it does affect the size of the change. Parameter estimates are made from information regarding binge drinking, where we find an increase in the recovery rate decreases the proportion of binge drinkers in the population.

Chapter 3 develops the preceding work by introducing a social learning bias into an SIS model framework. Epidemiological models have been applied to human health-related behaviours that are affected by social interaction, for example smoking (Sharomi and Gumel, 2008), drinking (Sanchez et al., 2007; Mulone and Straughan, 2011) or drug use (White and Comiskey, 2007; Mulone and Straughan, 2009). Typically these models have not considered conformity bias, which is the exaggerated propensity to adopt commonly observed behaviours or opinions, or con-

tent biases, where the content of the learned trait affects the probability of adoption. Here we consider an interaction of these two effects, presenting an SIS-type model for the spread and persistence of a behaviour which is transmitted via social learning. Uptake is controlled by a nonlinear dependence on the proportion of individuals demonstrating the behaviour in a population. Three equilibrium solutions are found, their linear stability analysed, and the results compared with a model for unbiased social learning. Our analysis focuses on the effects of the strength of the conformity bias and the effects of content biases which alter a conformity threshold frequency of the behaviour, above which there is an exaggerated propensity for adoption. The strength of the conformity bias is found to qualitatively alter the predictions regarding whether the trait becomes endemic within the population and the proportion of individuals who display the trait when it is endemic. As the conformity strength increases, the number of feasible equilibrium solutions increases from 2 to 3, leading to a situation where the stable equilibrium attained is dependent upon the initial state. Varying the conformity threshold frequency directionally alters the behaviour invasion threshold.

Neither of these models considers the spatial variation of individuals, which would increase the complexity of the system. Motivated by existing work by Kandler and Steele (2008), chapter 4 includes a PDE model for competing languages which incorporates both temporal and spatial variation. One language is assumed to be dominant so conversion between languages is one-directional to the dominant language. The system has four equilibria, including a coexistence state, and we analyse the global stability and instability of each solution. Stability thresholds are found in each case, and thus we conclude that the coexistence of languages is possible, subject to certain parameter constraints.

Finally, chapter 5 contains a general discussion of the outcomes of the previous chapters and how these results are situated within the wider literature. Potential extensions to the models are highlighted, with an overview of future work presented.

1.2 Introduction to cultural evolution

Cultural evolutionary theory seeks to answer questions about human evolution that have not been adequately answered by genetic evolutionary theory. For any sound theory to be developed, it is first necessary to define what is meant by the terms culture and cultural evolution. Culture has been defined as “information capable of affecting individuals’ behaviour that they acquire from other members of their species through teaching, imitation, and other forms of social transmission” (Richerson and Boyd, 2005, Page 5). Information may refer to particular beliefs, behaviours, ideas or knowledge. Whilst various definitions of culture exist, the key factor in cultural evolutionary theory is that information which affects behaviour is learned from other individuals, either consciously or subconsciously. Cultural evolution is therefore a “process of descent with modification” (Mesoudi, 2011) by which selected socially learned behaviours spread and persist within a population over time. Assuming that information is transmitted in this way allows for theories to be constructed within a Darwinian evolutionary framework, allowing for a scientific approach to be taken.

To allow formal models of cultural evolution to be developed, the concept of a cultural trait (analogous to a genetic trait in biological evolutionary model) is used. The trait is a specific behaviour or idea which may be socially transmitted. Unlike with genes, where transmission is typically from parent to offspring (vertical transmission), Cavalli-Sforza and Feldman (1981) present models for cultural traits also being transmitted between individuals of the same generation (horizontal transmission) or from other members of the parent generation to the offspring generation (oblique transmission). Information may be gained without copying, known as asocial or individual learning. This is where an individual acquires information on their own, such as through a trial and error method or by innovation. When combined with social learning this can give rise to cumulative cultural evolution whereby information is transmitted and modified over time, leading to more complex or efficient cultural traits being developed (Richerson and Boyd, 2005) . The effect of this process is that individuals adopt behaviours that could not be learned by a single individual in their lifetime (Mesoudi, 2011) and is thought to be unique to humans (Richerson and Boyd, 2005).

Cultural traits are often modelled as discrete units, analogous to genes, which are passed from one individual to another (Henrich et al., 2008). This approach is appropriate in some instances where the cultural trait is a discrete entity. For example, the model presented in chapter 3 allows individuals to be of type S or type A, where type S do not have trait A and type A do display trait A. Trait A in this instance could be binge drinking behaviour, so type A individuals are those that binge drink. Whilst there is debate over what constitutes binge drinking, once these limits have been defined then an individual can be classified as either a binge drinker or not a binge drinker. In some cases a binary choice model such as this is not appropriate as traits may be best envisioned as lying on a continuum. One example is arrowhead length, which may vary continuously over a certain value range (Mesoudi, 2011). In such cases the genetic analogue is no longer appropriate as genes are discrete entities which are replicated, whereas arrowhead length may be subject to blending effects where the transmitted length is some combination (such as a mean average) of all the available model arrowheads (Henrich et al., 2008). This is one example of where genetic and cultural evolution differ and thus require different modelling approaches. Another difference is the concept of guided variation in cultural evolution, which has no genetic analogue. Guided variation is the intentional modification of a cultural trait (Mesoudi, 2011). In genetic evolution, modifications are the result of random (unguided) mutations (Mesoudi, 2011), so no equivalent to guided variation exists.

The study of simultaneous and interacting genetic and cultural evolution is known as gene-culture coevolutionary theory or dual-inheritance theory (Laland and Brown, 2011). The influence between genes and culture is two-directional, so genes may favour the evolution of particular cultural traits which, in turn, then increase the favourability of specific genes. One widely-referenced example which supports the theory is the correlation between dairy farming and lactose tolerance in adults (see, for example, Laland and Brown (2011); Richerson and Boyd (2005)). Adult human populations vary in their ability to digest cows' milk (which contains lactose), controlled by a specific allele. Models suggest that this is an example of gene-culture coevolution, whereby the uptake of dairy farming increased the relative

fitness of the gene which allows for adult lactose tolerance as a result of the benefits of drinking milk (see Feldman and Cavalli-Sforza (1989); Itan et al. (2009); Gerbault et al. (2011)). This in turn may have influenced cultural practices in such a way that lactose consumption became more strongly favoured (Richerson and Boyd, 2005).

It is theorised that the persistence of a cultural trait may be influenced by a number of factors which affect transmission, which Richerson and Boyd (2005) refer to as forces of cultural evolution. Random forces are cultural mutation and cultural drift, discussed in both Cavalli-Sforza and Feldman (1981) and Boyd and Richerson (1985). Individual-level processes, such as misremembering a trait or lacking the ability to faithfully reproduce the observed trait, result in cultural mutation where the trait becomes unintentionally modified after transmission. Cultural drift can occur as a result of sampling size. If only a small number of individuals have a particular trait then it is possible that they never form part of an observed sample, so there is no opportunity for the trait to be transmitted. This mechanism may describe the loss of tool complexity in the Tasmanian population, which arose after their separation from mainland Australia (Henrich, 2004). Alternatively, a transmitted trait may be intentionally modified through guided variation. As the changes to the trait are wilful, this is a decision-making, rather than a random, force. The remaining decision-making forces are all a result of the biased social transmission of a cultural trait, which may be further split into content and context biases (Henrich and McElreath, 2003).

Content biases affect the likelihood of adopting a trait through intrinsic properties of the trait itself, such as its salience or as a result of cost-benefit analysis. Context biases refer to external influences which affect trait adoption and have been split into two categories: model-based biases and frequency-dependent biases (Henrich and McElreath, 2003; Richerson and Boyd, 2005). Model-based biases result from some characteristic of a sampled individual. For instance, a naïve individual may be more likely to choose to copy a specific individual because of perceived similarities with the model, or because of the perceived success of the model. The existence of such biases is supported by experimental work where individuals copy the most successful individual (Mesoudi, 2008). Other model-based biases consider

how prestigious or successful the model is perceived to be. Frequency-dependent biases represent when trait adoption is influenced by the frequency of the trait in the observed population in a fashion which does not replicate random sampling. If a trait has frequency of 70% within the sampled population then a naïve individual using a linear frequency-dependent strategy (equivalent to random sampling or unbiased social learning) has probability 0.7 of adopting the trait. This is equivalent to the individual randomly choosing one model from the population and copying them. For a bias to be in action, the probability of a naïve individual adopting the trait must differ from 0.7. Conformist frequency-dependent bias occurs when a common trait in the population is more likely to be acquired, so a 70% prevalence of the trait gives a probability of adoption which is greater than 0.7. Conversely, anti-conformist bias can also occur, where infrequent traits are more likely to be adopted so the adoption probability would be less than 0.7.

Conformist behaviour contributes to explanations of human cooperative behaviour and the use of punishment in large groups (Boyd and Richerson, 1985; Henrich and Boyd, 2001). Models for trait transmission show that conformist bias is favourable in spatially and temporally varying environments, including rapidly changing environments (Kendal et al., 2009; Nakahashi et al., 2012). In a stable environment social learning dominates, yet conformist transmission has little effect on a learner's ability to acquire the adaptive behaviour (Wakano and Aoki, 2007; Kendal et al., 2009). Whilst research so far has not reached a consensus regarding the evolution of conformist transmission, it remains a valid explanation for cultural trait transmission under certain conditions. For instance, Efferson et al. (2008) conducted a study where 28 out of 40 participants self-identified as conformist. These individuals completed a binary choice task where one choice has a greater expected payoff. The experiment was controlled so that these individuals could only utilise social information when making their choice. The 28 individuals who stated that they were conformist were found to act in a conformist fashion, indicating that some individuals act conformist, but not all.

Such studies have been criticised as it may be difficult for an individual to ascertain which of two options is most profitable after only a small number of trials, thus

copying others could be considered to be a rational choice rather than the result of conformity (Eriksson and Coultas, 2009). A further criticism is that experiments are generally limited to investigating only one social learning strategy, yet individuals may employ multiple strategies (Morgan et al., 2011). Experimental evidence where multiple learning biases can be in action simultaneously suggested that conformist behaviour was present when a subject had low confidence in their own ability and a sufficient number of demonstrators to copy (Morgan et al., 2011). Research into how frequently conformist behaviour occurs, if at all, is not in agreement, hence further work in this area may provide greater insights. This motivated the work in chapter 3 where a mathematical model for conformist cultural trait transmission is presented.

Research into cultural evolution has been approached via both theoretical and empirical methods. In particular, mathematical models have been devised to explain many facets of cultural evolutionary theory. To appreciate the utility of this approach it is necessary to understand the motivation behind such models, and both the strengths and limitations of the method. A discussion of these factors is presented in the next section.

1.3 Introduction to mathematical models

Models may be considered to be simplified representations of the real world which aid our understanding. By eliminating aspects which are not immediately pertinent to the problem at hand, they allow us to focus on the key features of interest, without unnecessary distraction. A model must always be fit for purpose: as simple as possible but no simpler (Keeling and Rohani, 2008). For example, the London Tube map is a model designed to help travellers navigate the underground rail network by presenting the railway line intersections and interchange stations (Degani, 2013). The map depicts the rail network's topology but not the exact location and relative distances of stations so includes the information of interest whilst omitting unnecessary details. In the same way that features of the London Underground can be encapsulated in a diagram, some real-world systems may be described by

mathematics.

Creating a mathematical model begins with determining the key real-world features required to address the question at hand. It is then assumed that only these variables have any influence on the system, thus mathematical models are always simplified representations of reality. Often mathematical models appear to be overly simplistic, however “... attempting to deduce the answer to a complex problem by direct inspection and unaided intuition requires even simpler models and entails great risk of erroneous reasoning” (Boyd and Richerson, 1985, Page 30). Simplification is necessary to enable the discovery of underlying causal relationships. Whilst results might seem obvious retrospectively, without the formalisation introduced by the modelling procedure there is no way of being certain that any inferences made are correct. In some cases counter-intuitive results are revealed only through mathematical analysis, as with the well-known Monty Hall problem (Appendix A.1). An understanding of the real-world problem is achieved by the interpretation of the mathematical results. If no sensible real-world explanation can be given then the model has not fulfilled its function and therefore must be refined. In the following research chapters the model variables represent population frequencies. Mathematically, these variables may be negative but such cases have no real-world meaning. This instigates the introduction of certain parameter restrictions to ensure that the final results can always be interpreted in a meaningful way. Thus the aim of mathematical modelling is to provide a good approximation of a reality in a way which allows for structured analysis. From this, a greater understanding of the real-world system may arise and enable future predictions to be made.

There is often a trade-off between accuracy, transparency and flexibility within mathematical models (Keeling and Rohani, 2008). Accuracy refers to how well the model reproduces observed data and predicts future outcomes, and will often be improved by increased model complexity. One way of assessing the accuracy of a mathematical model is to compare the predicted results to known scenarios. This could be information gained by comparing the model predictions against existing data sets, as in Bentley et al. (2007) and Hamilton and Buchanan (2009), or from complementary theoretical and empirical methods, as in Efferson et al. (2008) and

Eriksson and Coultas (2009). Transparency refers to how easy it is to interpret the effects of a single variable parameter on the system, independent of all other parameters. Finally, flexibility refers to how adaptable a model is to a changing situation. For instance, if there is a real-world system change, can varying a parameter account for this variation or is it necessary to formulate a new model? Model complexity can affect the methods of analysis which are used. Analytic methods give results which hold true for vast areas of parameter space and make it easy to identify regions where there is a qualitative difference in the results. This is a useful method for finding thresholds which indicate a qualitative change in system behaviour. As model complexity increases, transparency decreases and thus analytic methods become increasingly difficult and yield fewer tractable results. In such instances numerical computer simulations may be utilised, whereby a solution may be obtained for a specific set of parameter values.

To gain an understanding of the system as a whole, many simulations must be run (McElreath and Boyd, 2007). For example, a system consisting of 3 variables, each taking a possible 15 values, requires $15^3 = 3375$ calculations to be made to ensure all parameter sets have been considered. As simulation results only give a snapshot of the system at specific values, finding thresholds and general trends can be difficult. When possible, it may be advantageous to further simplify a model to enable analytic results to be found. A simplified model may reveal which parameter has the greatest effect on the system, thus leading to a more informed investigation of the complex model by simulations concentrated on varying this parameter. By comparing the results of the two models a greater understanding of system behaviour may be gained than could be obtained purely from numerical simulations of the complex model.

A mathematical approach to real-world problem solving has many advantages, often in conjunction with other methods, such as empirical studies. Mathematical language is precise, enabling clear communication of findings with respect to well-defined assumptions. This makes model results, and the context in which they are applicable, easily understandable to other researchers. Mathematical models may also be a cheaper way (both in time and money) of gaining information about a

system compared with empirical testing or observational studies. As they define the world in terms of a discrete number of variables, they may be used to highlight variables of interest and thus influence the direction of any empirical work before a large investment is made. As more knowledge is gained through the comparison of different models and empirical results, models can be refined to focus only on the most pertinent aspects of the system. Provided a model can replicate the results of empirical work, it may then be used to make future predictions about the state of the system.

1.4 Modelling approaches used in cultural evolutionary theory

The benefits of mathematical modelling led to its use in the study of cultural evolution, with much initial work consisting of mathematical models developed from the population genetics literature. For instance, Cavalli-Sforza and Feldman (1981) construct a model for vertical cultural trait transmission. Cultural traits are often assumed to be discrete and, in some cases, mutually exclusive. Models may be similar to those from population genetics representing the transmission of genes from parent to child. A simple example of a genetic model (without mutation) consists of two alleles: the dominant A and the recessive a . If both parents are type Aa then they display the phenotype coded for by the dominant allele A . If each parent contributes one allele to the child then the child displays the phenotype coded by the dominant allele A (from AA or Aa pairings) with probability $3/4$ and the phenotype coded by a (from pairing aa) with probability $1/4$.

In a model for vertical cultural trait transmission, Cavalli-Sforza and Feldman (1981) allow for a cultural trait to take one of two possible states, H or h , where each parent has one variant. Random mating results in the possible mother-father pairings HH , Hh , hH and hh . For each pairing there is some probability that the child acquires variant h which, when summed with the probability of acquiring H , totals unity. The probability of a child being type h with parental pairing HH is not assumed to be 0. This could be due to trait mutation, thus similar to a genetic

model with mutation, or a result of horizontal or oblique transmission, which are not common in genetic models (Cavalli-Sforza and Feldman, 1981). Alternatively, if H represents having some cultural trait and h represents not having the trait then the child may be of type h because of factors which affect the vertical transmission of the trait. Social learning biases, such as a content bias, may limit the child's ability to copy the trait.

By determining the frequency of both H and h over discrete time steps (representing generations), equilibrium frequencies of both traits can be obtained. The system has reached equilibrium when the frequencies of both H and h remain the same from one generation to the next. Equilibria may be classified as either stable or unstable, with the system only maintaining a stable equilibrium frequency. Conditions for stability in terms of the model parameters can often be found, either analytically or numerically. An analytic result was obtained by Cavalli-Sforza and Feldman (1981) for their model. More complex models for vertical trait transmission were investigated by Cavalli-Sforza and Feldman (1981), such as where assortative (as opposed to random) mating occurs or where the sex of the parent has a significant effect on the transmission of a trait. They then explore models which assume oblique and horizontal cultural trait transmission. In particular, they propose the use of a Lotka-Volterra type model to represent the adoption of a small family ideal, where a woman chooses to have only a small number of children. The two variables considered are the natural fertility number n and the voluntarily reduced fertility number m . Unlike with the previously discussed models, where traits are passed on at each generation, the adoption of a small family ideal is continuous in time and only oblique and horizontal transmission can occur. By determining the system equilibria, conditions on the model parameters can be found which ensure that the small family ideal either dies out or becomes dominant practice.

A similar approach to modelling cultural evolution was taken by Boyd and Richerson (1985), where again mathematical models from population genetics were used as a basis to develop theoretical models of cultural evolution. They consider a dichotomous cultural trait, with individuals either displaying trait A or not trait A (denoted by A'). Transmission is from a parent generation to a child generation so

both vertical and oblique transmission can occur. Naïve individuals in the child population choose a sample of three cultural parents to observe and socially learn from, where linear frequency-dependent transmission of the cultural traits is assumed. If all cultural parents display trait A then the child will adopt A , however if the parent generation consists of, say, AAA' then the child develops A with some probability in $(0, 1)$. This is dependent upon the weighted influence of each cultural parent on the child. Analysis reveals that if the frequency of A in the population at time t is p , then the frequency of A at time $t + 1$ is also equal to p .

This model is then altered to represent a nonlinear frequency-dependent transmission bias. The probability of having i parents with trait A , where the frequency of A is p , is taken to be binomially distributed. Accounting for a transmission bias, they find that, after transmission, the frequency of A is

$$p' = p + Dp(2p - 1)(1 - p), \quad (1.1)$$

where D is a parameter controlling the extent of the nonlinear frequency-dependent bias. The right-hand side expression of equation (1.1), representing both conformist and anticonformist transmission, has since been advanced, for example in Eriksson and Coultas (2009) and Kendal et al. (2009). It is also used in the model in chapter 3 where trait uptake is assumed to be continuous in time, rather than occurring at discrete time steps as with the Boyd and Richerson (1985) model. Equation (1.1) is not the only mathematical function representing a nonlinear frequency-dependent bias that has been investigated; alternatives are discussed in Nakahashi (2007) and Aoki et al. (2011).

Such developments of population genetics models to cultural trait models demonstrate how the abstract nature of mathematics allows for its application to a variety of different scenarios after only minor adjustments. However, the existence of genetic evolution models directed the formulation of similar models for cultural evolution, with certain assumptions introduced to ensure a fit to the pre-existing mathematical framework. The assumption that biological and cultural evolution can be understood by similar mechanisms has been questioned by Strimling et al. (2009). They argue that a fitness index, as used in biological models, is not an appropriate comparison measure for cultural traits. Unlike genetic information, which is acquired once,

cultural traits may be adopted and abandoned many times by a single individual during their lifetime. By constructing a model where both the propensity to adopt a trait and the propensity to keep that trait when presented with alternative options, the authors find a cultural success index under certain conditions. The index arises when individuals have a sufficiently large number of learning opportunities, which allows them to repeatedly develop and change traits, and is applicable whether the transmission is horizontal, vertical or oblique. Assumptions made in the mathematical model, for instance that all individuals adopt and retain traits with equal probabilities, reduce its accuracy in representing the complex mechanisms of human social learning. However, it addresses an important question of the synonymy of genetic transmission mechanisms with cultural trait transmission mechanisms.

As discussed in section 1.3, mathematical modelling can be most effective alongside other research methods. For example, a study by Henrich and Broesch (2011) into the existence and extent of social learning biases within a small-scale society used both ethnographic observation and interviews to obtain information. Results reported evidence of social learning biases, including biases towards copying individuals perceived to be successful or knowledgeable. Historical observations have also been used to support cultural evolutionary theories, such as the correlation between lactose tolerance in humans and the spread of dairy farming (discussed previously) or a wave of advance model for farming technologies (Ackland et al., 2007). If a theory accurately describes the recorded phenomena then it provides a plausible explanation; however, such methods are limited by the access to appropriate data sets and because often only correlation rather than causal relationships can be inferred (Simonton, 2003). Experimental work removes this latter problem as it allows for the control of variables so that casual relationships may be derived from the results (Simonton, 2003).

An experimental approach has been taken to investigate hypotheses regarding the social learning mechanisms that affected projectile-point design in the Great Basin around 300-600 AD (Mesoudi and O'Brien, 2008). The experiment tested whether guided variation (where individuals copy and then modify) or indirect bias produced results which matched the archaeological data. Indirect bias arises when

an individual uses one trait, called the indicator trait, to select a model and then adopts other traits displayed by this model (Bettinger, 1991). The bias is indirect as a preference for the indicator trait leads to the choice to copy other traits from the same model. During the experiment, participants played a computer simulated game where they designed projectile points and then tested them in a virtual hunting environment. The experiment was conducted in different phases, allowing control over the possible learning mechanisms that participants could use. For example, in the first phase participants had to choose to copy their point design from some presented options so no innovation or modification was possible. This permitted indirect-biased social learning. In the second phase they could choose to modify this design, allowing for guided variation.

As with mathematical modelling, experimental work requires certain details to be omitted. This is done to allow for causal relationships to be investigated by controlling certain key variables. Mesoudi and O'Brien (2008) highlight that the computer simulation task does not consider constraints on the availability of raw materials or the process of manufacturing, both of which could affect projectile point design. Laboratory experiments can be changed to focus on different aspects of a problem and build up a greater wealth of knowledge. The projectile-point design task was again implemented with different conditions, allowing for a comparison of results between the two studies (Mesoudi, 2008).

1.5 A review of a model for the horizontal transmission of drinking behaviour

A variety of mathematical techniques have been employed to describe different aspects of human behaviour, for example the kinetic theory of active particles (Bellomo et al., 2009; Bellomo and Carbonaro, 2011). This method models the dynamics of complex systems comprised of a large number of interacting living entities and has been applied to opinion formation (Bellomo et al., 2009) and socio-economic systems (Bellomo et al., 2004). One modelling approach discussed by Cavalli-Sforza and Feldman (1981) comes from epidemiological literature regarding the spread of

infectious diseases and the potential application to cultural trait transmission. As cultural traits can spread through a population from person to person, much like an infectious disease, the same mathematical techniques may be employed. Typically such models presume that a specified cultural trait is socially learned and spread via horizontal transmission. By considering a population of N individuals and splitting them into distinct classes, the equilibrium frequencies of each class can be determined and analysed. For example, a typical infectious disease model (known as an SIR model) consists of splitting the population into three groups: those susceptible to catching the disease; infected individuals, who are also assumed to be infectious; and recovered individuals, who have gained immunity. If a susceptible meets an infected then there is a chance that they will catch the disease and therefore move to the infected class. Over time an infected will recover from the disease and enter the recovered class. The aim is to determine whether the disease is sufficiently infectious for it to become endemic within the population, or whether it will die out. This can usually be determined from a threshold parameter known as the basic reproduction number, R_0 . At $R_0 = 1$ there is a change of state, where the disease moves from dying out to persisting within the population.

There is an increasing body of literature which uses these techniques to model the spread of health-related behaviours via social interaction; examples include smoking (Sharomi and Gumel, 2008), drinking (Sanchez et al., 2007; Benedict, 2007; Mulone and Straughan, 2011; Walters et al., 2012), drug use (White and Comiskey, 2007; Mulone and Straughan, 2009) and eating disorders (Gonzalez et al., 2003). Individuals prone to developing the behaviour are analogous to the susceptibles in infectious disease models. Similarly, those displaying the behaviour can be viewed as ‘infected’. To explain and assess the effectiveness of such models we consider an application to drinking behaviour, formulated by Sanchez et al. (2007) and further discussed by Benedict (2007). The model assumes that a total population of N individuals can be split into three distinct classes: occasional/moderate drinkers (S); problem drinkers (D); and temporarily recovered individuals (R). Homogeneous mixing of the population is assumed to occur, so an individual has an equal chance of meeting and being influenced by any other member of the population (horizontal

transmission). Implicit in this assumption is that no spatial or kinship factors affect interactions between individuals. Figure 1.1 depicts the dynamics of the system.

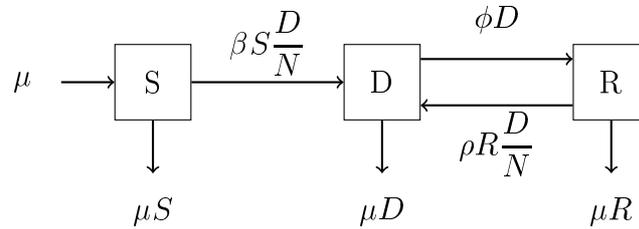


Figure 1.1: The model for problem drinking, developed by Sanchez et al. (2007). Arrows, with corresponding rates, indicate the direction of movement between the classes of moderate drinking, problem drinking and treatment. The parameter μ controls entry and exit into the modelled population.

Individuals enter the modelled population via the moderate drinking class once they reach drinking age. The assumption that the net flux of the system is zero (hence N is constant) is made to simplify the analysis. Moderate drinkers progress to the problem drinking class via a random copying mechanism, i.e. at a rate proportional to the frequency of problem drinkers within the population. This is shown by the arrow labelled $\beta SD/N$ in figure 1.1, where β is the rate at which contacts sufficient for behaviour transmission occur. A problem drinker may seek treatment, and thus enter the temporarily recovered class, without social influence at a fixed rate ϕ . This parameter could represent another type of influence, for instance an advertising campaign highlighting the harms of excessive drinking.

By considering a situation where problem drinking is so rare that a treatment programme is not required, the basic reproduction number $R_0 = \beta/\mu$ is calculated. This comprises the average length of time spent in the system, $1/\mu$, multiplied by the rate β . The basic reproduction number represents the average number of secondary cases generated from the introduction of a single problem drinker into a wholly susceptible population. If $R_0 > 1$ then, on average, more than one secondary case occurs and results in the development of a drinking culture. When $R_0 < 1$ the reproduction rate is too low for this to take place and problem drinking behaviour dies out. Thus $R_0 = 1$ is a threshold value for a problem drinking culture becoming

endemic.

Analysis through calculation of R_0 is in line with many epidemiological models for disease spread where transition through the system is one-directional, from susceptible to infected to recovered. For problem drinking, however, relapse from the temporarily recovered class to the problem drinking class is possible, thus providing an alternative supply of individuals to the drinking class D . To account for this, a second reproduction value is considered. By multiplying β by the average time spent in the problem drinking class, $1/(\mu + \phi)$, the reproduction number with a recovery class is defined as $R_\phi = \beta/(\mu + \phi)$, where $R_\phi < R_0$ for $\phi > 0$.

Unlike many epidemiological models, the basic reproduction number is not sufficient to determine the permanence of a subpopulation of problem drinkers under all circumstances. Provided the initial frequency of problem drinkers is low, then $R_0 = 1$ provides a threshold between problem drinking dying out and the behaviour persisting. This result does not apply when the initial frequency of problem drinkers is large, as the prevalence of problem drinking is greatly affected by the relapse rate. For a large initial frequency of problem drinkers and a high relapse rate, a drinking culture may emerge for $R_\phi < 1$ and is inevitable for $R_\phi > 1$. The maintenance of a drinking culture even when $R_\phi < 1$ is a result of ineffective treatment programmes. Any individuals entering treatment are unlikely to remain there for long (because of the high relapse rate) and thus quickly return to the problem drinking class D . This maintains a high frequency of problem drinkers in the population. Individuals always enter the system as moderate drinkers; if recruitment from S is minimal, indicated by a sufficiently small β value, then $R_\phi < 1$ and eventually problem drinking would die out. However, as the reproduction number is not dependent upon the relapse rate ρ , repopulation of the problem drinking class from those in treatment can offset a small recruitment rate β so that a drinking culture is maintained.

The authors conclude from the model that the reproduction number R_ϕ alone is not sufficient to predict the emergence of a drinking culture. There is also a dependence upon the initial state, especially when the recovery and relapse rates are high. This represents treatment programmes which are only short-term effective. Introducing such programmes into areas with high proportions of problem drinkers

serves to bolster the issue by creating a subgroup highly sensitive to influence from problem drinkers. Under such circumstances investing in reducing the initial uptake of drinking behaviour, controlled by the value of β , is the most effective way to tackle problem drinking.

A similar model by Mulone and Straughan (2011) splits the drinking class into admitting and non-admitting problem drinkers, an approach also used to model bulimia nervosa (Gonzalez et al., 2003). As with the previous model, individuals enter the system as moderate drinkers, referred to as susceptibles by the authors. Through interactions with current problem drinkers, susceptibles can move to being a non-admitting problem drinker as, initially, they are not aware that their behaviour is problematic. Only after advancement to the admitting class, through realising that they have a drinking problem, can individuals enter treatment. Both of these transitions are assumed to occur at constant rates. Different to the Sanchez et al. (2007) model, the relapse rate of those in treatment is taken to be a result of genetic factors rather than social influence, so the constant relapse rate to the admitting class is ρR . The basic reproduction number of the system is calculated, whereby R_0 increasing indicates a move from a problem- drinking-free state to one in which it is endemic. Using parameter estimates obtained from data for binge drinking behaviour in the North East of England, model predictions indicate that the behaviour will persist, plateauing when approximately 15% of the population reside in the drinking classes.

Sanchez et al. (2007) acknowledge that their model does not allow for a return to different drinking classes, an issue addressed by Walters et al. (2012) by allowing transition from the treatment class back to a moderate drinking state. The additional transition was introduced to account for the possibility of total recovery from problem drinking, so individuals return to what is termed as the susceptible class after completion of a treatment programme. By performing a sensitivity analysis of the value R_0 , the authors conclude that the most effective way to reduce drinking is by focusing on reducing the number of susceptible individuals that are recruited to the problem drinking class, concurring with the conclusions of Sanchez et al. (2007). Whilst this development addresses one concern of the Sanchez et al. (2007) model,

it is still an oversimplification of the dynamics. The primary issue is that the model assumes that an individual who has completed treatment will be equally susceptible to the influence of problem drinkers as someone that has just entered the population.

Whilst a variety of alternative models have been put forward to focus on different aspects of drinking behaviour, some key underlying assumptions feature which reduce their practical applications. For example, the level of contact between individuals is not the same for every possible pairing, so homogeneous mixing is an over-simplification. One possible refinement to such models is to introduce a network structure so that, in order for individuals to interact, they must be connected within the network. This approach was adopted by Gorman et al. (2006) where a 3-stage SDR alcohol model (with corresponding classes susceptible, drinkers, and former drinkers) was implemented on a network structure. Each network node corresponded to a location containing a subset of the total population, split into the three classes. At each time step individuals could move location and their class status could change in accordance with the described SDR dynamics.

This type of modelling is very powerful as often analytic results can be obtained, offering a certain outcome for all possible parameter combinations. As a trade-off, many simplifying assumptions are required for such analysis to be possible and it is important to tailor the model to the specific questions of interest, highlighted here by the variety of models discussed. For example, Sanchez et al. (2007) were interested in analysing a model where social influence was the driving factor for individuals to become problem drinkers, whereas Mulone and Straughan (2011) and Walters et al. (2012) do not consider a peer-influenced relapse term. Instead they focus on alternative aspects: the former on the eligibility of individuals to enter a treatment process by admitting they have a problem, and the latter on the effects of total recovery from an alcohol problem. Ideally a single model would consider all of these factors and more, but analysis would then become impossible. Despite this, the modelling technique does allow for population-wide information to be gained and can provide cheap and quick predictions regarding how top-down interventions may affect the system.

Chapters 2 and 3 were both motivated by the application of SIR-type models ap-

plied to horizontally transmitted behaviours. The SARS model in chapter 2 extends existing work by Mulone and Straughan (2011) to determine the effect of complete recovery from an alcohol problem on the basic reproduction number R_0 and the endemic equilibrium value. In chapter 3 an SAS model for cultural trait transmission is formulated, where the transmission of the trait may be subject to conformist and content biases. Whilst both models are constructed within an SIR-type framework, the varying complexities in terms of model variables and variable parameters require different methods of analysis to be conducted.

1.6 Mathematical models with spatial variation

Other developments of SIR (and similar) models have been analysed where the variable S , I and R may depend on more than just time. For instance, Murray (2003) discusses an SIS infectious disease model where the age, a , of the individual affects their vulnerability to catching the disease and their infectiousness, mathematically represented by $I = I(t, a)$. Infectious disease models with a spatial dependence have also been formulated. These represent the ability of individuals to move between regions, as with the patchy environment ODE model by Wang and Zhao (2004), or within a specified region where the variables have a spatial dependence (Mulone et al., 2007). For disease transmission to occur, an infected individual must be in the same location as a susceptible individual. As transmission is localised, the diffusion of infected individuals is required for the disease to spread (Keeling and Rohani, 2008). This can be represented by a reaction-diffusion PDE system of equations, as in Keeling and Rohani (2008), and similarly for an SIS model (without cross-diffusion) in Mulone et al. (2007).

These reaction-diffusion systems have the form

$$\frac{\partial u_i}{\partial t} = D_i \Delta u_i + f_i(u_1, \dots, u_m) \quad (1.2)$$

in $\Omega \times (0, \infty)$ for $i = 1, \dots, m$. Each u_i represents a class of individuals so, in the reaction-diffusion SIR model (Keeling and Rohani, 2008), $m = 3$ as individuals can either be susceptible (u_1), infected (u_2) or recovered (u_3). The area in which individuals can traverse is represented by the domain Ω , which has a boundary $\partial\Omega$.

For a model of disease spread, Ω may represent a particular country or region with $\partial\Omega$ defined by country borders or a geographically boundary, as with an island. In two spatial dimensions, $u_i = u_i(x, y, t)$ and represents the density of class u_i at location (x, y) at time t . The change in this density in time is controlled by a diffusion part, $D_i\Delta u_i$, and a reaction part, $f(u_1, \dots, u_m)$ (Kandler and Unger, 2010). Diffusion of u_i is controlled by the Laplace operator, which in two spatial dimensions is

$$\Delta = \frac{\partial^2}{\partial x^2} + \frac{\partial^2}{\partial y^2}, \quad (1.3)$$

and the diffusion coefficient D_i . The movement of individuals is from crowded areas to less populated areas which, in terms of human population spread, may represent moving to an area with a greater proportion of available or preferable resources, such as food or shelter (Mulone et al., 2007). The coefficient D_i represents the tendency of individuals to move to lesser populated regions. The reaction term is given by the function f_i and describes the remaining system dynamics, such as population growth or interaction terms between susceptible and infected individuals.

To solve for unique solutions to PDE problems, further conditions must be imposed (Strauss, 2008). Initial conditions describe the state of the system at time $t = 0$, an example being $u_i(x, y, 0) = g(x, y)$ for some function g . Boundary conditions can also be imposed which place constraints on the system at the boundary $\partial\Omega$. Two common types of boundary condition are Dirichlet conditions and Neumann conditions. Dirichlet conditions are when the function u_i is specified at the boundary, for example $u_i(x, y, t) = 0$ on $\partial\Omega$. This example means that no u_i individuals can exist at the boundary of the spatial region. This may be an appropriate assumption to make if individuals live on an island where areas at the edge are uninhabitable or undesirable locations due to environmental factors. Neumann conditions are where the normal derivative is specified at the boundary. For example if $\partial u_i / \partial n = 0$ on $\partial\Omega$, there is no flux across the boundary $\partial\Omega$ in the direction of the outward-pointing unit normal to the region, n . This example condition means that individuals are confined to stay within the region Ω , be that an island or country, but they may be located at the boundary $\partial\Omega$. The choice of boundary conditions is dependent upon the characteristics of the real-world situation which is being modelled.

One of the simplest nonlinear reaction-diffusion equations is

$$\frac{\partial u}{\partial t} = D \frac{\partial^2 u}{\partial x^2} + ku(1 - u), \quad (1.4)$$

known as the Fisher-Kolmogorov equation (Murray, 2003). Fisher (1937) proposed the model to represent the spread of an advantageous gene within a population and Kolmogorov et al. (1937) analysed a more general form of the model with the reaction component $ku(1 - u)$ replaced by a general function $f(u)$ (Murray, 2003). An analysis of this model and the associated travelling wave solutions may be found in Murray (2003). The Fisher-Kolmogorov equation also has applications in cultural evolutionary theory, such as in models for the evolution of both genes and culture (Aoki, 1987; Straughan, 2013a). One model considers a similar formulation for the spread of farmers into a region populated by hunter-gatherers (Aoki et al., 1996). Individuals can either be farmers (F), hunter-gatherers (H) or hunter-gatherers who have converted to farming (C). Initially farmers are localised, with the remaining area populated by hunter-gatherers. As the farmers migrate (initially F , then $F+C$) they interact with hunter-gatherers, resulting in some conversion of hunter-gatherers to (converted) farmers. It is assumed that the growth rate of converted farmers is greater than that of hunter-gatherers, suggesting that a conversion to farming may be advantageous to the hunter-gatherer population. The analysis presented in the paper indicates that farming will dominate and hunter-gatherers will become extinct, with the subpopulation growth rates affecting whether the surviving farming population comprises original farmers or converted farmers.

The use of ODE and PDE models has been expanded to investigate the spread of languages which are in competition for speakers (Abrams and Strogatz, 2003; Patriarca and Leppänen, 2004; Pinasco and Romanelli, 2006; Kandler and Steele, 2008). Motivated by current research in this area, chapter 4 includes a discussion of this literature and an analysis of a reaction-diffusion model for two competing languages.

Chapter 2

Modelling alcohol problems: Total recovery

2.1 Introduction

Smith and Foxcroft (2009) report that between 1998 to 2006 there was an overall increase in the proportion of individuals in Great Britain who exceed the recommended alcohol consumption limits, including a doubling of the proportion of women who binge drink. Excessive alcohol consumption can lead to a range of negative health and social effects (House of Commons Science and Technology Committee, 2012) and it is estimated that alcohol misuse costs the NHS £2.7 billion per year, with alcohol related hospital admissions having increased by 100% from 2002/03 to 2009/10 (Alcohol Concern, 2011). These figures suggest that there is an increasing trend of alcohol misuse, which is resulting in costs to health and the economy. Here we devise a predictive mathematical model which may offer an insight into the best strategy for tackling problems with alcohol and, in particular, binge drinking.

Mathematical models for behaviours such as alcoholism have been developed from epidemiological models for the spread of infectious diseases. One of the first infectious disease models by Kermack and McKendrick (1927) considers a constant population where individuals are split into those that are susceptible to catching the disease (S), infected individuals (I) and immune or dead individuals (R). To maintain a constant population, immune individuals and those that have died from

the disease enter the removed class, and so models of this form have become known as SIR models. Developments of SIR models and their extensions continue to be employed to describe various scenarios in mathematical epidemics: cf. Murray (2003); Wang and Mulone (2003); Wang and Ruan (2004); Wang and Zhao (2004); Boni and Feldman (2005); Lou and Ruggeri (2007); Buonomo and Lacitignola (2008); Capone (2008); Keeling and Rohani (2008); Li et al. (2008); Ma and Li (2009); Buonomo and Rionero (2010); Buonomo et al. (2010); Mulone et al. (2011); Rionero (2012b); Rionero and Vitiello (2012).

Another development of such models has been to apply them to situations where it is assumed that social interaction is the key factor in spreading the behaviour. Behaviours which can result in adverse health effects have been represented, such as drinking (Sanchez et al., 2007; Benedict, 2007; Manthey et al., 2008; Mubayi et al., 2010; Santonja et al., 2010; Mulone and Straughan, 2011), smoking (Sharomi and Gumel, 2008), drug use (White and Comiskey, 2007; Mulone and Straughan, 2009), obesity (Jodar et al., 2008; Hill et al., 2010b) and eating disorders (Gonzalez et al., 2003). Even though the models for each social problem may appear mathematically similar at the onset, there are fundamental differences which must be catered for. For example, a small intake of alcohol may be beneficial to health as shown by the J-shaped curve of alcohol intake against health problems (Marmot and Brunner, 1991; Kloner and Rezkalla, 2007). For smoking however, the graph of amount smoked against health problems immediately has an increasing gradient, indicating the detrimental effect of smoking on health.

In this paper we develop a three-stage model which represents the effect of social influence on drinking habits, with a particular interest in total recovery. The total population is split into susceptible individuals, individuals with alcohol problems and individuals in treatment. Susceptible individuals are those who do not consume alcohol in a way defined to be problematic. We refer to alcohol problems in general as the model is applicable to a variety of drinking behaviours, for example dependent drinkers who drink every day or binge drinkers who consume many units in one session. The precise definitions of each class must be determined by the nature of the behaviour being modelled, which we demonstrate in section 2.2.5 using

information regarding binge drinking. We consider the recovered class to represent those receiving treatment. Whilst it is possible for individuals to tackle an alcohol problem without professional help, data regarding the number of individuals opting for this approach is unavailable. If such information were to become available then a change to the definition of the class would perhaps be appropriate. The definition of treatment may also vary depending on the nature of the problem and any associated withdrawal effects.

The three subpopulations are similar to the classes defined in the work of Sanchez et al. (2007) (also see Benedict (2007)) and Mulone and Straughan (2011), however neither of these models allow for total recovery. Sanchez et al. (2007) found that the basic reproduction number alone is not always the key factor in controlling drinking in the population. Mulone and Straughan (2011) extended their model by splitting binge drinkers into those who admit that they have a problem and those that do not admit. Using data for the north east of England, they conclude that binge drinking is sustainable in the population.

Other models, by Manthey et al. (2008); Mubayi et al. (2010) and Santonja et al. (2010), do not contain a treatment class but instead split the population into three classes depending on the amount of alcohol an individual consumes. Manthey et al. (2008) consider a students' 5-year period in a university campus environment, which is deemed too short for recovery to be determined. Mubayi et al. (2010) also focused on the drinking habits of students, but they were interested in assessing how a change from low to high risk drinking environments affected the transition from susceptible to heavy drinker. Santonja et al. (2010) do not consider a treatment class, despite an individual spending 50 years in the system, as the aim of the work is to determine the health and economic costs of risky alcohol consumption. This is determined by the average alcohol intake alone, irrespective of any recovery process.

We have chosen to include a treatment class as we aim to discover the most effective way to reduce the proportion of the population in the alcohol problems class. Such information may be useful to health professionals and policy makers when devising strategies aimed at reducing the proportion of the population suffering from alcohol problems. We also allow for individuals to completely recover from

their alcohol problem. The motivation for this stems from recent ideas regarding the nature of recovery.

Best (2010) discusses various definitions of recovery and introduces the concept of recovery champions. These champions are individuals who have successfully recovered from misusing alcohol, or other similar problems, and appear as a role model or an example of success to inspire those currently in treatment. The UK Drug Policy Commission Recovery Consensus Group report (July 2008) does not contain a precise definition of recovery as it is an individual process, i.e. recovery cannot be given a fixed definition which applies to the whole population as it varies depending on the individual. Instead, a set of key principles of recovery are presented. The report concludes that some individuals will always remain in treatment, whereas others will feel that they are fully recovered. To accommodate both these options, we allow for individuals to move from the treatment class back to the susceptible population at a given rate.

The model we construct considers a population of N individuals separated into the three subclasses, represented by a system of three ordinary differential equations. Susceptible individuals, denoted by $S(t)$ where t is time, are those without an alcohol problem. We assume that a susceptible individual develops an alcohol problem through interactions with those in the alcohol problems class, $A(t)$. Finally an individual may be in the treatment class, $R(t)$, from which they may relapse and hence return to $A(t)$. Alternatively, an individual may remain in treatment for a sufficient length of time so that they totally recover, at which point they return to the susceptible population as they are no longer experiencing difficulties with alcohol.

Using stability analysis we calculate a critical threshold value, R_0 , which, once exceeded, determines that alcohol problems will persist in the population. Sensitivity analysis reveals which parameter has the greatest influence on this threshold value and thus may provide valuable insights into the most effective way of tackling alcohol misuse in the population. We then consider the stability of the endemic equilibrium solution and compare our results with the case where total recovery is not possible. Finally, we use numerical simulations to predict the future proportion

of binge drinkers in England.

2.2 The mathematical model

As stated in the introduction, we consider a population of N individuals and split them into three classes: $S(t)$, $A(t)$ and $R(t)$. The probability that a susceptible individual has contact with someone in the alcohol problems class is A/N . Not all such contacts will be sufficient for the susceptible individual to develop an alcohol problem, so we define β to be the rate at which sufficient contacts occur. This gives us the rate at which individuals move from being susceptible to having an alcohol problem as $\beta AS/N$. This sort of transmission term has been employed in modelling drug and alcohol problems, cf. Sanchez et al. (2007); Benedict (2007); Manthey et al. (2008); Santonja et al. (2010)

Individuals may move to the recovery class by entering a treatment programme, which we assume occurs at a constant rate φ . Once in treatment, an individual can either relapse or they can recover. Relapsing back to $A(t)$ is also assumed to happen at a constant rate, ρ , whereas recovery, and hence return to the susceptible class, is assumed to happen at a constant rate γ . We assume that individuals enter and leave the population at the same constant rate μ , where $1/\mu$ represents the average length of time spent in the system. The dynamics of this SAR system are given by the equations

$$\begin{aligned}\dot{S} &= \mu N - \frac{\beta AS}{N} + \gamma R - \mu S, \\ \dot{A} &= \frac{\beta AS}{N} + \rho R - (\varphi + \mu)A, \\ \dot{R} &= \varphi A - (\rho + \mu + \gamma)R,\end{aligned}\tag{2.1}$$

where the total population is given by $N = S + A + R$ with $N > 0$, $S \geq 0$, $A \geq 0$ and $R \geq 0$.

To preserve the direction of flow through the system (see figure 2.1), we take only positive values for the parameters $\beta, \mu, \varphi, \rho$ and γ . Following the method in Mulone and Straughan (2011), we now introduce the variables $s(t) = S(t)/N$, $a(t) = A(t)/N$

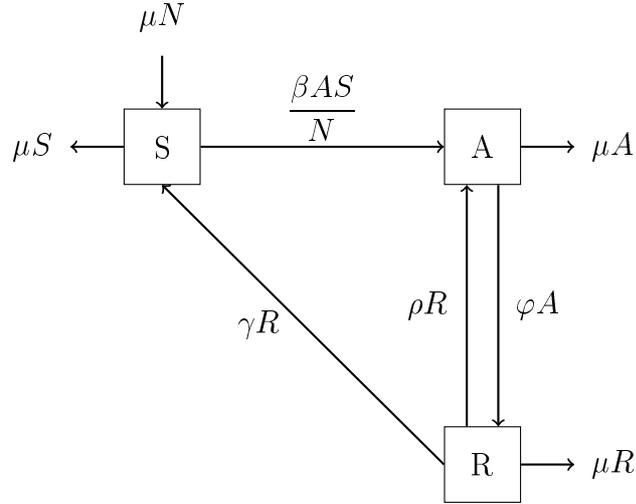


Figure 2.1: Flow diagram showing the movement between the three subpopulations $S(t)$, $A(t)$ and $R(t)$.

and $r(t) = R(t)/N$, which enables us to rewrite system (2.1) as

$$\begin{aligned}
 \dot{s} &= \mu - \beta as + \gamma r - \mu s, \\
 \dot{a} &= \beta as + \rho r - (\varphi + \mu)a, \\
 \dot{r} &= \varphi a - (\rho + \mu + \gamma)r,
 \end{aligned} \tag{2.2}$$

where $1 = s + a + r$. As $s = 1 - a - r$, we can reduce system (2.2) to the two equations

$$\begin{aligned}
 \dot{a} &= -\beta a^2 - \beta ar + (\beta - \varphi - \mu)a + \rho r, \\
 \dot{r} &= \varphi a - (\rho + \mu + \gamma)r.
 \end{aligned} \tag{2.3}$$

2.2.1 Stability analysis

We solve equations (2.3) to find the equilibrium points of the system, which are the problem-free solution $(a, r) = (0, 0)$ and the endemic solution $(a, r) = (\bar{a}, \bar{r})$. We will now analyse the local stability of the problem-free equilibrium solution by considering a linearisation of system (2.3) at $(a, r) = (0, 0)$. The linearisation of equations (2.3) around a general point (\hat{a}, \hat{r}) is given by $\dot{\mathbf{a}} = J(\hat{\mathbf{a}})(\mathbf{a} - \hat{\mathbf{a}})$ where J

is the Jacobian matrix and \mathbf{a} is the vector

$$\mathbf{a} = \begin{pmatrix} a \\ r \end{pmatrix}. \quad (2.4)$$

The Jacobian matrix at the point $(0, 0)$ is given by

$$J(0, 0) = \begin{pmatrix} (\beta - \varphi - \mu) & \rho \\ \varphi & -(\rho + \mu + \gamma) \end{pmatrix},$$

which has eigenvalues

$$\sigma_+ = \frac{-x_1 + \sqrt{x_1^2 - 4y_1}}{2} \quad \text{and} \quad \sigma_- = \frac{-x_1 - \sqrt{x_1^2 - 4y_1}}{2} \quad (2.5)$$

where

$$\begin{aligned} x_1 &= \varphi + \rho + 2\mu + \gamma - \beta, \\ y_1 &= -\rho\varphi + (\rho + \mu + \gamma)(\varphi + \mu - \beta). \end{aligned}$$

For the problem-free equilibrium point to be locally asymptotically stable we require the real part of both eigenvalues to be negative. This is true provided $x_1 > 0$ and $y_1 > 0$. It is sufficient to consider $y_1 > 0$ only as this condition guarantees $x_1 > 0$ (see appendix B.1), from which we determine that the inequality

$$\frac{\beta(\rho + \mu + \gamma)}{\mu(\rho + \mu + \gamma + \varphi) + \gamma\varphi} < 1 \quad (2.6)$$

must hold for the equilibrium point to be locally asymptotically stable. If this situation arises then alcohol problems will eventually die out in the population. If inequality (2.6) is reversed then the equilibrium solution is unstable and alcohol problems may persist in the population. We now define the basic reproduction number R_0 to be

$$R_0 := \frac{\beta(\rho + \mu + \gamma)}{\mu(\rho + \mu + \gamma + \varphi) + \gamma\varphi}, \quad (2.7)$$

where $R_0 < 1$ indicates stability and $R_0 > 1$ indicates instability of the problem-free equilibrium solution.

2.2.2 Endemic equilibrium solution

The second equilibrium solution of system (2.3) is $(a, r) = (\bar{a}, \bar{r})$, where

$$\begin{cases} \bar{a} = \frac{\beta(\rho + \mu + \gamma) - \mu(\rho + \mu + \gamma + \varphi) - \gamma\varphi}{\beta(\rho + \mu + \gamma + \varphi)}, \\ \bar{r} = \frac{\varphi}{(\rho + \mu + \gamma)} \cdot \frac{\beta(\rho + \mu + \gamma) - \mu(\rho + \mu + \gamma + \varphi) - \gamma\varphi}{\beta(\rho + \mu + \gamma + \varphi)}, \end{cases} \quad (2.8)$$

and only exists for $R_0 > 1$. The Jacobian of equations (2.3) at the point (\bar{a}, \bar{r}) is

$$J(\bar{a}, \bar{r}) = \begin{pmatrix} -2\beta\bar{a} - \beta\bar{r} + (\beta - \varphi - \mu) & \rho - \beta\bar{a} \\ \varphi & -(\rho + \mu + \gamma) \end{pmatrix} \quad (2.9)$$

and the corresponding eigenvalues are given by

$$\tilde{\sigma}_+ = \frac{-x_2 + \sqrt{x_2^2 - 4y_2}}{2} \quad \text{and} \quad \tilde{\sigma}_- = \frac{-x_2 - \sqrt{x_2^2 - 4y_2}}{2}, \quad (2.10)$$

where

$$\begin{aligned} x_2 &= 2\beta\bar{a} + \beta\bar{r} + \varphi + 2\mu + \rho + \gamma - \beta, \\ y_2 &= \varphi(\beta\bar{a} - \rho) + (\rho + \mu + \gamma)(2\beta\bar{a} + \beta\bar{r} + \varphi + \mu - \beta). \end{aligned}$$

For the equilibrium solution (\bar{a}, \bar{r}) to be linearly asymptotically stable then $x_2 > 0$ and $y_2 > 0$ must hold. Appendix B.2.1 gives the calculations which show that $x_2 > 0$ is always true provided $y_2 > 0$, so to find the local stability conditions we need only consider $y_2 > 0$. We can write y_2 in terms of the model parameters only by substituting in the values for \bar{a} and \bar{r} , which is shown in appendix B.2.2. From this we find that the inequality $y_2 > 0$ can be written as

$$\beta(\rho + \mu + \gamma) - \mu(\rho + \mu + \gamma + \varphi) - \gamma\varphi > 0.$$

This can be rearranged to give

$$1 < \frac{\beta(\rho + \mu + \gamma)}{\mu(\rho + \mu + \gamma + \varphi) + \gamma\varphi},$$

which is equivalent to $R_0 > 1$. From this we know that (\bar{a}, \bar{r}) is locally asymptotically stable when it exists, hence alcohol problems become endemic provided $R_0 > 1$.

Our analysis reveals that the equilibrium point $(0, 0)$ is locally asymptotically stable for $R_0 < 1$, whereas (\bar{a}, \bar{r}) is locally asymptotically stable for $R_0 > 1$. We can

see that the value of R_0 determines whether alcohol problems will die out or become endemic in the population and so we consider $R_0 = 1$ to be an invasion threshold value.

2.2.3 Sensitivity analysis

Having defined $R_0 = 1$ as a threshold for the invasion of alcohol problems, we are now interested in which model parameter has the greatest effect on R_0 and hence has the greatest effect in determining whether alcohol problems will persist in the population. To this end, we calculate the normalised sensitivity index, $NSI = (k/R_0)(\partial R_0/\partial k)$, which indicates how sensitive R_0 is to a change in some parameter k , where normalisation allows for a direct comparison between parameters. A negative normalised sensitivity index indicates that an increase in the parameter value results in a decrease in the R_0 value. As we are only interested in the magnitude of the change to the R_0 value, we consider the absolute value. The normalised sensitivity indices for the parameters are

$$\begin{aligned} \left| \frac{\beta}{R_0} \frac{\partial R_0}{\partial \beta} \right| &= 1, \\ \left| \frac{\mu}{R_0} \frac{\partial R_0}{\partial \mu} \right| &= \frac{\mu(\mu + \gamma)(\rho + \mu + \gamma) + \rho\mu(\rho + \mu + \gamma + \varphi)}{\mu(\mu + \gamma)(\rho + \mu + \gamma) + \rho\mu(\rho + \mu + \gamma + \varphi) + G} < 1, \\ \left| \frac{\rho}{R_0} \frac{\partial R_0}{\partial \rho} \right| &= \frac{\rho\varphi(\mu + \gamma)}{\rho\varphi(\mu + \gamma) + \varphi(\mu + \gamma)^2 + \mu(\rho + \mu + \gamma)^2} < 1, \\ \left| \frac{\varphi}{R_0} \frac{\partial R_0}{\partial \varphi} \right| &= \frac{\varphi(\mu + \gamma)}{\varphi(\mu + \gamma) + \mu(\rho + \mu + \gamma)} < 1, \\ \left| \frac{\gamma}{R_0} \frac{\partial R_0}{\partial \gamma} \right| &= \frac{\rho\gamma\varphi}{\rho\gamma\varphi + \gamma\varphi(\mu + \gamma) + \mu(\rho + \mu + \gamma)(\rho + \mu + \gamma + \varphi)} < 1, \end{aligned}$$

where $G = \gamma(\mu + \varphi)(\rho + \mu + \gamma) + \mu\varphi(\rho + \gamma)$.

From the calculations here we can see that R_0 is most sensitive to changes in the value of β , which represents the rate at which social interaction mediates the development of alcohol problems. We can see that equation (2.7) for R_0 has the form $R_0 = c\beta$ where

$$c = \frac{(\rho + \mu + \gamma)}{\mu(\rho + \mu + \gamma + \varphi) + \gamma\varphi}$$

so a factor α change in the β value results in a factor α change in the value of R_0 .

2.2.4 The effect of individuals in treatment returning to the susceptible population

We now compare the model with the situation where movement from $R(t)$ to $S(t)$ is removed, so complete recovery from an alcohol problem is not possible. This is achieved by allowing $\gamma = 0$, resulting in the model proposed by Mulone and Straughan (2011). We focus on how $\gamma = 0$ affects the basic reproduction number and the endemic equilibrium solution. With this comparison we aim to highlight any qualitative differences between the solutions of the two models.

Basic reproduction number

The basic reproduction number is given by equation (2.7). For the case where $\gamma = 0$ we define the basic reproduction number by \tilde{R}_0 , where

$$\tilde{R}_0 = \frac{\beta(\rho + \mu)}{\mu(\rho + \mu + \varphi)}.$$

To study the effect that $\gamma > 0$ has on the basic reproduction, the difference between R_0 and \tilde{R}_0 is calculated:

$$R_0 - \tilde{R}_0 = \frac{-\beta\rho\varphi\gamma}{\mu(\mu + \rho + \varphi)(\mu^2 + \mu\rho + \mu\gamma + \mu\varphi + \varphi\gamma)}. \quad (2.11)$$

As the right-hand side of equation (2.11) is always negative, we conclude that $R_0 < \tilde{R}_0$ for all possible parameter values. Thus excluding the return to the susceptible class increases the average number of secondary infections which result from a single infected individual entering a wholly susceptible population.

As R_0 is the average number of secondary cases which arise from a single infected being introduced into a wholly susceptible population, we know that by taking $\gamma = 0$ the average number of secondary cases increases. This means that a single infected individual will infect a greater proportion of the population when compared to the case where $\gamma > 0$.

Endemic equilibrium solution

Next we look at the change to the endemic equilibrium solution, $(\bar{s}, \bar{a}, \bar{r})$. The equilibrium value for the susceptible population, \bar{s} , is calculated using the expressions

for \bar{a} and \bar{r} from system of equations (2.8) in $\bar{s} = 1 - \bar{a} - \bar{r}$, resulting in

$$\bar{s} = \frac{\mu(\rho + \mu + \gamma + \varphi) + \gamma\varphi}{\beta(\rho + \mu + \gamma)}. \quad (2.12)$$

The special case $\gamma = 0$ has the endemic equilibrium solution $(\tilde{s}, \tilde{a}, \tilde{r})$ with

$$\begin{aligned} \tilde{s} &= \frac{\mu(\rho + \mu + \varphi)}{\beta(\rho + \mu)}, \\ \tilde{a} &= \frac{\beta(\rho + \mu) - \mu(\rho + \mu + \varphi)}{\beta(\rho + \mu + \varphi)}, \\ \tilde{r} &= \frac{\varphi}{(\rho + \mu)} \cdot \frac{\beta(\rho + \mu) - \mu(\rho + \mu + \varphi)}{\beta(\rho + \mu + \varphi)}. \end{aligned}$$

To study the effect that γ has on each individual class, we compare the solution $(\bar{s}, \bar{a}, \bar{r})$ with the $\gamma = 0$ solution $(\tilde{s}, \tilde{a}, \tilde{r})$.

We begin by examining the susceptible population. The equation

$$\bar{s} - \tilde{s} = \frac{\rho\gamma\varphi}{\beta(\rho + \mu)(\rho + \mu + \gamma)}$$

is always positive, so $\bar{s} > \tilde{s}$. Hence, removing the possibility of total recovery reduces the proportion of individuals in the susceptible class.

For the alcohol problems class, the change in the proportion of individuals in the class is not as simple as the previous case. The difference between the two equilibrium values is given by

$$\bar{a} - \tilde{a} = \frac{\gamma\varphi(\beta - \rho - \mu - \varphi)}{\beta(\rho + \mu + \gamma + \varphi)(\rho + \mu + \varphi)}.$$

The relationship between \bar{a} and \tilde{a} is determined by the sign of the expression $\beta - \rho - \mu - \varphi$. If $\beta > \rho + \mu + \varphi$ then $\bar{a} > \tilde{a}$, so preventing a return to the susceptible class results in a decrease in the proportion of the population with alcohol problems. If $\beta < \rho + \mu + \varphi$ the converse is true so $\bar{a} < \tilde{a}$. Finally, $\bar{a} = \tilde{a}$ only when $\beta = \rho + \mu + \varphi$. This expression is independent of γ , so it is possible for the two models to agree on the proportion of alcoholics in the population.

For the recovered class, we find that

$$\bar{r} - \tilde{r} = \frac{\gamma\varphi F}{\beta(\rho + \mu)(\rho + \mu + \gamma)(\rho + \mu + \varphi)(\rho + \mu + \gamma + \varphi)} \quad (2.13)$$

where

$$F = -\beta(\rho + \mu)(\rho + \mu + \gamma) + \mu(\rho + \mu)(\rho + \mu + \gamma) + \mu\varphi(\mu + \gamma) - \rho\varphi(\rho + \varphi). \quad (2.14)$$

As the denominator of the right-hand side of equation (2.13) is always positive, the sign of $\bar{r} - \tilde{r}$ depends on the numerator, specifically on the value of F . As we are considering the endemic equilibrium solution, the parameters are constrained by the inequality $R_0 > 1$. We use this information to determine that $F < 0$ must be true and hence $\bar{r} < \tilde{r}$ (see appendix B.4). Thus, removing the option of returning to the susceptible class results in an increase in the proportion of individuals in the recovered class.

Conclusions

Assuming that β , μ , ρ and φ are fixed, when $\gamma = 0$ the basic reproduction number is increased, i.e. the average number of secondary infections resulting from a single infected being introduced into a wholly susceptible population is increased. For example, we can consider a situation where $\tilde{R}_0 = 1$, thus $R_0 < 1$, and consider increasing the value of β . This increase instantly results in alcohol problems becoming endemic when $\gamma = 0$. When $\gamma > 0$ however, we find that the rate at which susceptible individuals develop alcohol problems may be increased without resulting in alcohol problems becoming endemic.

Alternatively we could consider R_0 as a strictly decreasing function of γ . In some situations, determined by the other parameter values, an increase in γ changes the stable equilibrium from the endemic to the alcohol problems-free solution. Increased γ values indicate that more individuals recover from an alcohol problem, perhaps achievable by improvements to treatment services which discourage individuals from relapsing. An example situation is shown in figure 2.2. When $\gamma = 0.550$, $R_0 = 1$ and the stable equilibrium moves from the endemic solution to the alcohol problems-free solution. This example highlights the importance in understanding the key variables that affect drinking behaviour. By comparing the model without total recovery (equating to considering $\gamma = 0$) with the model where $\gamma > 0.550$, we see that the value of γ affects the qualitative nature of the system and hence offers different predictions.

When $\gamma = 0$, the endemic equilibrium solution has a decreased proportion of susceptible individuals and an increased proportion of those in treatment. This

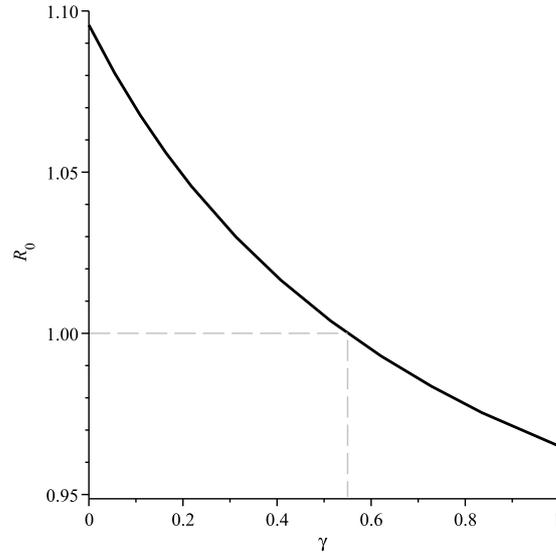


Figure 2.2: A change in the stability of the two equilibrium solutions occurs when $R_0 = 1$ at $\gamma = 0.550$. The fixed parameter values are $\mu = 0.25$, $\beta = 0.3$, $\rho = 0.8$ and $\varphi = 0.1$.

result is intuitive as $\gamma = 0$ prevents individuals moving from the treatment class back to the susceptible population. The relationship between γ and the alcohol problems class is not so obvious as whether there is an increase or a decrease does not depend on the value of γ , but the magnitude of the effect does. Recall that if $\beta < \rho + \mu + \varphi$ then $\gamma = 0$ increases the proportion of individuals in the alcohol class but if $\beta > \rho + \mu + \varphi$ then the proportion is decreased.

Assuming ρ , μ and φ are fixed, then the susceptible population is increased when $\gamma > 0$. As γ relates to totally recovered individuals, $\gamma > 0$ also results in a reduction in the treatment class size and hence fewer individuals available to relapse. If β is small then we have an increased number of individuals in the susceptible class, each with only a small chance of developing alcohol problems. The only way to repopulate $A(t)$ is by individuals who relapse or by susceptible individuals developing an alcohol problem. By combining small β with $\gamma > 0$, the number of individuals available to relapse is small and the chance that susceptible individuals will develop alcohol problems is low. From this we conclude that $\gamma > 0$ decreases the proportion of individuals with alcohol problems when β is small. Conversely, if β is large and $\gamma > 0$ then we have an increased number of susceptible individuals, each with a

large chance of developing alcohol problems, so we intuitively expect an increase in the proportion of those with alcohol problems in the population.

2.2.5 A model for binge drinking in England

We now estimate the parameter values based on recent information regarding binge drinking in England. Social influence, for example through social norms and peer pressure, is often considered to play a key role in binge drinking (Gill, 2002; French and Cooke, 2012). As our model represents the effect of social influence on drinking behaviour, it is appropriate to apply it to the situation of binge drinking. We shall consider those who binge drink to form the alcohol problems class.

The term binge drinking has not been strictly defined. However, according to Deacon et al. (2007), a binge drinker is usually regarded as someone who regularly consumes at least twice the guideline daily units of alcohol during the heaviest drinking day of the week. The UK guidelines state that binge drinking is consuming 8+ units for men and 6+ units for women in a single session. It is possible that an individual may occasionally binge drink in accordance with these guidelines, but this behaviour may be very rare and hence not indicative of a drinking problem. The data available in Deacon et al. (2007) considers one week only so may include information on infrequent binge drinkers, however it can be used as an upper bound when wishing to determine the proportion of regular binge drinkers in the population.

According to Jones et al. (2008), the government aims to reduce the harm caused by 18-24 year old binge drinkers so we shall restrict our population to this age group. In Britain binge drinking is most prevalent among young adults, though it is not restricted to this age group with those that binge drink in their early 20s being more likely to do so in their 40s than those that do not binge drink (Institute of Alcohol Studies, 2010). With this in mind, we argue that tackling the current problem of binge drinking will not only reduce antisocial behaviour and alcohol related accidents now, but may also contribute to reducing the number of individuals with alcohol-related illnesses and alcohol dependence in the future. The 18-24 year old age group spend a total of 7 years in the system so we take $\mu = 1/7 = 0.143$. Information for the number of binge drinkers in treatment could not be obtained so we shall

assume that it is the same proportion as for dependent drinkers, which is 6% of the drinking population according to the 2011 National Institute for Health and Clinical Excellence (NICE) report. We therefore consider the maximum annual probability of entering treatment to be 0.06, which we can convert to a rate using

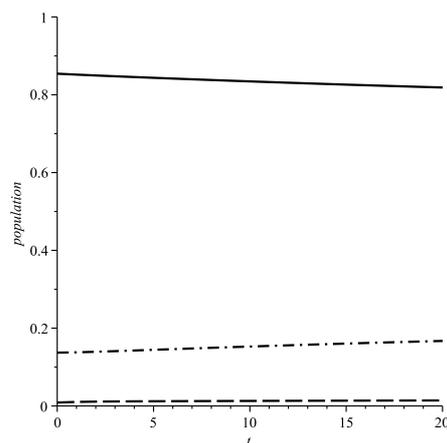
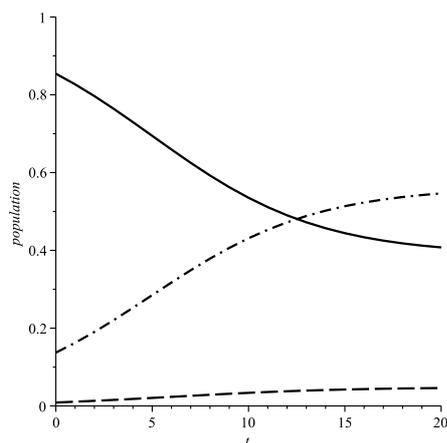
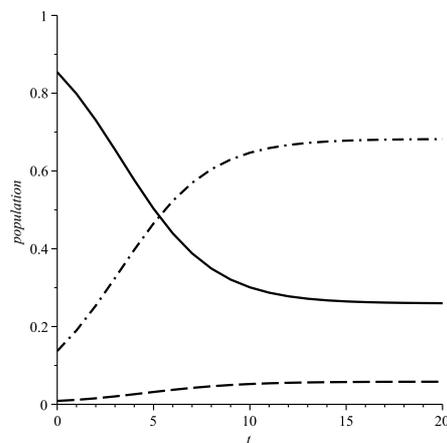
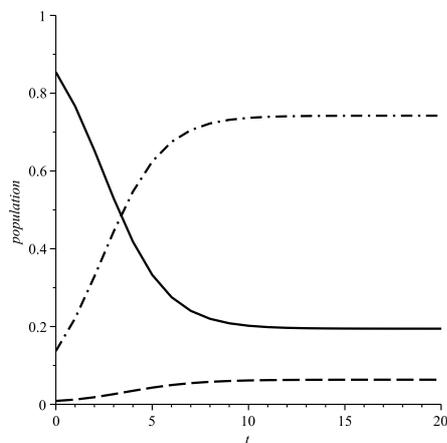
$$\text{rate} = \frac{-\ln(1-p)}{t} \quad (2.15)$$

where p is the probability of an event over the time period t . Using this equation we calculate a maximum value of $\varphi = 0.0619$.

El Sheikh and Bashir (2004) report that 35% of alcoholics in treatment relapse within the first 2 weeks and 58% within the first 3 months. After 4 years, 90% are expected to have relapsed (Alcohol Relapse and Craving). We use the data for the greatest time interval, which is 4 years, as we believe this will give the most accurate information and use equation (2.15) to estimate $\rho = 0.576$. Best (2010) indicates that an individual experiencing a 4 or 5 year period without consuming alcohol can be considered as recovered. If 90% of individuals relapse after 4 years in treatment then we know that 10% remain in treatment so we can use this in equation (2.15) to calculate the rate of recovery. We obtain a value of 0.0263 which we then divide across a 4-year time period to give a maximum value of $\gamma = 0.00659$. An estimate for β is difficult to determine so we will consider the minimum β value which ensures alcohol problems become endemic in the population, defined as β_{\min} . We find that this minimum value is $\beta_{\min} = 0.156$, calculated using the parameter values stated above and the equation $R_0 = 1$.

Deacon et al. (2007) give the 2005 percentages for adults that binge drink as 19.3% for males and 8.1% for females. Assuming an even sex-ratio, this averages to 13.7% of the adult population so we take an initial value of $a(0) = 0.137$. As we assume that 6% of binge drinkers are in treatment we take $r(0) = 0.00874$.

According to Smith and Foxcroft (2009), there has been an increase in the number of people drinking over the guideline weekly amounts from 1988-2006. We find that the value $\beta_{\min} = 0.156$ results in a decrease in the binge drinking population from our $a(0)$ value so this is not an appropriate lower bound. Instead the lowest value we consider is $\beta = 0.2$ as this results in a continuation of the trend. When a susceptible individual meets a binge drinker, the likelihood that they also become

(a) $\beta = 0.2$, $R_0 = 1.285$, $(\bar{s}, \bar{a}, \bar{r}) = (0.778, 0.205, 0.0174)$ (b) $\beta = 0.4$, $R_0 = 2.571$, $(\bar{s}, \bar{a}, \bar{r}) = (0.389, 0.562, 0.0480)$ (c) $\beta = 0.6$, $R_0 = 3.856$, $(\bar{s}, \bar{a}, \bar{r}) = (0.259, 0.682, 0.0582)$ (d) $\beta = 0.8$, $R_0 = 5.141$, $(\bar{s}, \bar{a}, \bar{r}) = (0.195, 0.742, 0.0633)$ 

—	— · —	- - -
$s(t)$	$a(t)$	$r(t)$

Figure 2.3: Simulations showing how the value of the parameter β affects the endemic equilibrium solution. The other parameters have values $\mu = 0.143$, $\varphi = 0.0619$, $\rho = 0.576$ and $\gamma = 0.00659$.

a binge drinker is proportional to the value of β . As β increases a susceptible individual is more likely to become a binge drinker, so as we increase the value of β we expect an increase in the proportion of binge drinkers in the population.

We now take starting values $(a(0), r(0)) = (0.137, 0.00874)$ and parameter values $\mu = 0.143$, $\varphi = 0.0619$, $\rho = 0.576$, $\gamma = 0.00659$ and let β take the values 0.2, 0.4, 0.6 and 0.8. Figure 2.3 shows how the fractions in each of the classes change over time for the different β values. The graphs plateau at the equilibrium solution values. It can be seen from figures 2.3a and 2.3b that the greatest increase in the proportion of binge drinkers in the population occurs when β changes from a value of 0.2 to 0.4. This 0.2 increase in β results in a change from 20% of the population binge drinking to 56%. Subsequent increases in β do not have such a great effect on the proportion of binge drinkers in the population. This highlights that, for large β values, any inaccuracy in the estimate for β will not greatly affect the results. If β is small however, then any inaccuracies could greatly alter the predicted outcome. Figure 2.4 shows this relationship.

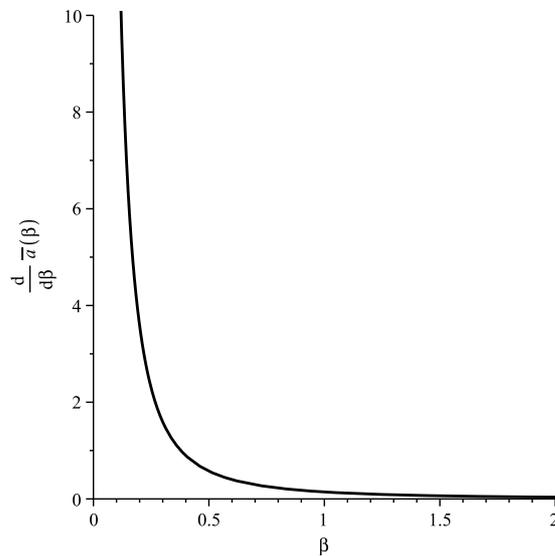


Figure 2.4: Graph showing the rate of change of \bar{a} with respect to β , where we consider \bar{a} to be a function of β only. As β increases, the rate of change in \bar{a} tends towards 0.

Figure 2.5 shows the phase portrait in the a, r -plane of the endemic equilibrium solution for the model where $\gamma > 0$ and for the case where $\gamma = 0$. The parameter

values have been taken as above, along with $\beta = 0.4$. We know that $\mu + \rho + \varphi = 0.7809$ is greater than the value $\beta = 0.4$, so from our analysis in section 2.2.4 we expect taking $\gamma = 0$ to increase both the equilibrium value for $a(t)$ and $r(t)$. Figure 2.5 shows that this is indeed the case.

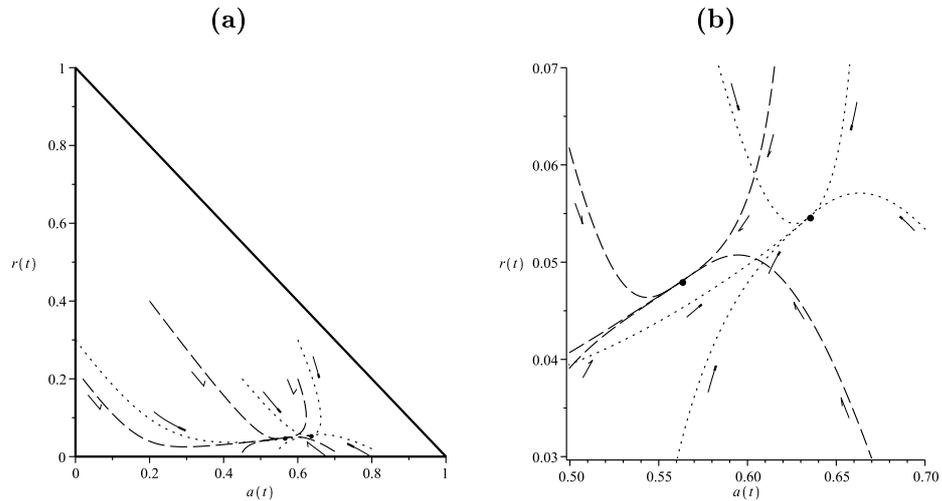


Figure 2.5: Phase portraits showing the endemic equilibrium point for $\gamma = 0.00659$ and for $\gamma = 0$, represented by the dashed and dotted lines, respectively. The other parameters take the values $\mu = 0.143$, $\beta = 0.4$, $\varphi = 0.0619$ and $\rho = 0.576$. The triangle with vertices $(0,0)$, $(1,0)$ and $(0,1)$ in figure (a) is the boundary of the positive invariant region $D = \{(a, r) \in \mathbb{R}^2 : a \geq 0, r \geq 0, a + r \leq 1\}$, where all solutions lie. A proof of the positive invariance of D is included in appendix B.3. Figure (b) shows the behaviour of the system close to the equilibrium solutions.

2.3 Discussion

We have constructed a model for alcohol problems in a population which allows for individuals to totally recover and return to the susceptible population. The threshold $R_0 = 1$ was found, where $R_0 < 1$ indicates that alcohol problems will die out and $R_0 > 1$ determines that alcohol problems become endemic in the population. We found that the R_0 value was most sensitive to changes in the parameter β , which affects the rate at which susceptible individuals develop an alcohol problem. Decreasing β results in a decrease in the value R_0 . This indicates that efforts to

reduce alcohol problems in the population should focus on preventing susceptible individuals from developing an alcohol problem.

We compared this model with the special case $\gamma = 0$, presented in Mulone and Straughan (2011), which prevents individuals from returning to the susceptible class. We found that taking $\gamma = 0$ increased the value of the basic reproduction number and led to an increase in the proportion of recovered individuals and a decrease in the proportion of susceptible individuals. Whether the proportion of individuals with alcohol problems increases or decreases is not determined by including total recovery, but the size of the change is affected by the value of γ . Thus our analysis reveals that the effect of γ on \bar{a} is not straightforward. If the situation were such that β were large then total recovery would have an adverse effect on reducing alcohol misuse in the population.

Estimates were made for the parameters using data for binge drinking in England. We were particularly interested in the effect of social influence on binge drinking and so we considered β values in the interval $[0.2, 0.8]$. Simulations using these values revealed that any inaccuracies in the β value could have a great effect on the proportion of binge drinkers in the population if β was small. For larger β values, any inaccuracies did not have such a great effect.

We have assumed that alcohol abuse is the result of social influence where all individuals are equally susceptible to developing a problem. This does not cover the full range of factors which may affect an individual's propensity to developing a drinking problem. Experimental evidence from both adoption and twin studies indicates that there may be a genetic contribution to the development of alcohol problems, resulting in some individuals being more prone to developing a problem (McGue, 1999; Hicks et al., 2004). However, whilst genetics factors may contribute to an individual's susceptibility to develop a drinking problem, social factors such as peer group influence had an independent effect on behaviour transmission (Hicks et al., 2004). Genetic factors are found to influence alcohol dependence, however binge drinking behaviour does not have a physiological addiction associated with the behaviour so in this specific case the genetic contribution may be of less importance when compared with peer group effects. Thus, although our model is not a complete

picture, it provides a basis for further investigation into the spread of drinking behaviour.

One model assumption is that fully recovered individuals who have returned to the susceptible class have the same probability of developing an alcohol problem for the second time as someone who has had no prior problem with alcohol. A development for the future would be to include a fourth class of fully recovered individuals, as in the four equation smoking model by Sharomi and Gumel (2008), rather than assuming that they return to the susceptible class. This would distinguish fully recovered individuals from susceptible individuals without prior alcohol problems. Alternatively the champion effect discussed by Best (2010) may be better represented by assuming that those in treatment recover because of interactions with a recovery champion.

Recovery can be viewed as contagious, as treatment success is improved by the introduction of an abstinent individual in the social networks of those in treatment (Best, 2010). Community recovery champions are inspirational figures who have experienced addiction and successfully completed treatment (Best, 2010). Their positive effect on those in treatment may be described by model-based social learning biases: similarity bias and success bias. An individual in treatment may experience a similarity bias as the recovery champion has experience of addiction and the difficulties of the treatment process. As the recovery champion has succeeded in completing treatment, a state which the recovering individual wishes to attain, a success bias may also be in operation.

The SARS model may be adapted to incorporate both a totally recovered class and recovery champions through the introduction of a totally recovered class, R_T , and a social influence term taking individuals from R to R_T . This may be modelled by the equations

$$\begin{aligned}\dot{S} &= \mu N - \frac{\beta AS}{N} - \mu S, \\ \dot{A} &= \frac{\beta AS}{N} + \rho R - (\varphi + \mu)A, \\ \dot{R} &= \varphi A - (\rho + \mu)R - \frac{\gamma RR_T}{N}, \\ \dot{R}_T &= \frac{\gamma RR_T}{N} - \mu R_T,\end{aligned}$$

with the dynamics shown in figure 2.6. The term $\gamma RR_T/N$ represents social influence, where totally recovery is dependent on those in treatment interacting with fully recovered individuals: the recovery champions. In this instance the recovery champion effect may be considered as a model-based bias, combining both similarity and success biases.

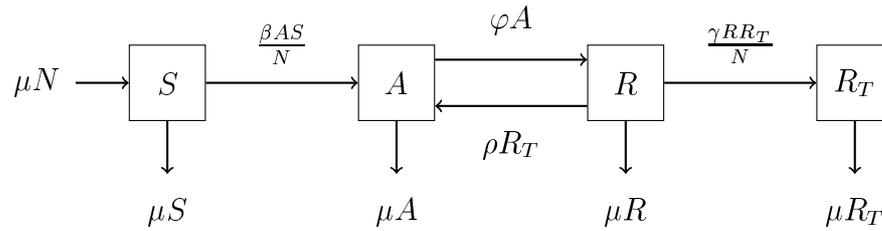


Figure 2.6: An alcohol model incorporating a separate totally recovered class, R_T , whose individuals act as recovery champions to those in the treatment class R .

The mathematics presented in this chapter offers a valuable insight into understanding patterns in drinking behaviour which are affected by social influence. Future work will continue to explore mathematical applications to the study of human behaviours.

Chapter 3

An SIS model for cultural trait transmission with conformity bias

3.1 Introduction

Epidemiological models for the spread of infectious diseases, known as SIR models, have been widely researched since the work of Kermack and McKendrick (1927). The name derives from the assumed model structure, classifying individuals as either susceptible, infected or recovered. Many variations of SIR models exist (Murray, 2003; Hethcote, 2000; McCallum et al., 2001; Keeling and Rohani, 2008), including SIS models where individuals can be either susceptible or infected. An SIS model for infectious disease spread considers how the subpopulations of susceptible and infected individuals change in time, represented mathematically by two ordinary differential equations (ODEs). It is assumed that all individuals entering the population are susceptible. They may become infected through contact with infected individuals at a rate proportional to the frequency of infected individuals in the population. Infected individuals recover to the susceptible state at a constant rate.

The assumption that infection is spread through contact has led to the application of SIS and similar models to be applied to a range of human health-related behaviours where social interaction affects the spread of the behaviour. Examples include models of addictive behaviours, such as smoking (Sharomi and Gumel, 2008), drug use (Song et al., 2006; White and Comiskey, 2007; Mulone and Straughan,

2009), drinking (Sanchez et al., 2007; Mubayi et al., 2010; Mulone and Straughan, 2011; Walters et al., 2012), the spread of happiness (Hill et al., 2010a) and the development of eating disorders (Gonzalez et al., 2003) or obesity (Hill et al., 2010b). Such models assume that the rate at which susceptible individuals adopt a behaviour is proportional to the prevalence of the behaviour in the population. However, we see from cultural evolutionary theory that this assumption may be oversimplifying the mechanisms involved in behaviour transmission, and that biases in transmission can result in qualitatively distinct model predictions.

Cultural evolutionary theory considers the spread and persistence of socially transmitted traits, including ideas, beliefs, behaviours and material culture (Cavalli-Sforza and Feldman, 1981; Boyd and Richerson, 1985; Mesoudi, 2011). A cultural trait is typically acquired by some form of social learning. If social learning is unbiased (random copying) then the probability that an individual adopts a cultural trait is equal to the trait's frequency in the population. The assumption that transmission is linearly frequency-dependent, i.e. unbiased, is commonly applied in the SIS model literature; cultural trait transmission, however, may be subject to a variety of content or contextual biases (Henrich and McElreath, 2003) which affect the transmission rate. Content-dependent biases arise from some intrinsic property of the cultural trait. Such biases make it, for example, easier to remember or intrinsically more attractive than other competing traits (Richerson and Boyd, 2005; Mesoudi, 2011). Context-dependent biases can be split into model-based and frequency-dependent biases (Henrich and McElreath, 2003; Richerson and Boyd, 2005; Mesoudi, 2011). The former is where the choice of a trait is affected by observable attributes of the cultural parent, for example copying individuals that are perceived to be successful. The latter is typically where the frequency of the trait in the population affects its uptake in a nonlinear fashion, such as a disproportionate tendency to adopt the most common trait. This is termed as a conformist bias whereas a disproportionate tendency to follow the minority is often known as anticonformist bias (Efferson et al., 2008; Eriksson and Coultas, 2009; Kendal et al., 2009; Morgan et al., 2011).

A variety of empirical studies examining the extent of conformist bias have been

conducted, with one of the earliest finding that participants would conform to the majority viewpoint expressed by confederates (Asch, 1956). This has since been criticised as the results do not demonstrate a disproportionate inclination to follow the majority and hence may reflect random copying (Efferson et al., 2008). By defining conformity as an exaggerated tendency to follow the majority, modelled by a sigmoidal curve, Efferson et al. (2008) conducted an experiment where players repeatedly chose between two technologies with different expected, but randomly distributed, payoffs. A subset of participants that indicated a conformist bias in their answers to questionnaires copied the technology choice of asocial learners with an S-shaped probability distribution, indicating conformist behaviour.

Later work by Eriksson and Coultas (2009) offers an alternative theoretical model of conformity. The authors argue that the S-shaped probability curve originally used by Boyd and Richerson (1985) is unrealistic. Particularly, the endpoints of the curve mean that a naïve individual cannot acquire a trait which is not being displayed in the population, nor can they reject a trait which is universally expressed by the population. Furthermore, the conformity threshold frequency, which we define to be the intermediate point where the nonlinear frequency dependence curve meets the linear curve, need not occur when exactly half of the population display the trait. Allowing the endpoints and the conformity threshold frequency to vary produces a model which can account for content-dependent biases, such that the attraction of the trait itself may interact with a nonlinear frequency-dependent probability of adoption. In applying their model to an experiment testing frequency-dependent effects on opinion formation they found evidence for anticonformist bias, suggesting that any expression of conformity bias may be conditional (also see Morgan et al. (2011)). Results from a series of experiments conducted by Morgan et al. (2011) suggest that subjects used conformist biased social learning. This, however, required a large number of demonstrators and for the individuals to have low confidence in their ability to complete the task independently. In contrast, a high magnitude of asocial influence resulted in a conformity bias where the conformity threshold frequency was greater than a half.

In light of these findings, we present a mathematical model to examine the

dynamics of a cultural trait under conformist biased transmission. Results are compared against the case of unbiased social transmission, before considering the effect of a variable conformity threshold parameter. Our analysis focuses on the effect of the strength of conformity on the existence and stability of equilibria. The formulation is equivalent to that of an SIS model, including a frequency-dependent rate of trait adoption and a constant rate of abandonment. The latter may reflect individual forgetting or the result of population-wide influences, such as mass media, or economic and environmental change. The formulation also approximates cases of frequency-dependent abandonment if this rate is very small. By way of an example, we discuss how the model may apply to the case of binge drinking within a population of young adults (see section 3.4).

3.2 Models for unbiased and conformist cultural trait transmission

We begin by assuming the existence of a cultural trait A within a population of N individuals, where trait transmission is frequency-dependent and abandonment of the trait is (approximately) frequency-independent. Individuals within the population can be categorised as type S, who do not display trait A, or type A, who do. The time-dependent variables $S(t)$ and $A(t)$ represent the number of type S and type A individuals respectively. We assume that all individuals enter the population as type S at a rate μ ; however, they may leave as either type at the same rate. Type S individuals can only acquire trait A through interactions with type A individuals, and we assume that the transmission rate is affected by the frequency of type A individuals in the population. We consider the transmission rate to be $\beta c(A/N)$ where β is the rate at which contact sufficient for transmission occurs. In the unbiased social learning model the function $c(A/N)$ represents the probability that contact is made with a type A individual. However, in the case of biased social learning, the function also includes a weighting which represents the conformist influence. Type A individuals revert to type S at a constant rate γ , although this term also approximates the effect of a social influence when γ is very small. For a

mathematical justification see appendix C.2.

From this we formulate the following equations

$$\begin{aligned}\dot{S}(t) &= \mu N - \beta S c(A/N) + \gamma A - \mu S, \\ \dot{A}(t) &= \beta S c(A/N) - (\gamma + \mu) A,\end{aligned}\tag{3.1}$$

where the total population $N = S + A$ is constant. Figure 3.1 represents these dynamics with arrows indicating the direction of flow through the system. The constant total population results from the entering and leaving rates, μ , being the same. This simplifying assumption is made so that the system may be reduced to one equation, which is non-dimensionalised by introducing the variables $s = S/N$ and $a = A/N$ to give

$$\dot{a}(t) = \beta(1 - a)c(a) - \rho a,\tag{3.2}$$

where $\rho = \gamma + \mu$ has been introduced to simplify the mathematical analysis.

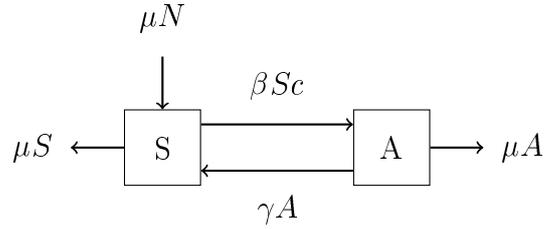


Figure 3.1: Pictorial representation of the SAS model for cultural trait transmission, relating to equations (3.1). The nodes S and A represent the subpopulations of type S and type A individuals respectively. The labelled arrows indicate the rate and direction of movement through the system.

We must now consider the function $c(a)$. This function determines the frequency-dependent relationship between the probability that type S individuals convert to type A and the frequency of type A individuals in the population. First we introduce a linear frequency-dependent function

$$\begin{aligned}c_L &: [0, 1] \rightarrow [0, 1], \\ c_L(a) &= a,\end{aligned}$$

which gives us a model for unbiased trait transmission, resulting in the standard SIS

model form. We then introduce a nonlinear frequency-dependent function

$$\begin{aligned} c_1 &: [0, 1] \rightarrow [0, 1], \\ c_1(a) &= a[1 + D(2a - 1)(1 - a)], \end{aligned} \tag{3.3}$$

which is the conformity function first proposed by Boyd and Richerson (1985). The conformity coefficient $D \in (0, 1]$ controls the strength of the bias. The value $D = 0$ is not considered as this would result in the linear function c_L . We see from figure 3.2 that c_1 is an appropriate function to represent a conformity bias as its sigmoidal shape ensures that all individuals have a disproportionate tendency to follow the majority. When the frequency of type A individuals in the population is less than a half, so $a < 1/2$, the probability of type S adopting trait A is $P(\text{adopting A}) < a$. When the frequency of type A individuals is greater than a half then $a > 1/2$ and $P(\text{adopting A}) > a$. We refer to $a_e = 1/2$ as the conformity threshold frequency as this is where $P(\text{adopting A}) = a$, i.e. where the linear and nonlinear frequency-dependent curves meet.

The criteria for an appropriate conformity function are that exactly one saddle point and no local extrema must exist in the region $(0, 1)$ and, initially, symmetry about the point $(1/2, 1/2)$. More complex real functions, such as higher order polynomials or trigonometric functions, can also satisfy these criteria; however, they may then be locally approximated to a cubic polynomial function. As a result, the behaviour of such systems pertaining to existence and stability of equilibria will be qualitatively similar to the results presented here. Precise relationships between the parameters and the conformity coefficient will, however, vary depending on the behaviour of the chosen conformity function with respect to the coefficient D .

We begin by analysing the linear frequency-dependent SIS model which is constructed from equation (3.2) using the linear function c_L to give

$$\dot{a}(t) = \beta a(1 - a) - \rho a. \tag{3.4}$$

As equation (3.4) is not analytically solvable we look for equilibrium solutions, which are values of a which satisfy $\dot{a}(t) = 0$, and analyse their stability. Once a stable equilibrium is reached, the proportion of type A individuals in the population remains constant in time and hence we can determine the prevalence of trait A within the

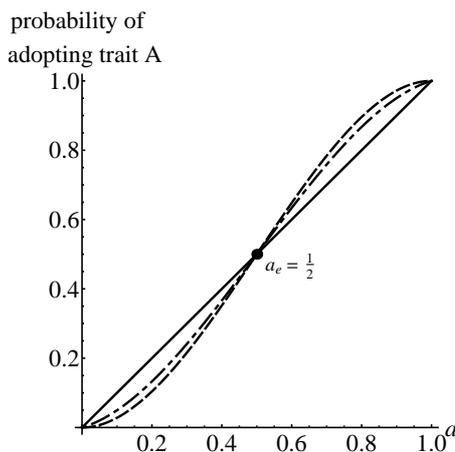


Figure 3.2: Plot of the functions c_L (bold) and c_1 , given by equations (3.2) and (3.3) respectively, with conformity strength values $D = 0.7$ (dot-dashed) and $D = 1$ (dashed). As the strength of the conformist tendency (D) increases, so does the concavity of the conformity curve c_1 . Consequently, as D increases, the probability of adopting trait A decreases for $a < 1/2$ and increases for $a > 1/2$.

population. To ensure that the model provides realistic predictions we seek feasible solutions characterised as those which are unique and lie in the interval $[0, 1]$. As we are interested in solutions for a we rewrite equation (3.4) as a function of this variable, giving

$$f_L(a) = a[\beta(1 - a) - \rho]. \quad (3.5)$$

Solving $f_L = 0$ results in two equilibrium solutions: $\bar{a}_0^L = 0$, which is feasible for all parameter values, and $\bar{a}_1^L = (\beta - \rho)/\beta$, which is feasible for $\rho < \beta$.

We now look at equation (3.2) with conformity function c_1 which gives

$$\dot{a}(t) = \beta a(1 - a)[1 + D(2a - 1)(1 - a)] - \rho a. \quad (3.6)$$

This can be written as $\dot{a} = a f_1(a)$ where we see that $\bar{a}_0^1 = 0$ is an equilibrium solution which always exists, independent of the values of β , ρ and D . The remaining equilibrium solutions are the roots of

$$f_1(a) = \beta(1 - a)[1 + D(2a - 1)(1 - a)] - \rho \quad (3.7)$$

which can be found explicitly, but their complexity makes further analysis difficult. By using properties of the function f_1 it is possible to determine the number and

nature of equilibrium points under certain conditions. The cubic polynomial f_1 has leading coefficient $2\beta D > 0$, so it always has one real root, and $f_1(a) \rightarrow \pm\infty$ as $a \rightarrow \pm\infty$. The roots of $f_1'(a) = \beta(6Da^2 - 10Da + 4D - 1)$ give the local maximum and local minimum of f_1 which are

$$a_-^1 = \frac{5}{6} - \frac{1}{6}\sqrt{\frac{D+6}{D}} \quad \text{and} \quad a_+^1 = \frac{5}{6} + \frac{1}{6}\sqrt{\frac{D+6}{D}}$$

respectively. The vertical intercept occurs at $f_1(0) = \beta(1 - D) - \rho$.

The parameter ρ only occurs in the constant term of equation (3.7) so serves to shift the graph of f_1 down the vertical axis as it increases; thus we know that the limiting case of $\rho = 0$ maximises the function. This observation leads us to introduce

$$g_1(a) = \beta(1 - a)[1 + D(2a - 1)(1 - a)] \tag{3.8}$$

which is equal to the function f_1 in the limiting case of $\rho = 0$ and hence has the same turning points as f_1 . The direct calculation of the turning points reveals $g_1(a_-^1) > 0$ and $g_1(a_+^1) < 0$, where $a_-^1 < 1 < a_+^1$, so g_1 has three real roots which are $a = 1$, $a \in (-\infty, a_-^1)$ and $a \in [a_+^1, \infty)$. Consequently $f_1(a_+^1) < 0$ and f_1 has three real roots for sufficiently small ρ ; however, the root lying in $[a_+^1, \infty)$ is never feasible as $a_+^1 > 1$ and is therefore disregarded. For the remaining two roots to exist and be unique we require $\rho < g_1(a_-^1)$, shown by the shaded region in figure 3.3a, where

$$g_1(a_-^1) = \frac{\beta}{54} \left[9 + D + (6 + D)\sqrt{\frac{6 + D}{D}} \right].$$

This existence condition allows us to determine the form of the actual solutions, which are shown in appendix C.1.

The feasibility of the remaining solutions, defined as $\bar{a}_1^1 \in (-\infty, a_-^1)$ and $\bar{a}_2^1 \in (a_-^1, 1)$, must be determined when they exist. As we already have the equilibrium solution $\bar{a}_0^1 = 0$ we require \bar{a}_1^1 and \bar{a}_2^1 to lie in $(0, 1]$ for the equilibrium points to be unique. By considering the sign of a_-^1 , which determines the location of the local maximum of f_1 , we construct two cases: $D \in (0, 1/4]$ and $D \in (1/4, 1]$, corresponding to $a_-^1 \leq 0$ and $a_-^1 > 0$ respectively. In the first case \bar{a}_2^1 can be feasible, which occurs when the vertical intercept is positive. This provides the condition $\rho < \beta(1 - D)$. For the second case, \bar{a}_2^1 is feasible for $\rho < g_1(a_-^1)$ (i.e. for when it exists), and \bar{a}_1^1 is

feasible for $\beta(1 - D) < \rho < g_1(a_-^1)$ which is where the vertical intercept is negative and the turning point is positive. These cases are shown in figure 3.3.

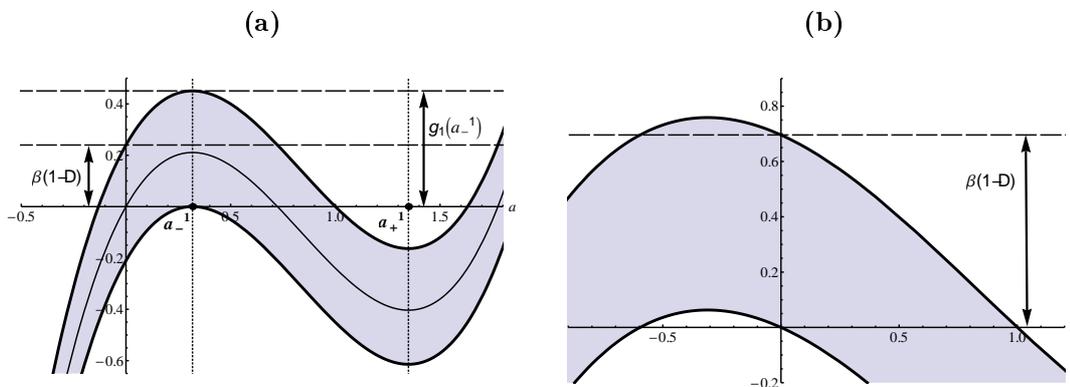


Figure 3.3: (a) The shaded region is the area bounded above and below by the curves $g_1(a)$ (equation (3.7)) and $f_1(a)$ (equation (3.8)) respectively, where $\beta = 0.8$, $D = 0.7$ and $\rho = g_1(a_-^1) = 0.451$. For f_1 in the limiting case of $\rho = 0$ (equivalent to curve g_1) only one root is feasible ($a = 1$, which is independent of β and D). As the value of ρ increases the two leftmost roots tend toward $a = a_-^1 = 0.318$. The central curve, with $\rho = \beta(1 - D) = 0.24$, highlights where two equilibria become feasible. Eventually, when $\rho = g_1(a_-^1)$, both of these equilibria cease to exist.

(b) The shaded region is bounded by the curves $g_1(a)$ and $f_1(a)$ with $\beta = 0.8$, $D = 0.13$ and $\rho = \beta(1 - D) = 0.696$. As the value of ρ increases, the only feasible solution decreases away from $a = 1$ toward $a = 0$, at which point it becomes unfeasible. This situation where only one equilibrium is feasible arises for $D \in (0, 1/4]$, unlike the scenario of (a) where two feasible solutions may exist and $D \in [1/4, 1)$.

3.2.1 Stability Analysis

To determine the local stability of an equilibrium solution we consider the system close to the equilibrium point and linearise around this point. For a function $F(a)$ and equilibrium point \bar{a} we consider $F(\bar{a} + a)$ where a is small. Linearising around the point \bar{a} gives

$$F(a) = aF'(\bar{a}) + \mathcal{O}(a^2)$$

as $F(\bar{a}) = 0$, so close to the equilibrium point we have $F(a) = ka$ for $k \in \mathbb{R}$ constant. In our system, linearising results in an ODE of the form $\dot{a} = ka$ which has solutions $a(t) = Ke^{kt}$ for $K \in \mathbb{R}$ constant. For asymptotic stability we require $k < 0$ as this ensures that the solution decays with time.

For the unbiased social learning model, equation (3.5), linearising gives

$$f_L = (\beta - \rho - 2\beta\bar{a})a$$

so \bar{a}_0^L and \bar{a}_1^L are asymptotically stable for $\rho > \beta$ and $\rho < \beta$ respectively. For the conformist biased model, equation (3.6), the condition for asymptotic stability is $f_1(\bar{a}) + \bar{a}f_1'(\bar{a}) < 0$ where $f_1(\bar{a}) = 0$ for $\bar{a} \neq 0$ and

$$\bar{a}f_1'(\bar{a}) = \beta\bar{a}(6D\bar{a}^2 - 10D\bar{a} + 4D - 1).$$

From this we know that \bar{a}_0^1 is asymptotically stable for $\rho > \beta(1 - D)$. Asymptotic stability of the remaining feasible solutions requires $f_1'(\bar{a}) < 0$ which is true provided $\bar{a} \in (a_-^1, a_+^1)$, so \bar{a}_1^1 is never stable and \bar{a}_2^1 is always asymptotically stable. These results are summarised in table 3.1.

Model comparison

We now identify how a conformity bias affects the persistence of trait A in the population compared with the linear case. Recall that $\rho = \mu + \gamma$ was introduced to simplify the analysis, so any interpretation of ρ requires an understanding of how μ and γ behave. As we are interested in the proportion of type A individuals in the population we consider the flow to and from this subpopulation, shown in figure 3.1.

Flow into A is only affected by the parameter β and flow out of A happens at rate $\mu + \gamma$, so ρ is the rate that individuals leave A. By considering ρ fixed across both the linear and nonlinear frequency-dependent models we can define threshold values of β required for type A individuals to persist in the population. In the linear frequency-dependent model the threshold value is $\beta_L = \rho$. In the nonlinear model the threshold is different as it depends upon the strength of the conformist tendency. The threshold value is $\beta_N^1 = \rho/(1 - D)$ so, for very small D , the linear and nonlinear threshold values are approximately equal. As the strength

		<i>Feasible</i>	<i>Asymptotically Stable</i>	<i>Unstable</i>
Linear	\bar{a}_0^L	Always	$\rho > \beta$	$\rho < \beta$
	\bar{a}_1^L	$\rho < \beta$	$\rho < \beta$	—
$D \in (0, 1/4]$	\bar{a}_0^1	Always	$\rho > \beta(1 - D)$	$\rho < \beta(1 - D)$
	\bar{a}_1^1	Never	—	—
	\bar{a}_2^1	$\rho < \beta(1 - D)$	$\rho < \beta(1 - D)$	—
$D \in (1/4, 1]$	\bar{a}_0^1	Always	$\rho > \beta(1 - D)$	$\rho < \beta(1 - D)$
	\bar{a}_1^1	$\beta(1 - D) < \rho < g_1(a_-^1)$	—	$\beta(1 - D) < \rho < g_1(a_-^1)$
	\bar{a}_2^1	$\rho < g_1(a_-^1)$	$\rho < g_1(a_-^1)$	—

Table 3.1: For the linear frequency-dependent model the stability of the equilibria switches when the rate of transmission (β) is equal to the rate of leaving the type A class (ρ). When the leaving rate is greater, $\rho > \beta$, trait A dies out. When $\rho < \beta$ however, trait A persists. For a conformity strength $D \in (0, 1/4]$ the stability of the zero solution and endemic solution switches when $\rho = \beta(1 - D)$, that is where the rate of leaving A is equal to the transmission rate, subject to a conformity effect. This threshold is greater than the linear case so a larger transmission rate β is required for trait A to become endemic. For an increased conformity strength ($D > 1/4$) a bistable state exists where the equilibrium attained is dependent upon the initial frequency of type A individuals.

of conformity increases so does the threshold value; thus $\beta_N^1 > \beta_L$. This indicates that when there is a conformity bias acting within a population, the contact rate β must be greater than in the linear case for trait A to become endemic within the population. This is demonstrated by simulation results, summarised in table 3.2 (section A), where increasing the value of β results in the endemic equilibrium solution becoming feasible and stable for a linear frequency-dependent relationship, but not with a nonlinear one. Section B of table 3.2 shows that, as the conformity strength increases, a larger value of β is required for the endemic equilibrium solution to become feasible. This indicates that conformity effects suppress the spread of trait A, which is intuitive as initially type A individuals are rare in the population so conformity acts against them, instead favouring type S individuals.

When $D > 1/4$, there exists a second threshold value. For trait A to persist in the population without any dependence on the initial frequency of type A individuals then the threshold value remains as $\beta_N^1 > \beta_L$. This corresponds to when the equilibrium solution \bar{a}_2^1 is feasible and stable whereas \bar{a}_1^1 is not feasible. As D increases so does the threshold value, though it is undefined at $D = 1$. This indicates that when conformity strength is at its maximum, it is not possible to have a contact rate which is sufficiently large to overcome the propensity to conform. Trait A, therefore, cannot become endemic in this scenario. By introducing a second threshold, $\beta_M^1 = \rho/k_1(D)$ where

$$k_1(D) = \frac{1}{54} \left[9 + D + (6 + D) \sqrt{\frac{6 + D}{D}} \right],$$

trait A may become endemic. Using the extreme values of D we can bound $k_1(D)$ from above by $k_1(D) < 45/54 < 1$ and therefore $\beta_M^1 > \beta_L$, so again the threshold value for the conformity model is greater than that of the unbiased social learning model. We also see from figure 3.3a that $\beta(1 - D) < g_1(a_-^1) = \beta k_1(D)$ and therefore $1/k_1(D) < 1/(1 - D)$ so $\beta_M^1 < \beta_N^1$. This lower nonlinear threshold value means that trait A can become endemic in the population even when $D = 1$, dependent upon the initial state. We know from our stability analysis (section 3.2.1) that the system can have two asymptotically stable solutions, \bar{a}_0^1 and \bar{a}_2^1 , so the solution that is reached depends on the initial frequency of type A individuals in the population.

By defining $t_0 = 0$ then for $a(t_0) < \bar{a}_1^1$ trait A cannot persist in the population and for $a(t_0) > \bar{a}_1^1$ it becomes endemic. This shows that beginning with very few type A individuals means it is likely that trait A will die out in the population. If at t_0 there was, for example, some major environmental change leading to a sufficiently large number of individuals becoming type A, then trait A would persist in the population. Table 3.2, section C, gives an example of where the two equilibrium solutions are feasible and stable for sufficiently large conformity strength, compared to the model with a weaker conformity strength.

3.3 Model for conformist cultural trait transmission with varying conformity threshold

We now generalise our model further by allowing the threshold value a_e to vary away from $1/2$, which could indicate a content bias acting in the population. We use the conformity function

$$c_2(a) = a[1 + D(2a - \eta)(1 - a)] \tag{3.9}$$

which produces an asymmetric sigmoidal curve. The threshold value is $a_e = \eta/2$ where $\eta \in (0, 2)$, but restrictions must be placed on the conformity coefficient D to ensure that c_2 is monotone increasing on $[0, 1]$. This is achieved by considering the local minimum and local maximum of c_2 ,

$$\tilde{a}_- = \frac{2 + \eta}{6} - \frac{\sqrt{D^2\eta^2 - 2D^2\eta + 4D^2 + 6D}}{6D}$$

and

$$\tilde{a}_+ = \frac{2 + \eta}{6} + \frac{\sqrt{D^2\eta^2 - 2D^2\eta + 4D^2 + 6D}}{6D}$$

respectively, where we require $\tilde{a}_- \leq 0$ and $\tilde{a}_+ \geq 1$. This gives conditions $D \leq 1/\eta$ and $D \leq 1/(2 - \eta)$. As $\text{Max}\{D\} = 1$, the first condition does not always hold for $\eta \in (1, 2)$ and the second for $\eta \in (0, 1)$. For example, when $\eta = 1/2$ then $D \leq 2/3$ which is a stricter condition on D than we desire. To eliminate this problem we restrict D so that $D \in (0, 1/(2 - \eta))$ for $\eta \in (0, 1]$ and $D \in (0, 1/\eta)$ for $\eta \in (1, 2)$.

	A			B			C		D		
Function	c_L	c_L	c_1	c_L	c_1	c_1	c_1	c_1	c_2	c_2	c_2
β	0.2	0.27	0.27	0.3	0.3	0.3	0.45	0.45	0.45	0.45	0.45
D	—	—	0.1	—	0.1	0.7	0.1	0.7	0.0.7	0.7	0.7
η	—	—	—	—	—	—	—	—	0.6	1	0.2
Stable Solution	0	0.741	0	0.167	0.103	0	0.441	0 or 0.380	0.515	0 or 0.380	0

Table 3.2: Table showing simulation results for different parameter values, with $\rho = 0.25$ fixed. The stable solution is the frequency of type A individuals in the population once the system has reached equilibrium, where all values are to three significant figures.

A: Comparison between the linear frequency-dependent function c_L and the conformity function c_1 highlighting the effect of the transmission rate β on the stability of an endemic equilibrium ($a > 0$).

B: For a fixed transmission rate β , the linear frequency-dependent model results in a higher frequency of type A individuals in the population than the conformity model. Provided that the conformity strength D is large enough, an endemic equilibrium will not be reached and type A individuals will always die out.

C: For certain parameter values, an increase in the conformity strength will result in a bistable system. In the example given, a threshold exists at $a(0) = 0.258$. For an initial frequency of type A individuals greater than 0.258, trait A will become endemic within the population with approximately 38% displaying the trait at equilibrium. For an initial frequency of type A individuals less than 0.258 the trait will eventually die out.

D: The effect of a content bias, controlled by η in conformity function c_2 , is investigated. As the value of η increases, the persistence of type A individuals first becomes dependent on their initial frequency before becoming impossible.

Figure 3.4a shows the function for $\eta \in (0, 1]$ where the intersection point a_e lies in the interval $(0, 1/2]$. This represents a situation where less than half of the population displaying trait A is sufficient for a naïve individual to be more likely to take up trait A than in the linear case. Figure 3.4b shows the function for $\eta \in (1, 2)$ and $a_e \in (1/2, 1)$. Here, more than half the population must display trait A in order for the probability of behaviour uptake to be greater than in the linear case. An increase in the value of η represents an increased aversion to adopting trait A. As with the previous conformity function c_1 , an increase in the conformity strength D increases the concavity of conformity function c_2 .

The nonlinear frequency-dependent SIS model with variable threshold point η is

$$\dot{a}(t) = \beta a(1 - a) [1 + D(2a - \eta)(1 - a)] - \rho a, \quad (3.10)$$

formed from equation (3.2) and the conformity function c_2 . We analyse this model by proceeding as in section 3.2, beginning by defining $f_2(a)$, where $\dot{a} = a f_2(a)$ so that the equilibrium solutions are $\bar{a}_0^2 = 0$ and the roots of

$$f_2(a) = \beta(1 - a) [1 + D(2a - \eta)(1 - a)] - \rho.$$

The function f_2 has distinct turning points

$$a_-^2 = \frac{4 + \eta}{6} - \frac{1}{6} \sqrt{(2 - \eta)^2 + \frac{6}{D}} \quad \text{and} \quad a_+^2 = \frac{4 + \eta}{6} + \frac{1}{6} \sqrt{(2 - \eta)^2 + \frac{6}{D}}$$

and vertical intercept $f_2(0) = \beta(1 - \eta D) - \rho$. Taking the limiting case of $\rho = 0$ we introduce the function

$$g_2(a) = \beta(1 - a) [1 + D(2a - \eta)(1 - a)]$$

and direct calculation reveals that $g_2(a_-^2) > 0$ and $g_2(a_+^2) < 0$ where $a_-^2 < 1$ and $a_+^2 > 1$. Hence g_2 has roots $a \in (-\infty, a_-^2)$, $a = 1$ and $a \in (a_+^2, \infty)$ so f_2 has three roots for sufficiently small ρ . One of the roots is never feasible so we ignore it. For the three solutions to exist the condition $\rho < g_2(a_-^2)$ must hold where

$$g_2(a_-^2) = \frac{\beta}{54} \left[9(2 - \eta) + D(2 - \eta)^3 + (6 + D(2 - \eta)^2) \sqrt{\frac{6 + D(2 - \eta)^2}{D}} \right].$$

As before, we can now determine the form of the exact solutions, shown in appendix C.1.

To determine the feasibility of the two roots $\bar{a}_1^2 \in (-\infty, a_-^2)$ and $\bar{a}_2^2 \in (a_-^2, 1)$, the sign of a_-^2 must be considered, where $a_-^2 \leq 0$ gives the case $D \leq 1/(2 + 2\eta)$. Only \bar{a}_2^2 is ever feasible given $\rho < g_2(0)$, where $g_2(0) = \beta(1 - \eta D)$ is the vertical intercept. When $D > 1/(2 + 2\eta)$ both solutions can be feasible if $\rho < g_2(a_-^2)$ for \bar{a}_2^2 and $g_2(0) < \rho < g_2(a_-^2)$ for \bar{a}_1^2 .

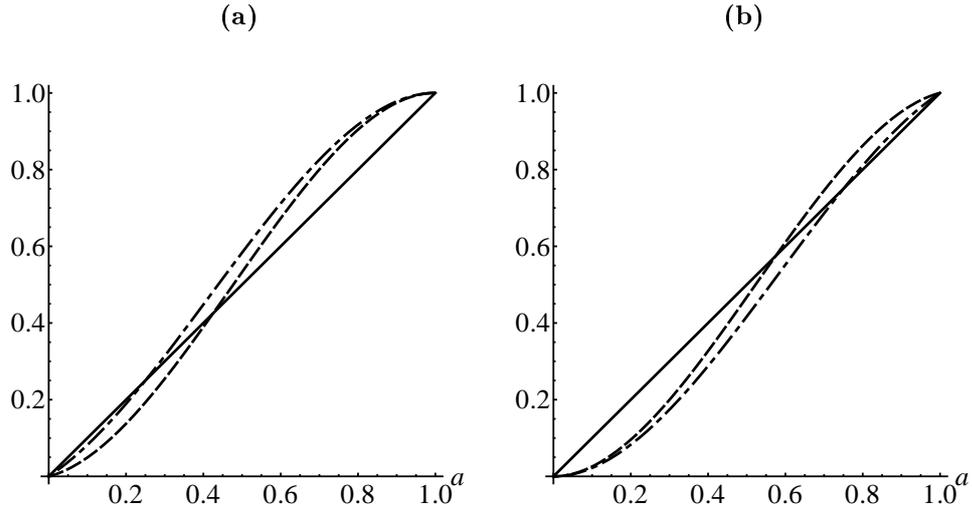


Figure 3.4: The figures show the functions c_L (equation (3.2), bold) and c_2 (equation (3.9)) with $D = 1$ and (a) $\eta = 0.5$ (dot-dashed), $\eta = 0.85$ (dashed) and (b) $\eta = 1.15$ (dashed), $\eta = 1.5$ (dot-dashed). When more than $\eta/2$ of the population display trait A, the probability of uptake is greater than that of the linear case. As the value of η increases, the probability of adopting trait A reduces, representing a content bias which dissuades individuals from adopting the trait. The probability of adopting trait A is (a) greater than for the function c_1 (equation (3.3), figure 3.2) when $\eta < 1$ and (b) less than c_1 when $\eta > 1$.

Stability Analysis

Following the method of linearisation from section 3.2.1 we find that the condition for asymptotic stability of an equilibrium solution of equation (3.10) is $f_2(\bar{a}) + \bar{a}f_2'(\bar{a}) < 0$. The equilibrium solution \bar{a}_0^2 is asymptotically stable for $\rho < g_2(0)$ and the stability of the remaining two solutions requires $f_2'(\bar{a}) < 0$, which corresponds to solutions lying in the interval (a_-^2, a_+^2) . Hence we find that a feasible \bar{a}_2^2 is always

asymptotically stable and a feasible \bar{a}_1^2 is never stable. The feasibility and stability conditions for the equilibrium solutions are summarised in table 3.3.

Model comparison

As in section 3.2.1, we can define threshold values of β for which the stable equilibrium changes from being trait-A-free to the trait persisting in the population. We first consider $D \leq 1/(2+2\eta)$ and define the threshold value to be $\beta_N^2 = \rho/(1-\eta D)$ so $\beta_N^2 > \beta_L$. For $\eta < 1$ we have $\beta_N^2 < \beta_N^1$, which is an intuitive result when comparing the curves c_1 and c_2 . Defining the distance between these two curves as

$$d(a) = c_1 - c_2 = D(\eta - 1)a(1 - a)$$

then $d < 0$ for $\eta < 1$ which signifies that $P(\text{adopting A} | c_2) > P(\text{adopting A} | c_1)$. Hence, for some fixed a value, the probability of adopting trait A is greater when we take conformity function c_2 . The threshold value β_N^2 is lower than β_N^1 as, for each individual contact, the probability of transmission is greater than with c_1 and hence fewer contacts are required for trait A to become endemic. For $\eta > 1$ the converse is true, whereby $d > 0$ and hence $P(\text{adopting A} | c_2) < P(\text{adopting A} | c_1)$. The effect of η is shown in table 3.2, section D, where the other parameter values are fixed. When $\eta = 0.7$ the endemic solution is feasible so type A individuals will persist in the population. Comparing this with the previous model (which is equivalent to $\eta = 1$) we see that the persistence of type A individuals is not certain but depends on the initial state. A further increase to $\eta = 1.2$ results in trait A dying out within the population, owing to the change in the conformity bias effect.

When $D > 1/(2 + 2\eta)$ the threshold β_N^2 is defined for $\eta D \neq 1$. As with the previous conformity model, a second threshold exists where trait A persisting in the population is dependent upon initial state. This threshold is $\beta_M^2 = \rho/k_2(D)$ where

$$k_2(D) = \frac{1}{54} \left[9(2 - \eta) + D(2 - \eta)^3 + (6 + D(2 - \eta)^2) \sqrt{\frac{6 + D(2 - \eta)^2}{D}} \right]$$

and $\beta_M^2 < \beta_N^2$. Again this threshold value increases with η so $\beta_M^2 < \beta_M^1$ when $\eta < 1$ and $\beta_M^2 > \beta_M^1$ for $\eta > 1$.

	<i>Feasible</i>	<i>Asymptotically Stable</i>	<i>Unstable</i>	
$D \in (0, 1/4]$	\bar{a}_0^2	Always	$\rho > \beta(1 - \eta D)$	$\rho < \beta(1 - \eta D)$
	\bar{a}_1^2	Never	—	—
	\bar{a}_2^2	$\rho < \beta(1 - \eta D)$	$\rho < \beta(1 - \eta D)$	—
$D \in (1/4, 1]$	\bar{a}_0^2	Always	$\rho > \beta(1 - \eta D)$	$\rho < \beta(1 - \eta D)$
	\bar{a}_1^2	$\beta(1 - \eta D) < \rho < g_2(a_-^2)$	—	$\beta(1 - \eta D) < \rho < g_2(a_-^2)$
	\bar{a}_2^2	$\rho < g_2(a_-^2)$	$\rho < g_2(a_-^2)$	—

Table 3.3: For $D \in (0, 1/4]$ the stability of the zero and endemic solutions switches at $\rho = \beta(1 - \eta D)$. This is where the leaving rate is equal to the transmission rate, modified by a combined conformity and content bias term. The value of η , representing a content bias, affects the magnitude of variation between this threshold and the threshold associated with conformity function c_1 (see table 3.1 for comparison). As with the previous conformity model (section 3.2), increasing the conformity strength ($D > 1/4$) allows for a bistable solution where the initial frequency of type A individuals affects their long-term survival.

3.4 Discussion

Our analysis reveals that varying the conformity threshold frequency a_e affects the β value required for cultural trait A to become endemic in the population, where β represents the average rate of contacts sufficient for transmission of cultural trait A. Lowering a_e results in an increased probability of adopting trait A for some fixed a value, and hence lowers the threshold value of β which is required for the trait to persist. In contrast, β must be large for this to occur when a_e is high.

Morgan et al. (2011) found that an increased conformity threshold frequency was consistent with strong confidence in information acquired asocially. Here we have a similar asymmetric conformity function, but without requiring asocial learning. Instead, the value of the conformity threshold frequency coefficient η may capture the interaction of a content bias with conformity bias. For instance, the conformity threshold frequency for an attractive cultural trait may be smaller than that of a trait not in possession of the same intrinsic appeal. Our analysis shows that the value of η can affect the conditions for trait A extinction.

The effect of a content bias on social transmission may, however, be more complex than simply altering the conformity threshold. A content bias may also affect the value of the adoption and abandonment rates, β and γ . For example, a trait that is highly attractive or salient would have a high rate β at which contact sufficient for transmission occurs. From the results of our conformity model, we can see that content bias affecting β will alter the unfeasibility of an endemic equilibrium for a given conformity bias strength D .

Evidence from Efferson et al. (2008) and Morgan et al. (2011) suggests that some individuals will exhibit conformist bias under certain circumstances whereas others will not. An extension to the work here would be to consider the spontaneous uptake of trait A to account for some of this variation. This development would remove the trait-free equilibrium and affect the initial trait frequency which, we have shown in our current model, can have important consequences, such as when conformity bias is strong and the system is bistable.

The general models presented here can be applied to health-related behaviours and thus provide an extension to the existing epidemiological literature, some of

which was discussed in section 3.1. One possible application could be to model the drinking habits of young people in the U.K. Alcohol consumption within this age group is predominantly binge drinking (Institute of Alcohol Studies, 2010, 2013), which is defined as drinking 8+ units for men and 6+ units for women in one drinking session (Deacon et al., 2007). Evidence suggests that peer group influence is a major contributor to an individual choosing to binge drink (French and Cooke, 2012; Institute of Alcohol Studies, 2013), so such behaviour could be considered to be driven by social learning with a likely conformist bias. Our model does not assume differential mortality as the long term health effects of alcohol misuse are unlikely to develop within the modelled timescale. Instead, young adults are likely to leave the modelled population at rate μ as a result of lifestyle changes such as movement out of a student community, or starting a family. For example, Seaman and Ikegwonu (2010) found that young adults in the U.K. were more likely to moderate their drinking when becoming parents.

The frequency-independent term γ may represent reversion resulting from exposure to governmental or mass media campaigns to abstain from binge drinking, while assuming any frequency-dependent influence of susceptible individuals on binge drinkers is small by comparison. The effect of top-down impositions, such as alcohol minimum pricing or the reduction of sweet-tasting or otherwise attractive alcoholic drinks, on binge drinking may be predicted. Such scenarios may be modelled by altering the reversion rate γ and the value of the conformity threshold through η to introduce a content bias into the system. This may provide an initial indicator as to the potential success of proposed strategies to reduce the prevalence of binge drinking within the young adult population.

If appropriate data were obtained then it may be possible to determine whether a biased or unbiased social learning model is most representative of the real-world situation. Once this is understood, comparisons between the actual situation and ideal scenario predictions may be used to inform policy makers or health professionals about how to successfully reduce binge drinking within the population. For instance, if reducing the value of β significantly reduces the number of binge drinkers then efforts would be best focussed on deterring individuals from adopting the be-

haviour, perhaps through media campaigns or by increasing alcohol prices.

In conclusion, we have developed a model for cultural trait transmission within an SIS framework by introducing a nonlinear frequency-dependent relationship with a variable conformity threshold frequency, which could account for the interaction of conformity and content biases acting within the population. Hence, the analysis of the conformity threshold frequency advances cultural evolutionary theory in line with empirical evidence, suggesting that individuals may employ multiple non-independent learning biases.

Chapter 4

A reaction-diffusion model for competing languages

4.1 Introduction

It is widely thought that of the estimated 6000 - 7000 languages in the world, over half will have become extinct by the end of the century (Grenoble and Whaley, 2005). This is a cause of great concern as language death can lead to the irrevocable loss of cultural information. Language provides a means by which individuals can maintain links with their cultural heritage and serves to protect unique aspects of their culture in the present (Grenoble and Whaley, 2005). As culture develops within a particular linguistic framework, the nuances of specific cultural traits may not faithfully translate into an alternative language (Fishman, 2001). Consequently, the trait may not be accurately represented, with subtle differences unapparent to speakers of an alternative language, and hence information may be lost (Fishman, 2001).

The two methods of language extinction are the death of the language-speaking population or speakers abandoning their language in favour of another, known as language shift (Tsunoda, 2006). Population death may occur through natural disasters: for instance all speakers of the Tamboran language of Sumbawa, Indonesia, died following a volcanic eruption in 1815 (Nettle and Romaine, 2000; Hickey, 2013). Genocide is also a cause of population death, as was the case of the Yahi Indians

who were wiped out by white settlers moving into California (Nettle and Romaine, 2000). The languages Wappo and Yuki, also from California, died out via language shift. The last speaker of Wappo, Laura Fish Somersal, regularly used the language throughout her life to communicate with her sister (Hickey, 2013). Arthur Anderson, the final Yuki speaker, last spoke the language in 1908 despite dying in 1990 (Nettle and Romaine, 2000; Golla, 2011). Unlike Somersal, who spent her childhood at home caring for her mother, Anderson was schooled in English and shifted to that language for everyday use (Nettle and Romaine, 2000). Schooling is a key influence on language shift as, via a process of cultural assimilation, individuals will often adopt a common group language (Nettle and Romaine, 2000).

For language shift to occur, speakers of two different languages must interact. Despite this two-way contact, language shift is usually one-directional, with individuals moving from a lower status language to a higher status language (Hickey, 2013). What constitutes lower status is not well-defined, however speakers of minority languages are often stigmatised or excluded from political and educational participation (Brenzinger, 1992). It can therefore be viewed as an advantage to speak the majority language in order to avoid such problems, which may be interpreted as a status advantage. This indicates that minority languages are those that are at risk from language shift. In order for the minority language to be maintained, its speakers must value it highly to overcome the incentive to switch (Brenzinger, 1992).

Such strong language loyalty has been displayed by speakers of Catalan, also known as Valenciana (Catalan, Language of Europe). The majority of its speakers reside in territories located in Spain, with the remainder in Andorra, France and Italy (Catalan, Language of Europe). These Catalan-speaking regions are surrounded by areas dominated by an alternative language, for example Castillian in Spain. During the dictatorship of Spain (1939-1975) the use of Catalan in education, publications and telecommunications was prohibited, but it remained as the language spoken at home in regions such as Catalonia or the Balearic Islands (Catalan, Language of Europe). Since the end of the dictatorship, Catalan became recognised as the native language of the territories of Catalonia, the Balearic Islands and the Comunitat

Valenciana. This allowed for its usage in schools and in government in these areas. From linguistic census data in the region of Catalonia (Idescat Linguistic Census), displayed in Table 4.1, the number of speakers of Catalan forms an increasing trend from 1991 to 2007. This indicates that Catalan is not currently a dying language.

Year	Population (thousands)	Catalan-speaking population	
		Number (thousands)	% of total
1991	5.949	4.066	68.3
1996	5.948	4.506	75.3
2001	6.215	4.603	74.46
2007	7.050	5.331	75.6

Table 4.1: Data from *Idescat Linguistic Census* showing the number of Catalan speakers in Catalonia. This number has increased from 1991 to 2007, implying that Catalan is gaining speakers with the region of Catalonia.

To help understand the conditions under which a language dies or coexists with another, mathematical techniques can be employed. In the last decade, population-wide analytical models pertaining to the spread and persistence of languages have been motivated by the work of Abrams and Strogatz (2003). They model how the numbers of speakers of two competing languages change over time. It is assumed that each language is fixed in structure, for instance grammatically and syntactically, and that they are in competition for speakers. Thus the model does not consider the evolution of a language itself, but the propagation of a language through a population. For simplicity it is assumed that homogeneous mixing occurs within the population and all individuals are monolingual. The attractiveness of the languages increases with the number of speakers and relative perceived status of the language. This takes into account the view that a more dominant language is perceived to have an increased status associated with it, providing an advantage to its speakers over those of the subordinate language. Analysis revealed that the coexistence of two languages, X and Y, was never stable, hence one language would always die out. Predictions were found to correspond with data for the decline of Scottish Gaelic,

Quechua and Welsh.

The model has been criticised for its simplicity. In particular, Steele and Kandler (2010) highlighted the following: languages are assumed to be fixed, spatial and social structure is not incorporated, individuals are monolingual, and the population size is assumed to be constant. Such assessments led to a variety of alternative models being proposed which address some of these criticisms. By including spatial dependence in the Abrams and Strogatz (2003) model, languages can coexist, with speakers divided into distinct zones (Patriarca and Leppänen, 2004). Alternatively, Pinasco and Romanelli (2006) adapted a two-species Lotka-Volterra competition model, where the population does not remain constant, and applied it to language competition. Two languages, $u(t)$ and $v(t)$, increase independently via a logistic growth function. This ensures that there is a cap on population size to represent, for example, environmental constraints on the number of speakers that can be supported. Language u is assumed to be dominant so an interaction term is included allowing speakers of v to convert to dominant language u . Four equilibria exist, one being a stable coexistence state, again contrary to the findings of Abrams and Strogatz (2003). The addition of a spatial component by Kandler and Steele (2008) leads to the same four constant equilibria, however the authors conclude that language coexistence is not possible. Modification of the equations (by further capping the dominant language) yields the possibility of language coexistence under certain conditions.

The inclusion of bilingual speakers into language models is another possible development as, realistically, people do not suddenly switch from being monolingual in one language to monolingual in another. Modifications to the Abrams and Strogatz (2003) model allow for bilingualism between two similar languages (Mira and Paredes, 2005; Mira et al., 2011). Similar languages are those that have a common grammatical structure and some shared vocabulary, such as the Spanish languages Castillian and Galician (Mira and Paredes, 2005; Mira et al., 2011). Bilingualism is found to be stable within the population under some circumstances, with a dependence upon the similarity of the two languages.

A mathematical study of Britain's Celtic languages by Kandler et al. (2010) gave

conflicting results regarding the persistence of a bilingual state, depending on the defined nature of the state (Kandler et al., 2010). In the basic model, bilingualism served as a transitional state; in the other it represented a diglossic state. Diglossia arises within multilingual communities when a certain language is used only in specific circumstances (Romaine, 2000). In Egypt, for example, the publicly recognised language is standard Arabic, whereas a local variant may be used at home (Romaine, 2000). Numerical simulations revealed that one language would always become extinct with the basic model, but a bilingual state is sustainable when assuming a diglossic environment. The models were fitted to 20th century census data to describe language shift from Welsh to English in Wales. Results motivated the implementation of the diglossia model in predicting effective strategies for the revival of Gaelic in Highland Scotland. Other mathematical works also address bilingualism, for example see Minett and Wang (2008) for a general case or Bakalis and Galani (2012) for a study of Greek and Aromanian.

Motivated by previous work, we construct a model to examine the population-wide dynamics of language competition. Our treatment of the model differs from pre-existing work as a global stability analysis of each constant equilibrium is conducted. This builds upon previous language competition models where only local stability criteria have been discussed, e.g. Abrams and Strogatz (2003); Patriarca and Leppänen (2004); Pinasco and Romanelli (2006); Kandler and Steele (2008). In such instances, an equilibrium state can only be classified as stable up to small disturbances. We seek to broaden understanding of how such systems behave by analysing the response to arbitrarily sized disturbances via methods initially employed within the fluid mechanics literature. Research from this area has been applied to aspects of human behaviour. For example, the Cahn-Hilliard equation for fluid phase transitions (see Fabrizio and Mongiovi (2013a,b); Berti et al. (2014)) has been utilised in the study of integration between migrant and resident human populations (Fabrizio and Rivera, in press).

The contribution presented here consists of a logistic growth model with diffusion and a conversion term, first proposed by Kandler and Steele (2008). A key feature of the model is the small number of variables and variable parameters, which is advan-

tageous as it allows for a clear understanding how parameter interactions affect the survival of a language. To ensure a minimal number of parameters are introduced, a separate bilingual class is not considered. Other modelling techniques have been used to assess language survival, such as agent-based models (see Minett and Wang (2008)). These incorporate individual perspectives into the model, however their increased complexity diminishes the clarity of any mathematical results. A review of such methods, assessing their strengths and limitations, may be found in Vogt (2009).

4.2 Reaction-diffusion model for language competition

We construct a model for two competing languages, where one language is assumed to be dominant. We denote the number of speakers of these languages to be $u(\mathbf{x}, t)$ and $v(\mathbf{x}, t)$, which vary over space and time with $\mathbf{x} \in \Omega \subset \mathbb{R}^2$ and $t \in [0, \infty)$. Following the model construction of Kandler and Steele (2008), both languages diffuse and grow logistically, independent of each other. This gives rise to a term $d_1 \Delta u + \alpha_1 u - \beta_1 u^2$ for language u , where d_1 is the diffusion coefficient and α_1 and β_1 are the coefficients associated with logistic growth. A similar result follows for language v .

As indicated by the case of Arthur Anderson (discussed in the Introduction), greater exposure to an different language increases the chance of an individual switching to that language. Combining this with shift being one-directional toward the dominant language, we introduce the cross term γuv into the equations. Choosing u to be dominant, the rate of change in the number of speakers of u is affected by the contact between speakers of the two languages, scaled by a constant γ . This constant represents the strength of the perceived status of language u over language v . As this process involves individuals shifting languages, this also affects

the number of speakers of v . Formulating this gives the equations

$$\begin{aligned}\frac{\partial u}{\partial t} &= d_1 \Delta u + \alpha_1 u - \beta_1 u^2 + \gamma uv, \\ \frac{\partial v}{\partial t} &= d_2 \Delta v + \alpha_2 v - \beta_2 v^2 - \gamma uv,\end{aligned}\tag{4.1}$$

where

$$\Delta = \frac{\partial^2}{\partial x_1^2} + \frac{\partial^2}{\partial x_2^2}$$

is the Laplacian operator in two dimensions. The system can be nondimensionalised by following the method in Cantrell and Cosner (2003). This is advantageous as it simplifies the problem by reducing the number of variable parameters without qualitatively affecting the results. By introducing the variables $t^* = t/T$ and $x^* = x/M$, where T and M are constants that can be chosen, the system with variables $u(\mathbf{x}^*, t^*)$ and $v(\mathbf{x}^*, t^*)$ becomes

$$\begin{aligned}\frac{1}{T} \frac{\partial u}{\partial t^*} &= \frac{d_1}{M^2} \Delta^* u + \alpha_1 u - \beta_1 u^2 + \gamma uv, \\ \frac{1}{T} \frac{\partial v}{\partial t^*} &= \frac{d_2}{M^2} \Delta^* v + \alpha_2 v - \beta_2 v^2 - \gamma uv.\end{aligned}$$

Multiplying both equations by T and choosing $T = M^2/d_1$ this becomes

$$\begin{aligned}\frac{\partial u}{\partial t^*} &= \Delta u + \frac{M^2}{d_1} (\alpha_1 u - \beta_1 u^2 + \gamma uv), \\ \frac{\partial v}{\partial t^*} &= \frac{d_2}{d_1} \Delta v + \frac{M^2}{d_1} (\alpha_2 v - \beta_2 v^2 - \gamma uv).\end{aligned}$$

Choosing $M^2 = d_1/\gamma$ and introducing the positive coefficients $d = d_2/d_1$, $a_1 = \alpha_1/\gamma$, $b_1 = \beta_1/\gamma$, $a_2 = \alpha_2/\gamma$ and $b_2 = \beta_2/\gamma$ leads to the nondimensionalised equations

$$\begin{aligned}\frac{\partial u}{\partial t} &= \Delta u + a_1 u - b_1 u^2 + uv, \\ \frac{\partial v}{\partial t} &= d \Delta v + a_2 v - b_2 v^2 - uv,\end{aligned}\tag{4.2}$$

where the superscript star notation has been dropped for convenience.

It is now of interest to consider the constant equilibria of system (4.2) to determine whether languages u and v will persist or die out over time. At equilibrium, u and v do not vary in time so $\partial u/\partial t = 0$ and $\partial v/\partial t = 0$. As the equilibria are constant solutions, they do not vary spatially so $\Delta u = 0$ and $\Delta v = 0$. Thus the constant equilibria of the system, which we denote generally as (\bar{u}, \bar{v}) , are obtained

by solving

$$0 = \bar{u}(a_1 - b_1\bar{u} + \bar{v}),$$

$$0 = \bar{v}(a_2 - b_2\bar{v} - \bar{u}).$$

Four constant equilibria exist:

$(0, 0)$	Languages u and v become extinct,
$\left(\frac{a_1}{b_1}, 0\right)$	Language u persists and language v dies out,
$\left(0, \frac{a_2}{b_2}\right)$	Language u dies out and language v persists,
$\left(\frac{a_1b_2 + a_2}{b_1b_2 + 1}, \frac{a_2b_1 - a_1}{b_1b_2 + 1}\right)$	Languages u and v coexist.

The first three solutions are easily determined, however the fourth requires solving

$$0 = a_1 - b_1\bar{u} + \bar{v},$$

$$0 = a_2 - b_2\bar{v} - \bar{u}.$$

Following a matrix equation procedure, these equations may be written as

$$\begin{pmatrix} a_1 \\ a_2 \end{pmatrix} = \begin{pmatrix} b_1 & -1 \\ 1 & b_2 \end{pmatrix} \begin{pmatrix} \bar{u} \\ \bar{v} \end{pmatrix}$$

and \bar{u} , \bar{v} found from

$$\begin{pmatrix} \bar{u} \\ \bar{v} \end{pmatrix} = \begin{pmatrix} b_1 & -1 \\ 1 & b_2 \end{pmatrix}^{-1} \begin{pmatrix} a_1 \\ a_2 \end{pmatrix} = \frac{1}{b_1b_2 + 1} \begin{pmatrix} b_2 & 1 \\ -1 & b_1 \end{pmatrix} \begin{pmatrix} a_1 \\ a_2 \end{pmatrix},$$

with $b_1b_2 + 1 \neq 0$ always holding. Equilibria must satisfy $\bar{u} \geq 0$ and $\bar{v} \geq 0$ to ensure that populations are always non-negative, thus the coexistence solution requires $a_2b_1 > a_1$ for it to be feasible. This condition will be assumed to hold in the subsequent analysis.

To establish which of these solutions the system reaches over time we need to analyse the stability of each solution in turn. This is done by investigating the behaviour of the system (4.2) at a point $(\bar{u} + u, \bar{v} + v)$ where $(u, v) \neq (0, 0)$ is some disturbance to the system at equilibrium. The nonlinear perturbation equations of the system are

$$\frac{\partial u}{\partial t} = \Delta u + (a_1 - 2b_1\bar{u} + \bar{v})u + \bar{u}v + (uv - b_1u^2), \quad (4.3)$$

$$\frac{\partial v}{\partial t} = \Delta v + (a_2 - 2b_2\bar{v} - \bar{u})v - \bar{v}u - (uv + b_2v^2). \quad (4.4)$$

We assume that the population sizes remain constant at the boundary so impose the Dirichlet boundary conditions

$$u(\mathbf{x}, t) = v(\mathbf{x}, t) = 0 \text{ on } \partial\Omega,$$

where $\partial\Omega$ is the boundary of the domain Ω .

We begin by determining instability criteria for the equilibria from the linearised perturbation equations, before progressing to analyse the nonlinear system. Examples of using this method of analysis may be found in texts, e.g. Straughan (2004).

4.3 Linear instability analysis

The linearised perturbation equations, found by discarding the second order terms and higher in equations (4.3) and (4.4), are

$$\begin{aligned} \frac{\partial u}{\partial t} &= \Delta u + (a_1 - 2b_1\bar{u} + \bar{v})u + \bar{u}v, \\ \frac{\partial v}{\partial t} &= d\Delta v + (a_2 - 2b_2\bar{v} - \bar{u})v - \bar{v}u. \end{aligned} \quad (4.5)$$

Denoting the eigenfunctions of the Laplacian operator as $\varphi_n(\mathbf{x})$ leads to the eigenfunction equation $\Delta\varphi_n(\mathbf{x}) = -\lambda_n\varphi_n(\mathbf{x})$. The λ_n are the corresponding eigenvalues and we may assume $\lambda_n \leq \lambda_{n+1} \forall n \in \mathbb{N}$. As the eigenfunctions form a basis of $L^2(\Omega)$ we can consider solutions to the linearised system (4.5) to be a linear combination of these functions. We therefore look at solutions of the form

$$\begin{aligned} u &= \sum_{n=1}^{\infty} u_n \varphi_n(\mathbf{x}) e^{\sigma_n t}, \\ v &= \sum_{n=1}^{\infty} v_n \varphi_n(\mathbf{x}) e^{\sigma_n t}, \end{aligned} \quad (4.6)$$

where u_n , v_n and σ_n are constants. For instability, either u or v must grow in time, achieved by any one summation term $\varphi_k(\mathbf{x})e^{\sigma_k t}$ increasing in time. The sign of the real part of σ_n , denoted $\Re(\sigma_n)$, controls whether a solution grows or decays in time. Accordingly, we seek conditions for when the largest $\Re(\sigma_n)$ becomes positive as this represents the first growing term. By choosing σ_1 to have the largest real part, the first growing term will always correspond to $n = 1$ in equations (4.6).

Substituting the $n = 1$ term from equations (4.6) into the linearised equations (4.5) and rearranging leads to

$$\begin{aligned} 0 &= (\sigma_1 + \lambda_1 - a_1 + 2b_1\bar{u} - \bar{v})u_1\varphi_1(\mathbf{x})e^{\sigma_1 t} - \bar{u}v_1\varphi_1(\mathbf{x})e^{\sigma_1 t}, \\ 0 &= (\sigma_1 + d\lambda_1 - a_2 + 2b_2\bar{v} + \bar{u})v_1\varphi_1(\mathbf{x})e^{\sigma_1 t} + \bar{v}u_1\varphi_1(\mathbf{x})e^{\sigma_1 t}. \end{aligned}$$

This gives rise to the equations

$$\begin{aligned} 0 &= (\sigma_1 + \lambda_1 - a_1 + 2b_1\bar{u} - \bar{v})u_1 - \bar{u}v_1, \\ 0 &= (\sigma_1 + d\lambda_1 - a_2 + 2b_2\bar{v} + \bar{u})v_1 + \bar{v}u_1, \end{aligned} \quad (4.7)$$

as $\varphi_1(\mathbf{x})e^{\sigma_1 t} \neq 0$. Instability occurs for $\sigma_1 > 0$ so there is an instability threshold at $\sigma_1 = 0$. By writing equations (4.7) in matrix form as $M(u_1, v_1)^T = \mathbf{0}$, where

$$M = \begin{pmatrix} \sigma_1 + \lambda_1 - a_1 + 2b_1\bar{u} - \bar{v} & -\bar{u} \\ \bar{v} & \sigma_1 + d\lambda_1 - a_2 + 2b_2\bar{v} + \bar{u} \end{pmatrix}, \quad (4.8)$$

then $\det(M) = 0$ as we desire $M(u_1, v_1) \neq (0, 0)$. Calculating this determinant reveals a quadratic equation in σ_1 ,

$$\begin{aligned} 0 &= \sigma_1^2 + (\lambda_1 - a_1 + 2b_1\bar{u} - \bar{v} + d\lambda_1 - a_2 + 2b_2\bar{v} + \bar{u})\sigma_1 \\ &\quad + (\lambda_1 - a_1 + 2b_1\bar{u} - \bar{v})(d\lambda_1 - a_2 + 2b_2\bar{v} + \bar{u}) + \bar{u}\bar{v}, \end{aligned} \quad (4.9)$$

from which the instability boundary for (\bar{u}, \bar{v}) can be established.

Zero solution

For the equilibrium solution $(\bar{u}, \bar{v}) = (0, 0)$, quadratic equation (4.9) becomes

$$\begin{aligned} 0 &= \sigma_1^2 + (\lambda_1 - a_1 + d\lambda_1 - a_2)\sigma_1 + (\lambda_1 - a_1)(d\lambda_1 - a_2) \\ &= (\sigma_1 + \lambda_1 - a_1)(\sigma_1 + d\lambda_1 - a_2). \end{aligned}$$

For this equation to hold then either

$$\sigma_1 = -\lambda_1 + a_1 \quad \text{or} \quad \sigma_1 = -d\lambda_1 + a_2.$$

Instability occurs when any one solution grows: that is, the first instance where $\sigma_1 > 0$. From this we determine the instability boundary for equilibrium point $(0, 0)$ to be

$$\lambda_1 = \min \left\{ a_1, \frac{a_2}{d} \right\}. \quad (4.10)$$

Language u persists

Equilibrium solution $(\bar{u}, \bar{v}) = (a_1/b_1, 0)$ substituted into equation (4.9) gives

$$\begin{aligned} 0 &= \sigma_1^2 + (\lambda_1 + a_1 + d\lambda_1 - a_2)\sigma_1 + (\lambda_1 + a_1) \left(d\lambda_1 - a_2 + \frac{a_1}{b_1} \right) \\ &= (\sigma_1 + \lambda_1 + a_1) \left(\sigma_1 + d\lambda_1 - a_2 + \frac{a_1}{b_1} \right). \end{aligned}$$

This holds for either

$$\sigma_1 = -\lambda_1 - a_1 \quad \text{or} \quad \sigma_1 = -d\lambda_1 + a_2 - \frac{a_1}{b_1}.$$

The first σ_1 solution is always negative, so the only instability condition (resulting from the second σ_1 solution) is the threshold

$$d\lambda_1 + a_1/b_1 = a_2. \quad (4.11)$$

Language v persists

Equilibrium solution $(\bar{u}, \bar{v}) = (0, a_2/b_2)$ substituted into equation (4.9) gives

$$\begin{aligned} 0 &= \sigma_1^2 + \left(\lambda_1 - a_1 - \frac{a_2}{b_2} + d\lambda_1 + a_2 \right) \sigma_1 + \left(\lambda_1 - a_1 - \frac{a_2}{b_2} \right) (d\lambda_1 + a_2) \\ &= \left(\sigma_1 + \lambda_1 - a_1 - \frac{a_2}{b_2} \right) (\sigma_1 + d\lambda_1 + a_2) \end{aligned}$$

so either

$$\sigma_1 = -\lambda_1 + a_1 + \frac{a_2}{b_2} \quad \text{or} \quad \sigma_1 = -d\lambda_1 - a_2.$$

As with the previous case, one solution for σ_1 is always negative, so here the instability threshold is

$$\lambda_1 = a_1 + a_2/b_2. \quad (4.12)$$

Coexistence solution

For the coexistence solution

$$(\bar{u}, \bar{v}) = \left(\frac{a_1 b_2 + a_2}{b_1 b_2 + 1}, \frac{a_2 b_1 - a_1}{b_1 b_2 + 1} \right),$$

equation (4.9) simplifies to

$$0 = \sigma_1^2 + (\lambda_1 + b_1 \bar{u} + d\lambda_1 + b_2 \bar{v})\sigma_1 + (\lambda_1 + b_1 \bar{u})(d\lambda_1 + b_2 \bar{v}) + \bar{u}\bar{v} \quad (4.13)$$

as $\bar{u}, \bar{v} > 0$. Solutions for σ_1 are found from the quadratic formula, so

$$2\sigma_1 = -p \pm \sqrt{p^2 - 4q}$$

where

$$p = \lambda_1 + b_1\bar{u} + d\lambda_1 + b_2\bar{v},$$

$$q = (\lambda_1 + b_1\bar{u})(d\lambda_1 + b_2\bar{v}) + \bar{u}\bar{v},$$

with $p, q > 0$. If the discriminant of equation (4.13) is negative ($p^2 - 4q < 0$) then the solutions for σ_1 are complex conjugates with a negative real part of $\Re(\sigma_1) = -p$. When the discriminant is positive, therefore $\sigma_1 \in \mathbb{R}$, then $-p - \sqrt{p^2 - 4q} < 0$ so $\sigma_1 < 0$. For $\sigma_1 = -p + \sqrt{p^2 - 4q}$, the solution for σ_1 is again negative because $q > 0$, leading to $p > \sqrt{p^2 - 4q}$. In all cases $\Re(\sigma_1) < 0$, therefore no instability criteria can be determined for this solution.

Whilst instability criteria for three equilibria has been obtained by analysing the linearised system, it is not sufficient to determine regions of global stability. In order to obtain this information the nonlinear system must be addressed.

4.4 Nonlinear stability analysis

Global stability criteria for equilibria can be determined by the construction of an energy functional. Energy methods have been used to determine the stability of fluid flow since the work of Orr (1907), with developments by Serrin (1959), Joseph (1965, 1966, 1970) and Rionero (1967, 1968). The aim is to determine if some disturbance to a flow will result in a radical change in behaviour or a progression back to the original flow as $t \rightarrow \infty$. By considering the energy difference between the original and disturbed flow, conditions for which the energy decreases indicate stability of the initial flow. Developments of these methods have been widely used to analyse stability of equilibria, with recent work by Capone and De Luca (2012); Hill and Malashetty (2012); Mulone et al. (2007); Rionero (2009, 2012a); Straughan (2013b). Using an energy argument, we progress to finding a stability threshold for each equilibrium. It is desirable to obtain a stability bound which coincides with the instability threshold as this provides information about the system's behaviour for all possible combinations of parameter values. This is not always possible, but in

some cases may be achieved by placing conditions on the initial state of the system. As will be demonstrated in the following analysis, such conditions are necessary here.

We use $\|\cdot\|$ and (\cdot, \cdot) to denote the L^2 norm and inner product over Ω respectively, so that, for example,

$$\|u\|^2 = \int_{\Omega} u^2 d\Omega \quad \text{and} \quad (u, v) = \int_{\Omega} uv d\Omega. \quad (4.14)$$

We notice that multiplying the left-hand side of equation (4.3) by u and integrating over Ω gives rise to

$$\int_{\Omega} u \frac{\partial u}{\partial t} d\Omega = \frac{1}{2} \frac{d}{dt} \int_{\Omega} u^2 d\Omega = \frac{1}{2} \frac{d}{dt} \|u\|^2$$

by using the definition of the L^2 norm. We achieve a similar result for v from equation (4.4). This is a useful observation as it allows us to construct a function $E(t)$ which is a linear combination of $\|u\|^2$ and $\|v\|^2$, for example

$$E(t) = \frac{1}{2} (\xi \|u\|^2 + \eta \|v\|^2)$$

with $\xi, \eta > 0$ constant. When $t \rightarrow \infty$, $E(t) \rightarrow 0$ only if $u, v \rightarrow 0$, which is the requirement for stability of the solution (\bar{u}, \bar{v}) . Hence we aim to find conditions on the parameters such that $E(t)$ is a decreasing function in time, that is $\dot{E}(t) < 0$ where the dot indicates a time differential.

Multiplying equation (4.3) by u , equation (4.4) by v , and integrating both over the spatial domain Ω results in

$$\begin{aligned} \frac{1}{2} \frac{d}{dt} \int_{\Omega} u^2 d\Omega &= \int_{\Omega} u \Delta u d\Omega + (a_1 - 2b_1 \bar{u} + \bar{v}) \int_{\Omega} u^2 d\Omega + \bar{u} \int_{\Omega} uv d\Omega \\ &\quad + \int_{\Omega} (u^2 v - b_1 u^3) d\Omega, \end{aligned} \quad (4.15)$$

$$\begin{aligned} \frac{1}{2} \frac{d}{dt} \int_{\Omega} v^2 d\Omega &= d \int_{\Omega} v \Delta v d\Omega + (a_2 - 2b_2 \bar{v} - \bar{u}) \int_{\Omega} v^2 d\Omega - \bar{v} \int_{\Omega} uv d\Omega \\ &\quad - \int_{\Omega} (uv^2 + b_2 v^3) d\Omega. \end{aligned} \quad (4.16)$$

These equations may be rewritten via use of the L^2 norm and inner product, and the divergence theorem. The divergence theorem states that, for some function \mathbf{f} over the spatial domain Ω ,

$$\int_{\Omega} \nabla \cdot \mathbf{f} d\Omega = \int_{\partial\Omega} \mathbf{f} \cdot \mathbf{n} dS$$

where \mathbf{n} is the outward-pointing unit normal to the boundary of Ω . Using the method of integration by parts and the divergence theorem we manipulate the integrand $u\Delta u$ from equation (4.15) in the following manner:

$$\begin{aligned}\int_{\Omega} u\Delta u d\Omega &= \int_{\Omega} \nabla \cdot (u\nabla u) d\Omega - \int_{\Omega} (\nabla u)^2 d\Omega \\ &= \int_{\partial\Omega} u\nabla u \cdot \mathbf{n} dS - \int_{\Omega} (\nabla u)^2 d\Omega \\ &= - \int_{\Omega} (\nabla u)^2 d\Omega,\end{aligned}$$

where the integral over $\partial\Omega$ vanishes as $u = 0$ on the boundary. Utilising this method, along with the definitions of the L^2 norm and inner product, equations (4.15) and (4.16) become

$$\begin{aligned}\frac{1}{2} \frac{d}{dt} \|u\|^2 &= -\|\nabla u\|^2 + (a_1 - 2b_1\bar{u} + \bar{v})\|u\|^2 + \bar{u}(u, v) + \int_{\Omega} (u^2v - b_1u^3) d\Omega, \\ \frac{1}{2} \frac{d}{dt} \|v\|^2 &= -d\|\nabla v\|^2 + (a_2 - 2b_2\bar{v} - \bar{u})\|v\|^2 - \bar{v}(u, v) - \int_{\Omega} (uv^2 + b_2v^3) d\Omega.\end{aligned}\tag{4.17}$$

We now construct and analyse a suitable energy function for each equilibrium in turn, in order to derive a stability threshold.

4.4.1 Zero solution

To analyse the solution $(\bar{u}, \bar{v}) = (0, 0)$ we construct the energy function

$$E(t) = \frac{1}{2}(\|u\|^2 + \|v\|^2)\tag{4.18}$$

so that

$$\begin{aligned}\frac{dE}{dt} &= \frac{1}{2} \frac{d}{dt} (\|u\|^2 + \|v\|^2) \\ &= -\|\nabla u\|^2 - d\|\nabla v\|^2 + a_1\|u\|^2 + a_2\|v\|^2 + \int_{\Omega} u^2v d\Omega - b_1 \int_{\Omega} u^3 d\Omega \\ &\quad - \int_{\Omega} uv^2 d\Omega - b_2 \int_{\Omega} v^3 d\Omega\end{aligned}\tag{4.19}$$

after substitution from equations (4.17) with $\bar{u} = 0$ and $\bar{v} = 0$. Introducing

$$D = \|\nabla u\|^2 + d\|\nabla v\|^2,\tag{4.20}$$

$$I = a_1\|u\|^2 + a_2\|v\|^2,\tag{4.21}$$

$$N = \int_{\Omega} u^2v d\Omega - b_1 \int_{\Omega} u^3 d\Omega - \int_{\Omega} uv^2 d\Omega - b_2 \int_{\Omega} v^3 d\Omega\tag{4.22}$$

and comparing with equation (4.19) enables us to write

$$\frac{dE}{dt} = -D + I + N. \quad (4.23)$$

Thus, equation (4.23) involves a right-hand side which separates into a positive-definite quadratic term D , a quadratic term I , and a cubic term N . As will become apparent, writing dE/dt in this way allows for it to be bounded from above by a function of the form $-KE(t)$, $K \in \mathbb{R}$ positive, subject to certain parameter constraints. For a derived range of parameter values, $dE/dt < 0$, so $u, v \rightarrow 0$ as $t \rightarrow \infty$, indicating that $(\bar{u}, \bar{v}) = (0, 0)$ is globally stable.

Bounding $\dot{E}(t)$ for the equilibrium $(0, 0)$

Consider

$$\frac{I}{D} \leq \max_{\mathcal{H}} \left(\frac{I}{D} \right)$$

for $\mathcal{H} = \{u, v | u, v \in \mathcal{H}_0^1(\Omega)\}$ and introduce a constant R_E which satisfies

$$\frac{1}{R_E} = \max_{\mathcal{H}} \left(\frac{I}{D} \right). \quad (4.24)$$

Using $I = D(I/D) \leq D/R_E$, the right-hand side of equation (4.23) may be bounded above to reveal

$$\frac{dE}{dt} \leq -D \left(1 - \frac{1}{R_E} \right) + N.$$

We define the value R_E in this way so that the term $-D(1 - 1/R_E)$ is negative for $R_E > 1$. To progress we assume $R_E > 1$ and then let $q = (1 - 1/R_E)$, thus obtaining

$$\frac{dE}{dt} \leq -Dq + N. \quad (4.25)$$

We now concentrate on bounding N (equation (4.22)), addressing each term separately. To do this we first need the Cauchy-Schwarz inequality for the L^2 inner product space:

$$\left(\int_{\Omega} fg \, d\Omega \right)^2 \leq \int_{\Omega} f^2 \, d\Omega \int_{\Omega} g^2 \, d\Omega, \quad (4.26)$$

where f, g are functions of the domain Ω . To correspond to the terms in N , we use an alternative form of the Cauchy-Schwarz inequality,

$$\left| \int_{\Omega} fg \, d\Omega \right| \leq \left(\int_{\Omega} f^2 \, d\Omega \right)^{\frac{1}{2}} \left(\int_{\Omega} g^2 \, d\Omega \right)^{\frac{1}{2}}. \quad (4.27)$$

We also require the Sobolev inequality

$$\left(\int_{\Omega} f^4 d\Omega \right)^{\frac{1}{4}} \leq c^{\frac{1}{2}} \left(\int_{\Omega} |\nabla f|^2 d\Omega \right)^{\frac{1}{2}}$$

in the form

$$\left(\int_{\Omega} f^4 d\Omega \right)^{\frac{1}{2}} \leq c \left(\int_{\Omega} |\nabla f|^2 d\Omega \right), \quad (4.28)$$

where c is a constant which depends upon the domain Ω (see Gilbarg and Trudinger (1998)).

The first term of N is

$$\int_{\Omega} u^2 v d\Omega \leq \left| \int_{\Omega} u^2 v d\Omega \right|.$$

Applying inequality (4.27), followed by (4.28) with $c = c_1$, gives

$$\int_{\Omega} u^2 v d\Omega \leq \left(\int_{\Omega} u^4 d\Omega \right)^{\frac{1}{2}} \left(\int_{\Omega} v^2 d\Omega \right)^{\frac{1}{2}} \leq c_1 \int_{\Omega} |\nabla u|^2 d\Omega \left(\int_{\Omega} v^2 d\Omega \right)^{\frac{1}{2}},$$

where c_1 is a constant dependent upon Ω . Now we can transform the right-hand side of this inequality using the definition of the L^2 norm (equation (4.14)) to give

$$\int_{\Omega} u^2 v d\Omega \leq c_1 \|\nabla u\|^2 \|v\|. \quad (4.29)$$

Applying the same procedure to the remaining terms of N yields

$$-b_1 \int_{\Omega} u^3 d\Omega \leq b_1 c_1 \int_{\Omega} |\nabla u|^2 d\Omega \left(\int_{\Omega} u^2 d\Omega \right)^{\frac{1}{2}} \leq b_1 c_1 \|\nabla u\|^2 \|u\|, \quad (4.30)$$

$$-\int_{\Omega} uv^2 d\Omega \leq c_2 \int_{\Omega} |\nabla v|^2 d\Omega \left(\int_{\Omega} u^2 d\Omega \right)^{\frac{1}{2}} \leq c_2 \|\nabla v\|^2 \|u\|, \quad (4.31)$$

and

$$-b_2 \int_{\Omega} v^3 d\Omega \leq b_2 c_2 \int_{\Omega} |\nabla v|^2 d\Omega \left(\int_{\Omega} v^2 d\Omega \right)^{\frac{1}{2}} \leq b_2 c_2 \|\nabla v\|^2 \|v\|, \quad (4.32)$$

where c_2 is a constant dependent upon Ω . Comparing the information from inequalities (4.29) to (4.32) with equation (4.22) gives

$$N \leq c_1 \|\nabla u\|^2 (b_1 \|u\| + \|v\|) + c_2 \|\nabla v\|^2 (\|u\| + b_2 \|v\|).$$

From equations (4.18) and (4.20) we may determine that

$$\|u\| \leq \sqrt{2} E^{\frac{1}{2}}(t), \quad \|v\| \leq \sqrt{2} E^{\frac{1}{2}}(t), \quad \|\nabla u\|^2 \leq D \quad \text{and} \quad \|\nabla v\|^2 \leq \frac{D}{d}.$$

Using these, we now bound N by

$$N \leq k_1 D E^{\frac{1}{2}}(t),$$

where

$$k_1 = \sqrt{2} \left(c_1 + c_1 b_1 + \frac{c_2}{d} + \frac{c_2 b_2}{d} \right).$$

An upper bound on $\dot{E}(t)$, following on from inequality (4.25), is therefore

$$\frac{dE}{dt} \leq -D(q - k_1 E^{\frac{1}{2}}(t)). \quad (4.33)$$

We reiterate that we are seeking a bound $\dot{E}(t) \leq -K E(t)$, $K \in \mathbb{R}$ positive, so we need

$$q - k_1 E^{\frac{1}{2}}(t) > 0. \quad (4.34)$$

By assuming the constant value $E^{\frac{1}{2}}(0) < q/k_1$, where $E(0)$ is the initial state of the system at $t = 0$, we can show that

$$E^{\frac{1}{2}}(0) > E^{\frac{1}{2}}(t). \quad (4.35)$$

As a consequence we achieve inequality (4.34) as

$$0 < q - k_1 E^{\frac{1}{2}}(0) < q - k_1 E^{\frac{1}{2}}(t).$$

To prove inequality (4.35) we begin with the assumption $E^{\frac{1}{2}}(0) < q/k_1$, where $E^{\frac{1}{2}}(0) > 0$. It therefore follows from inequality (4.33) evaluated at $t = 0$ that $dE/dt < 0$, so either:

1. $E^{\frac{1}{2}}(t) < q/k_1 \forall t > 0$, indicating that $E(t)$ is a decreasing function;
2. $\exists \eta$ such that for $t = \eta$, $E^{\frac{1}{2}}(\eta) = q/k_1$.

Suppose the second of these options is true. Then, for $t \in (0, \eta)$, $E^{\frac{1}{2}}(t) < q/k_1$ so $E(t)$ is a decreasing function because $dE/dt < 0$. Hence $E(t) < E(0)$ for $t \in (0, \eta)$. By continuity, $E(\eta) < E(0)$ and therefore $E^{\frac{1}{2}}(\eta) < E^{\frac{1}{2}}(0) < q/k_1$ which is a contradiction. Option 2 is therefore impossible so option 1 must be true, that is $E^{\frac{1}{2}}(t) < q/k_1 \forall t > 0$ provided $E^{\frac{1}{2}}(0) < q/k_1$. In this case $E(t)$ is a decreasing

function so it follows that $E^{\frac{1}{2}}(0) > E^{\frac{1}{2}}(t)$. Applying these results to inequality (4.33) we see that

$$\frac{dE}{dt} \leq -D(q - k_1 E^{\frac{1}{2}}(t)) \leq -\alpha D \quad (4.36)$$

where $\alpha = q - k_1 E^{\frac{1}{2}}(0)$.

Poincaré's inequality for functions $\varphi \in \mathcal{H}_0^1(\Omega)$ is

$$\|\nabla\varphi\|^2 \geq \lambda_1 \|\varphi\|^2, \quad (4.37)$$

where $\lambda_1 > 0$ is the first eigenvalue of the membrane problem $\Delta\varphi = -\lambda\varphi$ in Ω and $\varphi_n = 0$ on $\partial\Omega$. Applying Poincaré's inequality to functions $\|u\|^2$ and $\|v\|^2$ in equation (4.20) gives

$$D \geq 2\lambda_1 \frac{\|u\|^2}{2} + 2\lambda_1 d \frac{\|v\|^2}{2} \geq \mu E(t) \quad \text{for } \mu = \min\{2\lambda_1, 2\lambda_1 d\},$$

hence $-D \leq -\mu E(t)$. Using this gives a bound of the desired form:

$$\frac{dE}{dt} \leq -\mu\alpha E(t). \quad (4.38)$$

We now know that when inequality (4.38) holds, $E(t)$ decays in time and therefore $(0,0)$ is a globally stable point. In deriving this bound we assumed that $R_E > 1$ (equivalent to $q > 0$) so we now must find appropriate parameter restrictions which ensure this, and thus find a stability condition in terms of the model parameters.

Finding R_E for $(0,0)$

For inequality (4.38) to hold we have assumed that $R_E > 1$. We now determine the value of R_E and thus obtain constraints on the parameters required for stability. Equation (4.24) informs us that we must maximise I/D which can be done by calculating its variation, $\delta(I/D)$, by the method which we now outline.

We introduce indicial notation to keep calculations compact. In this notation

$$\nabla u = \begin{pmatrix} \frac{\partial u}{\partial x_1} \\ \frac{\partial u}{\partial x_2} \end{pmatrix} \equiv u_{,i}$$

where the subscript comma indicates differentiation and i is the i^{th} component of \mathbf{x} . When an index is repeated it is summed over, so

$$\frac{\partial}{\partial x_i} \left(\frac{\partial f_I}{\partial u_{,i}} \right) = \frac{\partial}{\partial x_1} \left(\frac{\partial f_I}{\partial u_{,1}} \right) + \frac{\partial}{\partial x_2} \left(\frac{\partial f_I}{\partial u_{,2}} \right) = \nabla \cdot \left(\frac{\partial f_I}{\partial (\nabla u)} \right)$$

and

$$\frac{\partial}{\partial x_i} (u_{,i}) = \frac{\partial}{\partial x_1} \frac{\partial u}{\partial x_1} + \frac{\partial}{\partial x_2} \frac{\partial u}{\partial x_2} = \Delta u.$$

Now consider the general form of functionals I and D to be

$$I = \int_{\Omega} f_I(\mathbf{x}, u, v, u_{,i}, v_{,i}) \, d\Omega,$$

$$D = \int_{\Omega} f_D(\mathbf{x}, u, v, u_{,i}, v_{,i}) \, d\Omega.$$

Then, to find the maximum of I/D , we use the method of calculus of variations. Assume u, v are admissible functions such that I/D is maximised. Consider the admissible functions

$$\hat{u} = u + \epsilon \eta^1,$$

$$\hat{v} = v + \epsilon \eta^2,$$

where ϵ is some real number and η^1, η^2 are admissible functions which are zero at the boundary $\partial\Omega$. Then

$$I = \int_{\Omega} f_I(\mathbf{x}, \hat{u}, \hat{v}, \hat{u}_{,i}, \hat{v}_{,i}) \, d\Omega,$$

$$D = \int_{\Omega} f_D(\mathbf{x}, \hat{u}, \hat{v}, \hat{u}_{,i}, \hat{v}_{,i}) \, d\Omega.$$

To maximise $I(\epsilon)/D(\epsilon)$ we need $\epsilon = 0$ (as then $\hat{u} = u$ and $\hat{v} = v$ are maximising functions) and the first derivative with respect to ϵ to equal zero, so

$$\left. \frac{d}{d\epsilon} \left(\frac{I}{D} \right) \right|_{\epsilon=0} = \frac{1}{D} \left[\left. \frac{dI}{d\epsilon} - \frac{I}{D} \frac{dD}{d\epsilon} \right] \right|_{\epsilon=0} = 0.$$

As I/D is maximised, we can replace this with $1/R_E$ and use the standard notation δI and δD to represent the derivatives of I and D with respect to ϵ at $\epsilon = 0$. This gives

$$R_E \delta I - \delta D = 0. \quad (4.39)$$

To calculate δI ,

$$\begin{aligned} \delta I &= \int_{\Omega} \frac{d}{d\epsilon} f_I(\mathbf{x}, \hat{u}, \hat{v}, \hat{u}_{,i}, \hat{v}_{,i}) \, d\Omega \\ &= \int_{\Omega} \eta^1 \frac{\partial f_I}{\partial u} + \eta^2 \frac{\partial f_I}{\partial v} + \eta^1_{,i} \frac{\partial f_I}{\partial u_{,i}} + \eta^2_{,i} \frac{\partial f_I}{\partial v_{,i}} \, d\Omega \end{aligned} \quad (4.40)$$

This integral equation may be manipulated so that dependence on both $\eta^1_{,i}$ and $\eta^2_{,i}$ is eliminated. Using integration by parts,

$$\int_{\Omega} \eta^1_{,i} \frac{\partial f_I}{\partial u_{,i}} \, d\Omega = \int_{\Omega} \frac{\partial}{\partial x_i} \left(\eta^1 \frac{\partial f_I}{\partial u_{,i}} \right) \, d\Omega - \int_{\Omega} \eta^1 \frac{\partial}{\partial x_i} \frac{\partial f_I}{\partial u_{,i}} \, d\Omega.$$

By the divergence theorem

$$\begin{aligned} \int_{\Omega} \frac{\partial}{\partial x_i} \left(\eta^1 \frac{\partial f_I}{\partial u_{,i}} \right) d\Omega &= \int_{\partial\Omega} \eta^1 \frac{\partial f_I}{\partial u_{,i}} n_i dS \\ &= 0 \end{aligned}$$

as a consequence of the boundary conditions on η^1 . Combining these results reveals

$$\int_{\Omega} \eta^1 \frac{\partial f_I}{\partial u_{,i}} d\Omega = - \int_{\Omega} \eta^1 \frac{\partial}{\partial x_i} \frac{\partial f_I}{\partial u_{,i}} d\Omega.$$

The same method may be applied to the $\eta^2_{,i}$ term allowing equation (4.40) to be written as

$$\delta I = \int_{\Omega} \eta^1 \left(\frac{\partial f_I}{\partial u} - \frac{\partial}{\partial x_i} \frac{\partial f_I}{\partial u_{,i}} \right) + \eta^2 \left(\frac{\partial f_I}{\partial v} - \frac{\partial}{\partial x_i} \frac{\partial f_I}{\partial v_{,i}} \right) d\Omega.$$

Similarly, for δD ,

$$\delta D = \int_{\Omega} \eta^1 \left(\frac{\partial f_D}{\partial u} - \frac{\partial}{\partial x_i} \frac{\partial f_D}{\partial u_{,i}} \right) + \eta^2 \left(\frac{\partial f_D}{\partial v} - \frac{\partial}{\partial x_i} \frac{\partial f_D}{\partial v_{,i}} \right) d\Omega.$$

Replacing δI and δD in equation (4.39) leads to

$$\begin{aligned} \int_{\Omega} \eta^1 \left(R_E \frac{\partial f_I}{\partial u} - R_E \frac{\partial}{\partial x_i} \frac{\partial f_I}{\partial u_{,i}} - \frac{\partial f_D}{\partial u} + \frac{\partial}{\partial x_i} \frac{\partial f_D}{\partial u_{,i}} \right) \\ + \eta^2 \left(R_E \frac{\partial f_I}{\partial v} - R_E \frac{\partial}{\partial x_i} \frac{\partial f_I}{\partial v_{,i}} - \frac{\partial f_D}{\partial v} + \frac{\partial}{\partial x_i} \frac{\partial f_D}{\partial v_{,i}} \right) d\Omega = 0. \end{aligned}$$

As this must hold for all possible combinations of admissible functions η^1 and η^2 we have

$$\begin{aligned} R_E \left(\frac{\partial f_I}{\partial u} - \frac{\partial}{\partial x_i} \frac{\partial f_I}{\partial u_{,i}} \right) - \left(\frac{\partial f_D}{\partial u} - \frac{\partial}{\partial x_i} \frac{\partial f_D}{\partial u_{,i}} \right) &= 0, \\ R_E \left(\frac{\partial f_I}{\partial v} - \frac{\partial}{\partial x_i} \frac{\partial f_I}{\partial v_{,i}} \right) - \left(\frac{\partial f_D}{\partial v} - \frac{\partial}{\partial x_i} \frac{\partial f_D}{\partial v_{,i}} \right) &= 0. \end{aligned} \tag{4.41}$$

These are the Euler-Lagrange equations associated with the problem of maximising I/D , which solutions u and v must satisfy.

For the $(0, 0)$ solution,

$$\begin{aligned} f_I &= a_1 u^2 + a_2 v^2, \\ f_D &= (\nabla u)^2 + d(\nabla v)^2, \end{aligned}$$

which are found from applying the definition of the L^2 norm to I (equation (4.21)) and D (equation (4.20)). For these functions, the non-zero terms of equations (4.41)

are

$$\begin{aligned}\frac{\partial f_I}{\partial u} &= 2a_1u, \\ \frac{\partial f_I}{\partial v} &= 2a_2v, \\ \frac{\partial}{\partial x_i} \frac{\partial f_D}{\partial u_i} &= 2 \frac{\partial}{\partial x_i} (u_i) = 2\Delta u, \\ \frac{\partial}{\partial x_i} \frac{\partial f_D}{\partial v_i} &= 2d \frac{\partial}{\partial x_i} (v_i) = 2d\Delta v,\end{aligned}$$

hence the Euler-Lagrange equations are calculated to be

$$R_E a_1 u + \Delta u = 0,$$

$$R_E a_2 v + d\Delta v = 0.$$

These equations are uncoupled so we expect different minimal eigenvalue conditions for the decay of u and v . As the equations are linear we consider solutions of the form

$$\begin{aligned}u &= \sum_{n=1}^{\infty} u_n \varphi_n(\mathbf{x}), \\ v &= \sum_{n=1}^{\infty} v_n \varphi_n(\mathbf{x}),\end{aligned}\tag{4.42}$$

so the Euler-Lagrange equations become

$$R_E a_1 \sum_{n=1}^{\infty} u_n \varphi_n(\mathbf{x}) = \sum_{n=1}^{\infty} \lambda_n u_n \varphi_n(\mathbf{x}),\tag{4.43}$$

$$R_E a_2 \sum_{n=1}^{\infty} v_n \varphi_n(\mathbf{x}) = d \sum_{n=1}^{\infty} \lambda_n v_n \varphi_n(\mathbf{x})\tag{4.44}$$

As λ_1 is the smallest eigenvalue, from equation (4.43) we know that

$$R_E a_1 \sum_{n=1}^{\infty} u_n \varphi_n(\mathbf{x}) > \lambda_1 \sum_{n=1}^{\infty} u_n \varphi_n(\mathbf{x})\tag{4.45}$$

The condition for u to decay is $R_E > 1$. Combining this condition to inequality (4.45), we determine the parameter constraint $\lambda_1 > a_1$ ensures that u will decay. By similar reasoning applied to equation (4.44) we determine that v decays for $d\lambda_1 > a_2$. As both u and v must decay for global stability of the equilibrium $(0, 0)$, the stability threshold is

$$\lambda_1 = \min \left\{ a_1, \frac{a_2}{d} \right\}.\tag{4.46}$$

Through comparison of this result with equation (4.10), we conclude that the instability and global stability thresholds coincide for equilibrium $(\bar{u}, \bar{v}) = (0, 0)$.

4.4.2 Language u survives, v dies out

To analyse the global stability of $(\bar{u}, \bar{v}) = (a_1/b_1, 0)$ we proceed as in section 4.4.1.

First we construct the function

$$E(t) = \frac{1}{2} (\xi \|u\|^2 + \|v\|^2), \quad (4.47)$$

where ξ is a constant that we may choose. The differential with respect to time is

$$\frac{dE}{dt} = \frac{1}{2} \frac{d}{dt} (\xi \|u\|^2 + \|v\|^2),$$

which, after substituting for \bar{u} and \bar{v} in equations (4.17), is equivalent to

$$\begin{aligned} \frac{dE}{dt} = & \xi \|\nabla u\|^2 + d \|\nabla v\|^2 + \xi a_1 \|u\|^2 + \left(a_2 - \frac{a_1}{b_1}\right) \|v\|^2 + \xi \frac{a_1}{b_1}(u, v) \\ & + \xi \int_{\Omega} u^2 v d\Omega - \xi b_1 \int_{\Omega} u^3 d\Omega - \int_{\Omega} u v^2 d\Omega - b_2 \int_{\Omega} v^3 d\Omega. \end{aligned} \quad (4.48)$$

We introduce the terms

$$D = \xi \|\nabla u\|^2 + d \|\nabla v\|^2 + \xi a_1 \|u\|^2, \quad (4.49)$$

$$I = \xi \frac{a_1}{b_1}(u, v) + \left(a_2 - \frac{a_1}{b_1}\right) \|v\|^2, \quad (4.50)$$

$$N = \xi \int_{\Omega} u^2 v d\Omega - \xi b_1 \int_{\Omega} u^3 d\Omega - \int_{\Omega} u v^2 d\Omega - b_2 \int_{\Omega} v^3 d\Omega, \quad (4.51)$$

so that equation (4.48) may be written as

$$\frac{dE}{dt} = -D + I + N. \quad (4.52)$$

Again we wish to bound dE/dt in such a way that allows us to conclude that $E(t)$ is a decreasing function, implying that the perturbations u and v decay, and therefore that the equilibrium is globally stable.

Bounding $\dot{E}(t)$ for $(a_1/b_1, 0)$

To achieve a bound of the form $dE/dt < -KE(t)$, $K \in \mathbb{R}$, we follow the method of section 4.4.1. The constant R_E is introduced, where

$$\frac{1}{R_E} = \max_{\mathcal{H}} \left(\frac{I}{D} \right)$$

for $\mathcal{H} = \{u, v \mid u, v \in \mathcal{H}_0^1(\Omega)\}$. Assuming that $R_E > 1$ and defining $q = 1 - 1/R_E$ to simplify notation, the inequality

$$\frac{dE}{dt} \leq -Dq + N$$

is determined from equation (4.52). We now seek to bound N in such a way that $R_E > 1$ is a sufficient condition for stability.

We bound N (equation (4.51)) term by term using the Cauchy-Schwarz inequality (4.27) and Sobolev inequality (4.28), following the procedure detailed in section 4.4.1. Applying the L^2 norm gives the bound

$$N \leq \xi c_3 \|\nabla u\|^2 (b_1 \|u\| + \|v\|) + c_4 \|\nabla v\|^2 (\|u\| + b_2 \|v\|),$$

where c_3, c_4 are constants depending upon the domain Ω . From equations (4.47) and (4.49), for $E(t)$ and D respectively, we may conclude

$$\|u\| \leq \sqrt{\frac{2}{\xi}} E(t), \quad \|v\| \leq \sqrt{2} E(t), \quad \xi \|\nabla u\|^2 \leq D, \quad \text{and} \quad \|\nabla v\|^2 \leq D/d.$$

Thus

$$N \leq k_2 D E^{\frac{1}{2}}(t)$$

with

$$k_2 = \sqrt{2} \left(c_3 + \frac{c_3 b_1}{\sqrt{\xi}} + \frac{c_4}{d\sqrt{\xi}} + \frac{c_4 b_2}{d} \right).$$

Combining the inequality for N with the bound for $\dot{E}(t)$ gives

$$\frac{dE}{dt} \leq -D(q - k_2 E^{\frac{1}{2}}(t)).$$

By applying Poincaré's inequality (4.37) to $\|u\|^2$ and $\|v\|^2$ in equation (4.49), we find that

$$D \geq 2\xi\lambda_1 \frac{\|u\|^2}{2} + 2\lambda_1 d \frac{\|v\|^2}{2} \geq \mu E(t) \quad \text{for} \quad \mu = \min\{2\lambda_1, 2\lambda_1 d\}.$$

As proven in section 4.4.1, $E^{\frac{1}{2}}(t) < E^{\frac{1}{2}}(0)$ for $E^{\frac{1}{2}}(0) < q/k_2$, assuming that $R_E > 1$ and replacing the constant k_1 with k_2 . So,

$$\frac{dE}{dt} \leq -\mu\alpha E(t)$$

with constant $\alpha = q - k_2 E^{\frac{1}{2}}(0)$. As with the previous case, we must now determine parameter values for this inequality to be true by finding conditions which ensure $R_E > 1$.

Finding R_E for $(a_1/b_1, 0)$

We are interested in when $R_E = 1$ as this provides a global stability threshold for the equilibrium solution $(a_1/b_1, 0)$. Following the general method presented in section 4.4.1, we evaluate equations (4.41) with the functions

$$f_I = \left(a_2 - \frac{a_1}{b_1}\right)v^2 + \xi \frac{a_1}{b_1}uv,$$

$$f_D = \xi(\nabla u)^2 + d(\nabla v)^2 + \xi a_1 u^2.$$

The Euler-Lagrange equations are

$$\Delta u - a_1 u + \frac{a_1 R_E}{2b_1}v = 0,$$

$$\frac{\xi a_1 R_E}{2b_1}u + d\Delta v + R_E \left(a_2 - \frac{a_1}{b_1}\right)v = 0$$

and again we consider solutions of the form of equations (4.42). The Euler-Lagrange equations become

$$\sum_{n=1}^{\infty} \left(-(\lambda_n + a_1)u_n + \frac{a_1 R_E}{2b_1}v_n \right) \varphi_n = 0,$$

$$\sum_{n=1}^{\infty} \left(\frac{\xi a_1 R_E}{2b_1}u_n + \left(-d\lambda_n + R_E \left(a_2 - \frac{a_1}{b_1} \right) \right) v_n \right) \varphi_n = 0.$$

As before, it is sufficient to consider $n = 1$ and, as $u_n, v_n \neq 0$,

$$\begin{vmatrix} -(\lambda_1 + a_1) & \frac{a_1 R_E}{2b_1} \\ \frac{\xi a_1 R_E}{2b_1} & -\left(d\lambda_1 - R_E \left(a_2 - \frac{a_1}{b_1} \right) \right) \end{vmatrix} = 0$$

must hold. After substitution of $R_E = 1$, this gives the stability threshold equation

$$\left(d\lambda_1 - \left(a_2 - \frac{a_1}{b_1} \right) \right) = \frac{\xi a_1^2}{4b_1^2(\lambda_1 + a_1)}. \quad (4.53)$$

Comparing this result with the instability boundary, the two coincide in the limit $\xi \rightarrow 0$.

4.4.3 Language v survives, u dies out

To analyse the global stability of $(\bar{u}, \bar{v}) = (0, a_2/b_2)$ we proceed as in section 4.4.1, beginning with constructing the function

$$E(t) = \frac{1}{2} (\|u\|^2 + \eta \|v\|^2), \quad (4.54)$$

where η is a constant that we may choose. The differential with respect to time is

$$\frac{dE}{dt} = \frac{1}{2} \frac{d}{dt} (\|u\|^2 + \eta \|v\|^2),$$

which, after substituting for \bar{u} and \bar{v} in equations (4.17), is equivalent to

$$\begin{aligned} \frac{dE}{dt} = & -\|\nabla u\|^2 - d\eta \|\nabla v\|^2 + \left(a_1 + \frac{a_2}{b_2}\right) \|u\|^2 - a_2\eta \|v\|^2 - \eta \frac{a_2}{b_2}(u, v) \\ & + \int_{\Omega} u^2 v d\Omega - b_1 \int_{\Omega} u^3 d\Omega - \eta \int_{\Omega} uv^2 d\Omega - b_2\eta \int_{\Omega} v^3 d\Omega. \end{aligned}$$

We introduce

$$D = \|\nabla u\|^2 + d\eta \|\nabla v\|^2 + a_2\eta \|v\|^2, \quad (4.55)$$

$$I = \left(a_1 + \frac{a_2}{b_2}\right) \|u\|^2 - \eta \frac{a_2}{b_2}(u, v), \quad (4.56)$$

$$N = \int_{\Omega} u^2 v d\Omega - b_1 \int_{\Omega} u^3 d\Omega - \eta \int_{\Omega} uv^2 d\Omega - b_2\eta \int_{\Omega} v^3 d\Omega, \quad (4.57)$$

so that

$$\frac{dE}{dt} = -D + I + N.$$

Bounding $\dot{E}(t)$ for $(0, a_2/b_2)$

Following the method outlined in section 4.4.1, we introduce R_E where

$$\frac{1}{R_E} = \max_{\mathcal{H}} \left(\frac{I}{D} \right), \quad \mathcal{H} = \{u, v \mid u, v \in \mathcal{H}_0^1(\Omega)\}.$$

By assuming $R_E > 1$ and letting $q = 1 - 1/R_E$, the bound

$$\frac{dE}{dt} \leq -Dq + N$$

is achieved. We now focus on bounding N so that $R_E > 1$ is a sufficient stability condition. We bound N (equation (4.51)) term by term using the Cauchy-Schwarz inequality (4.27) and Sobolev inequality (4.28), following the method in section 4.4.1. Applying the L^2 norm gives the bound

$$N \leq c_5 \|\nabla u\|^2 (\|v\| + b_1 \|u\|) + \eta c_6 \|\nabla v\|^2 (\|u\| + b_2 \|v\|),$$

where c_5, c_6 are constants depending upon the domain Ω . From equations (4.54) and (4.55), respectively, we see that

$$\|u\| \leq \sqrt{2}E(t), \quad \|v\| \leq \sqrt{\frac{2}{\eta}}E(t), \quad \|\nabla u\|^2 \leq D, \quad \text{and} \quad \eta \|\nabla v\|^2 \leq D/d,$$

so

$$N \leq k_3 D E^{\frac{1}{2}}(t)$$

where

$$k_3 = \sqrt{2} \left(\frac{c_5 b_1}{\sqrt{\eta}} + c_5 b_1 + \frac{c_6}{d} + \frac{c_6 b_2}{\sqrt{\eta d}} \right).$$

Combining the inequality for N with the bound for $\dot{E}(t)$ gives

$$\frac{dE}{dt} \leq -D \left(q - k_3 E^{\frac{1}{2}}(0) \right),$$

where we have used $E^{\frac{1}{2}}(0) > E^{\frac{1}{2}}(t)$ for $E^{\frac{1}{2}}(0) > q/k_3$. By applying Poincaré's inequality (4.37) to $\|u\|^2$ and $\|v\|^2$ in equation (4.55), we find that

$$D \geq 2\lambda_1 \frac{\|u\|^2}{2} + 2\lambda_1 d \eta \frac{\|v\|^2}{2} \geq \mu E(t) \quad \text{for } \mu = \min\{2\lambda_1, 2\lambda_1 d\}.$$

Consequently, we may bound $\dot{E}(t)$ by

$$\frac{dE}{dt} \leq -\mu \alpha E(t)$$

with $\alpha = q - k_3 E^{\frac{1}{2}}(0)$. We now find conditions which ensure $R_E > 1$.

Finding R_E for $(0, a_2/b_2)$

To find a stability threshold for $(0, a_2/b_2)$, we again follow the method in section 4.4.1, using the functions

$$\begin{aligned} f_I &= \left(a_1 + \frac{a_2}{b_2} \right) u^2 - \eta \frac{a_2}{b_2} uv, \\ f_D &= (\nabla u)^2 + d\eta (\nabla v)^2 + \eta a_2 v^2. \end{aligned}$$

The Euler-Lagrange equations

$$\begin{aligned} \Delta u + R_E \left(a_1 + \frac{a_2}{b_2} \right) u - \frac{\eta a_2 R_E}{2b_2} v &= 0, \\ \frac{\eta a_2 R_E}{2b_2} u - d\eta \Delta v + \eta a_2 v &= 0 \end{aligned}$$

and again we consider solutions of the form of equations (4.42). The Euler-Lagrange equations become

$$\begin{aligned} \sum_{n=1}^{\infty} \left(\left(-\lambda_n + R_E \left(a_1 + \frac{a_2}{b_2} \right) \right) u_n - \frac{\eta a_2 R_E}{2b_2} v_n \right) \varphi_n &= 0, \\ \sum_{n=1}^{\infty} \left(\frac{\eta a_2 R_E}{2b_2} u_n + \eta (d\lambda_n + a_2) v_n \right) \varphi_n &= 0. \end{aligned}$$

It is again sufficient to consider $n = 1$, so

$$\begin{vmatrix} -\left(\lambda_1 - R_E \left(a_1 + \frac{a_2}{b_2}\right)\right) & -\frac{\eta a_2 R_E}{2b_2} \\ -\frac{\eta a_2 R_E}{2b_2} & -\eta(d\lambda_1 + a_2) \end{vmatrix} = 0$$

as $u_n, v_n \neq 0$. Substituting in the value $R_E = 1$ gives the stability threshold

$$\left(\lambda_1 - \left(a_1 + \frac{a_2}{b_2}\right)\right) = \frac{\eta a_2^2}{4b_2^2(d\lambda_1 + a_2)}.$$

In the limit $\eta \rightarrow 0$, this coincides with with the instability boundary.

4.4.4 Languages u and v coexist

To analyse the global stability of

$$(\bar{u}, \bar{v}) = \left(\frac{a_1 b_2 + a_2}{b_1 b_2 + 1}, \frac{a_2 b_1 - a_1}{b_1 b_2 + 1}\right)$$

we proceed as in section 4.4.1, beginning with constructing the function

$$E(t) = (\|u\|^2 + \psi\|v\|^2)/2, \quad (4.58)$$

where ψ is a constant that we may choose. The differential with respect to time is

$$\frac{dE}{dt} = \frac{1}{2} \frac{d}{dt} (\|u\|^2 + \psi\|v\|^2)$$

which, after substituting for \bar{u} and \bar{v} in equations (4.17), is equivalent to

$$\begin{aligned} \frac{dE}{dt} = & -\|\nabla u\|^2 - d\psi\|\nabla v\|^2 - b_1 \bar{u}\|u\|^2 - b_2 \psi \bar{v}\|v\|^2 + (\bar{u} - \psi \bar{v})(u, v) \\ & + \int_{\Omega} u^2 v d\Omega - b_1 \int_{\Omega} u^3 d\Omega - \psi \int_{\Omega} u v^2 d\Omega - b_2 \psi \int_{\Omega} v^3 d\Omega. \end{aligned} \quad (4.59)$$

By choosing

$$\psi = \frac{\bar{u}}{\bar{v}} = \frac{a_1 b_2 + a_1}{a_2 b_1 - a_1}$$

the (u, v) term in equation (4.59) is eliminated, thus $\dot{E}(t)$ may be split into a positive definite part

$$D = \|\nabla u\|^2 + d\psi\|\nabla v\|^2 - b_1 \bar{u}\|u\|^2 - b_2 \bar{u}\|v\|^2 \quad (4.60)$$

and an integral part

$$N = \int_{\Omega} u^2 v d\Omega - b_1 \int_{\Omega} u^3 d\Omega - \psi \int_{\Omega} u v^2 d\Omega - b_2 \psi \int_{\Omega} v^3 d\Omega,$$

so

$$\frac{dE}{dt} = -D + N. \quad (4.61)$$

We now seek to bound $\dot{E}(t)$ from above, as in the previous cases. Again we need to address the terms which form N .

Bounding $\dot{E}(t)$ for the coexistence equilibrium

We bound N (equation (4.51)) term by term using the Cauchy-Schwarz inequality (4.27) and Sobolev inequality (4.28), following the method in section 4.4.1. Applying the L^2 norm gives the bound

$$N \leq c_7 \|\nabla u\|^2 (\|v\| + b_1 \|u\|) + \psi c_8 \|\nabla v\|^2 (\|u\| + b_2 \|v\|),$$

where c_7, c_8 are constants depending upon the domain Ω . From equations (4.58) and (4.60), for $E(t)$ and D respectively, we see that

$$\|u\| \leq \sqrt{2}E(t), \quad \|v\| \leq \sqrt{\frac{2}{\psi}}E(t), \quad \|\nabla u\|^2 \leq D \quad \text{and} \quad \psi \|\nabla v\|^2 \leq D/d.$$

Thus we may bound N by

$$N \leq k_4 D E^{\frac{1}{2}}(t), \quad (4.62)$$

where

$$k_4 = \sqrt{2} \left(\frac{c_7 b_1}{\sqrt{\psi}} + c_7 b_1 + \frac{c_8}{d} + \frac{c_8 b_2}{\sqrt{\psi} d} \right).$$

Combining inequality (4.62) for N with equation (4.61) gives

$$\frac{dE}{dt} \leq -D(q - k_4 E^{\frac{1}{2}}(0)),$$

where we have used $E^{\frac{1}{2}}(0) > E^{\frac{1}{2}}(t)$ for $E^{\frac{1}{2}}(0) > 1/k_4$. The proof of this is the same as in section 4.4.1 with q replaced by 1. By applying Poincaré's inequality (4.37) to $\|u\|^2$ and $\|v\|^2$ in equation (4.60), we find that

$$D \geq 2\lambda_1 \frac{\|u\|^2}{2} + 2\lambda_1 d \psi \frac{\|v\|^2}{2} \geq \mu E(t) \quad \text{for} \quad \mu = \min\{2\lambda_1, 2\lambda_1 d\},$$

so

$$\frac{dE}{dt} \leq -\mu \alpha E(t) \quad (4.63)$$

with $\alpha = 1 - k_4 E^{\frac{1}{2}}(0)$. Provided $E^{\frac{1}{2}}(0) > 1/k_4$, hence $\alpha > 0$, we conclude from inequality (4.63) that the coexistence state is always globally stable.

4.5 Results

From the analysis of the perturbation equations (4.3) and (4.4), we have established that it is possible for each of the four states to be globally stable under certain conditions. These results are summarised in table 4.2. We find that the coexistence state is globally stable when it is feasible, so for $a_2b_1 > a_1$, provided that initially $E^{\frac{1}{2}}(0) > 1/k_4$. This is demonstrated in figure 4.1. These figures show the perturbations $u(\mathbf{x}, t)$ and $v(\mathbf{x}, t)$ around the coexistence state over the spatial domain $x, y \in [0, 1] \times [0, 1] \subset \mathbb{R}^2$ at two fixed time points. The perturbations at time $t = 0$ are given by

$$\begin{aligned} u(x, y, t) &= \sin(\pi x) \sin(\pi y) \\ v(x, y, t) &= 0.8 \sin(\pi x) \sin(\pi y), \end{aligned}$$

which are zero on the boundary of $[0, 1] \times [0, 1]$.

The sign of the expression $a_2b_1 - a_1$ is of interest as it controls whether the coexistence state is feasible and stable. From 4.2, we see that the stability condition for the solution $(a_1/b_1, 0)$ becomes

$$d\lambda_1 - a_2 + \frac{a_1}{b_1} > 0$$

in the limit $\xi \rightarrow 0$. By comparing this with the condition for the coexistence state to be unfeasible, $a_1/b_1 > a_2$, the solution $(a_1/b_1, 0)$ is stable provided $E(0)$ is sufficiently small. Thus we know that, regardless of the sign of $a_2b_1 - a_1$, it is always possible for the dominant language u to survive, either solely or in conjunction with v . This result is intuitive as u is the dominant language.

When $a_1 > a_2b_1$ the coexistence of languages is not possible, yet u may survive alone. This may be an intuitive result when examining the roles of the parameters a_1 , b_1 and a_2 in the the model equations (4.2). Growth of language u is affected by the term a_1u and an influx of individuals converting from language v . As u increases, the capping term $-b_1u^2$ has a greater effect on the rate of change of u , thus limits the growth of the language. Language v grows according to the term a_2v and loses individuals to language u . We now consider the inequality $a_1 > a_2b_1$ and assume that one value is significantly greater or smaller than the other two. To highlight this we now simulate the model equations (4.2) in one spatial dimension

<i>Constant Equilibrium</i>	<i>Feasible</i>	<i>Unstable</i>	<i>Stable</i>
$(0, 0)$	Always	$a_1 - \lambda_1 > 0$ or $a_2 - d\lambda_1 > 0$	$a_1 - \lambda_1 < 0$ and $a_2 - d\lambda_1 < 0$
$\left(\frac{a_1}{b_1}, 0\right)$	Always	$a_2 - \frac{a_1}{b_1} - d\lambda_1 > 0$	$a_2 - \frac{a_1}{b_1} - d\lambda_1 < \frac{\xi a_1^2}{4b_1^2(a_1 + \lambda_1)}$
$\left(0, \frac{a_2}{b_2}\right)$	Always	$a_1 + \frac{a_2}{b_2} - \lambda_1 > 0$	$a_1 + \frac{a_2}{b_2} - \lambda_1 < \frac{\eta a_2^2}{4b_2^2(a_2 + d\lambda_1)}$
$\left(\frac{a_1 b_2 + a_2}{b_1 b_2 + 1}, \frac{a_2 b_1 - a_1}{b_1 b_2 + 1}\right)$	$a_2 b_1 > a_1$	—	$a_2 b_1 > a_1$

Table 4.2: Summary of the results from the linear instability and global stability analysis for the four constant equilibria.

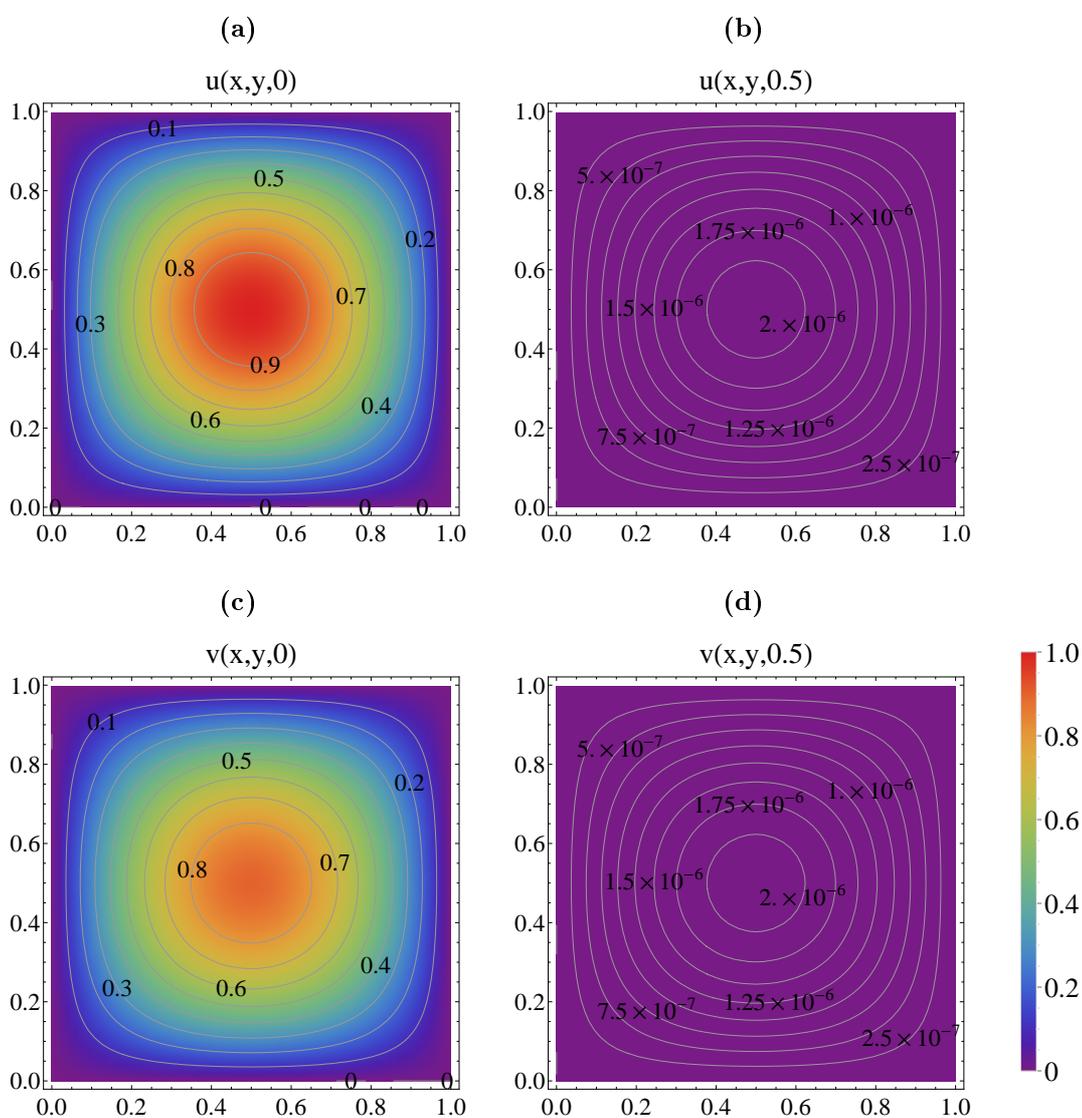


Figure 4.1: Density plots of the perturbations $u(x, y, t)$ and $v(x, y, t)$ around the coexistence equilibrium with $a_1 = 22$, $a_2 = 16$, $b_1 = 6$, $b_2 = 3$ and $d = 0.8$. As time increases the perturbations will eventually reach the x, y -plane, as indicated by the plots at the discrete time points $t = 0$ and $t = 0.5$.

$x \in [0, 1]$. In this instance $u(x, t)$ and $v(x, t)$ represent the speakers of each language rather than the perturbations. The zero-flux boundary conditions $\partial u/\partial x = 0$ and $\partial v/\partial x = 0$ when $x = 0$ and $x = 1$ are assumed, preventing speakers from leaving the spatial region $[0, 1]$.

If a_1 is large in comparison to a_2 and b_1 then language u grows quickly, and faster than language v . This can be seen in figure 4.2a. The logistic growth term $(a_1 - b_1 u)u$ is positive even for large u provided a_1 is sufficiently large, contributing to a positive value for the rate of change of u with respect to time. The proportion of v speakers is depleted by conversions to language u so, over time, large a_1 allows for language u to prevail whilst v becomes extinct. If instead a comparatively small value for a_2 is assumed then language v grows slowly. As demonstrated in figure 4.2b, a small a_2 may be insufficient for the growth of the language. As before, u initially increases rapidly compared to v and some individuals will convert to language u . The cap controlled by b_1 may have greater effect, however the population growth of v may be so slow that the language may die out before the cap on u can have any significant effect. Finally, if b_1 is presumed to be significantly smaller than a_1 and a_2 then the capping term $-b_1 u^2$ has very little effect on the growth rate of language u , even when u is large. As such, language u will continually increase independently by the term $a_1 u$ and also by gaining speakers from language v . If a_1 and a_2 are similar growth rates, then language v will not increase quickly enough to offset the loss of speakers to language u and thus will eventually die out. In the example in figure 4.2c, $a_1 < a_2$ yet language u still dominates.

4.6 Discussion

We have presented a model for two competing languages, seeking to determine if language coexistence is possible. The stability analysis results indicate that, subject to appropriate parameter constraints, each of the four equilibria may be stable. Thus we conclude that the coexistence of two competing languages is possible (in line with Patriarca and Leppänen (2004) and Pinasco and Romanelli (2006)) but stability of the state may be dependent upon the initial number of speakers of both

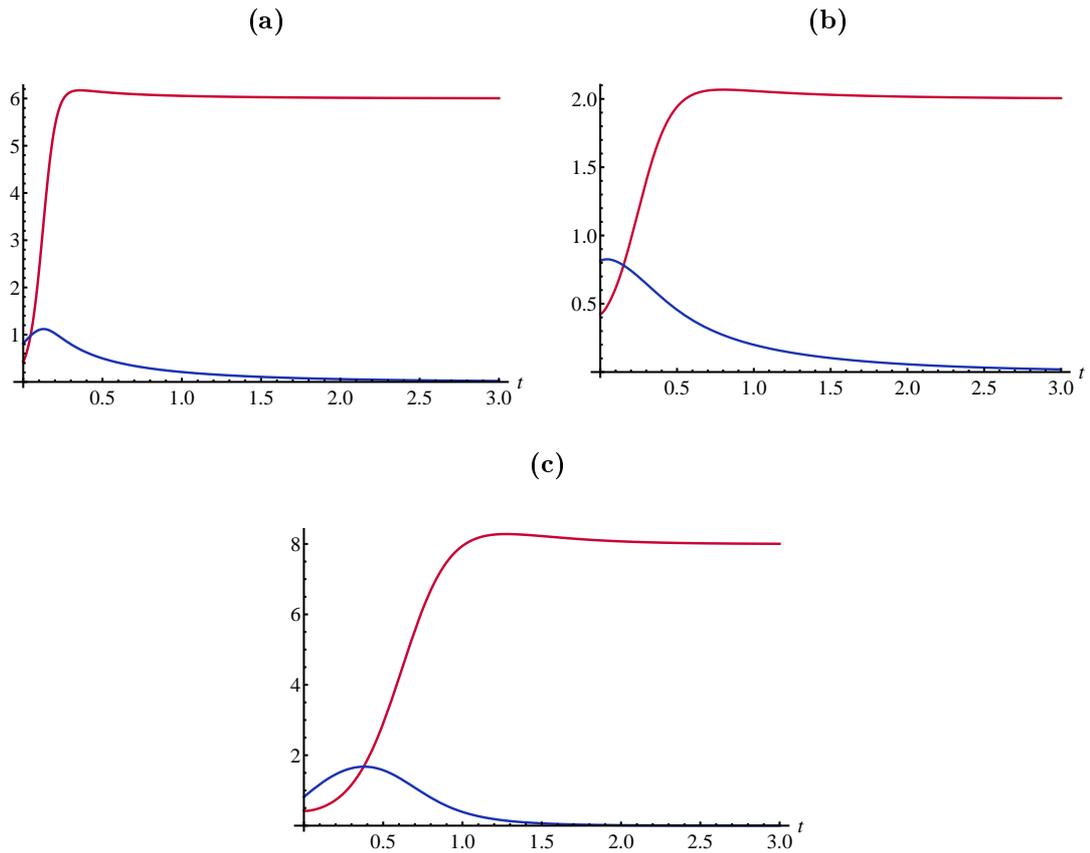


Figure 4.2: Plots of $u(x,t)$ (pink line) and $v(x,t)$ (blue line) from equations (4.2) showing the evolution of speakers in time where $x = 0.7$. The parameter values are $b_2 = 2$, $d = 0.8$ and **(a)** $a_1 = 24$, $a_2 = 5$ and $b_1 = 4$, **(b)** $a_1 = 8$, $a_2 = 1$ and $b_1 = 4$, **(c)** $a_1 = 4$, $a_2 = 5$ and $b_1 = 0.5$. The distribution of speakers at $t = 0$ is given by the functions $u(x,0) = [2\pi x - \sin(2\pi x)]/4\pi$ and $v(x,0) = [\pi + \cos(\pi x)]/\pi$, so $u(0.7,0) = 0.426$ and $v(0.7,0) = 0.813$.

u and v . This conclusion is supported by the real-world example of Catalan as, despite being a minority language, it is gaining speakers within certain regions (as demonstrated in table 4.1).

To fully utilise this model, data for a specific instance of language competition is required. Once obtained, predictions may be made which indicate whether language coexistence is possible, determined by the parameter values. Ideal scenarios can be run where the effect of theoretical campaigns to save an endangered language are incorporated into the parameter values. This could advise policy makers on appropriate strategies required to save an endangered language, such as providing education in the minority language.

Our conclusion that all four equilibria may be stable differs from the findings of Kandler and Steele (2008). They determine that none of the equilibria are both feasible and locally stable. This difference is a result of two modelling factors: the choice of boundary conditions and the imposition of carrying capacity restrictions. By choosing zero-flux boundary conditions, which restricts individuals to remaining within the specified spatial domain, a different analytical approach may be undertaken to analyse the local stability of the system equilibria (see Wang and Zhao (2012)). They also introduce separate carrying capacities for each language, which represents environmental constraints on the number of speakers able to be maintained. We chose to assume that any environmental cap on the number of speakers will affect the total number of individuals, comprised of both u and v speakers, rather than assuming separate capacities for each language. This is because we consider environmental constraints to affect human population growth as a whole, and languages to then compete for speakers within this total population. Thus the cap is on the size of the human population rather than on the number of speakers of a particular language. Our results are therefore not incompatible with the conclusions of Kandler and Steele (2008), but highlight the effect of mathematical assumptions on model predictions.

Whilst our model does not contain a separate class of bilingual speakers, this does not exclude its applicability to such circumstances as it is not necessary to assume that u and v represent monolingual speakers. A two-state model for the Aromanian

language in Greece denotes monolingual Greek speakers as X and bilingual speakers of Greek and Aromanian as Y (Bakalis and Galani, 2012). The survival of the minority language Aromanian is dependent upon bilingual speakers, so a two-variable model can appropriately represent bilingual societies. Alternatively, u and v could represent the preferred language used by individuals. Whilst this may not accurately predict the complete eradication of a language, it could still provide useful information regarding sociolinguistic preferences over time.

As discussed in the section 4.1, not all mathematical models for language competition contain a spatial component (see Abrams and Strogatz (2003); Pinasco and Romanelli (2006)). In particular, the construction of the model by Pinasco and Romanelli (2006) is equivalent to equations (4.1) with the spatial dependence removed. The stability results found here should therefore coincide with the results of Pinasco and Romanelli (2006) if we dismiss the terms associated with diffusion (λ_1 and $d\lambda_1$). From table 4.2 we see that, without these terms, the equilibria $(0, 0)$ and $(0, a_2/b_2)$ would always be unstable. Thus the inclusion of diffusion into the model has a qualitative effect on the predicted outcome when compared with the model without diffusion.

If diffusion of u or v speakers is rapid compared with the respective population growth rates then fewer individuals will be concentrated in one region. This reduces the interaction between speakers of the same language and thus reduces reproductive opportunities, which may lead to population extinction. Rapid diffusion also decreases interaction between speakers of competing languages. The equilibrium $(0, a_2/b_2)$ can be stable when the diffusion of language v is not sufficient for the language to die out, however a comparatively slow growth rate of u combined with few opportunities to convert speakers of language v results in language u dying out. As the model differs with the one without diffusion, the choice of model must depend on the specifics of the real-world situation. For instance, for a model for monolingual and bilingual speakers like that of Bakalis and Galani (2012), diffusion may be an unnecessary complication to the model. If both languages are established within a population and the advantage of the dominant language comes from, for example, trade opportunities with neighbouring regions where the dominant language

is spoken, then the modelled population may be unlikely to migrate to new areas and thus spatial factors may have little effect on language dynamics. If, however, a situation arose where speakers of language u invaded a region populated with v speakers (similar to farmers invading regions of hunter-gatherers (Aoki et al., 1996)) then diffusion dynamics may play a greater role in the spread and survival of both languages.

This model was motivated by existing literature regarding the coexistence of languages with the aim of furthering understanding about the extinction of minority languages. Our contribution extends current knowledge by providing a global stability analysis of the equilibria of a system which assume that language survival is dependent upon both space and time. Binary choice models such as this language may be applied to other behaviours, for example religion (Abrams et al., 2011). Thus the application of this model may extend to a variety of cultural traits where one variant is deemed to have an advantage over the other.

Chapter 5

Conclusion

5.1 Discussion

Chapters 2 to 4 comprise mathematical models for cultural trait transmission via frequency-dependent social learning processes. Such models provide population-level information regarding the persistence and frequency of a cultural trait over time. Each model is a system of differential equations which are not analytically solvable, but the long-term behaviour of the systems may be determined by analysing the stability of the equilibria.

In chapter 2 we assume that problem drinking is a socially learned behaviour where uptake occurs in a linear (unbiased) frequency-dependent fashion. Individuals are classified as either susceptible to developing a drinking problem, problem drinkers, or those recovering from a drinking problem. Analysis revealed that to reduce the frequency of problem drinkers it would be most effective to discourage the initial adoption of the problem drinking behaviour. The effect of total recovery, controlled by parameter γ , was investigated by comparing results with those from the model without total recovery ($\gamma = 0$). Removing the possibility of total recovery affects the endemic frequency of individuals with alcohol problems, however whether the frequency is increased or decreased is dependent upon other variable parameters.

A model for the biased transmission of a cultural trait is presented in chapter 3, seeking to identify how learning biases may affect the population-wide persistence of a cultural trait by utilising some of the mathematical techniques introduced in

chapter 2. The population is split into individuals susceptible to acquiring trait A (type S) and individuals displaying trait A (type A), where the adoption of trait A by type S individuals is dependent upon the frequency of trait A individuals within the population. The model with linear (unbiased) frequency-dependent trait transmission is evolved to represent cases of nonlinear (conformist) biased and content biased transmission, controlled by model parameters D and η respectively. The effects of these social learning biases can be determined by comparing the number and nature of equilibria of the model with transmission biases to the model without biased learning. We find that increasing the conformity strength leads to a bistable equilibrium, hence the persistence of type A individuals within the population is dependent upon the initial state. In table 3.2, section C, the conformity function c_1 with values $\beta = 0.45$ and $D = 0.7$ results in a bistable equilibrium with values $\bar{a} = 0$ and $\bar{a} = 0.380$. An initial value of $a(0) = 0.258$ provides a threshold which determines which of these states is attained. Manipulation of η in c_2 , controlling the effect of a content bias, alters the behaviour invasion threshold such that increasing η increases the conformity threshold frequency.

This model may be applied to a variety of cultural traits where an individual can be in one of two states: displaying trait A (type A) or not displaying trait A (type S). In chapter 3 an application of the model to binge drinking behaviour is discussed, although it may be applied to a variety of different health-related behaviours. For example, both the SARS (chapter 2) and SAS model may represent drug-taking, smoking and eating behaviours, with parameter constraints imposed when appropriate. The choice of model will depend upon the characteristics of the behaviour. Tobacco contains nicotine which is highly addictive and therefore individuals find stopping smoking difficult and will usually enter a period of treatment or recovery before quitting (Benowitz and Henningfield, 2013). The SARS model would be the more appropriate choice as a treatment period is incorporated. In other situations a recovery period may be unnecessary or be of insignificant duration so the SAS model would be the preferred choice. The drug LSD is not thought to be addictive so behaviour cessation may not entail a period of recovery (Lüscher and Ungless, 2006); the SAS model may be more appropriate for representing LSD use.

If applying the SARS model to different behaviours it may be desirable to include a conformity bias in order to better represent the social learning process driving the behaviour adoption. For example, adolescents in the USA are more likely to take up smoking if many of their peers smoke, and this may be in a conformist fashion (Simons-Morton and Farhat, 2010). Whilst the inclusion of a conformity bias may increase the accuracy of the SARS model when applied to some behaviours, it will also increase the model complexity. As a result, finding the model equilibria and analysing their stability will be more difficult. The equilibria of the SARS model in chapter 2 may be found in terms of the model parameters. Equations (2.3) at equilibrium, where $(\dot{a}, \dot{r}) = (0, 0)$, are easily manipulated to find

$$r = \frac{\varphi}{\rho + \mu + \gamma}a,$$

from which

$$0 = a[-\beta(\rho + \mu + \gamma + \varphi)a + \beta(\rho + \mu + \gamma) - \mu(\rho + \mu + \gamma + \varphi) - \gamma\varphi], \quad (5.1)$$

an equation in terms of a only, is obtained. Equation (5.1) is a factorised quadratic polynomial, therefore solving for a , and consequently finding the system equilibria, is possible and relatively simple.

For the SAS model with conformity, finding the system equilibria requires solving the cubic polynomial (3.7) which cannot be factorised. As discussed in chapter 3, finding the equilibria in terms of the model parameters is possible, however their complexity greatly reduces their utility with regard to interpreting the results in a real-world context. The stability analysis was possible because the system is 1-dimensional, which allowed for properties of cubic polynomials to be utilised. If a similar conformity function was introduced into the SARS model then it would be necessary to solve a system of nonlinear polynomial equations in order to find the system equilibria. As demonstrated by the analysis of the SAS model, finding these equilibria in terms of the model parameters will be difficult and, if obtained, are unlikely to be mathematically tractable. As the SARS system only reduces to a 2-dimensional problem, the procedure to determine the stability of equilibria which is presented in chapter 2 cannot be implemented. It is probable that the increased complexity resulting from the inclusion of a conformity bias in the SARS model will

render analytic methods unfruitful. A numerical solving approach would be more appropriate for analysing such a model.

Aside from the inclusion of a conformity bias, the SARS model of chapter 2 could be developed to include the asocial learning of drinking behaviour. Mathematically this could be represented by introducing a variable parameter α and a term αS taking individuals from class S to class A . This allows individuals to develop a behaviour, possibly through innovation or trial and error learning, which is currently not present within the population. Similarly, an asocial learning term could be introduced into the SAS conformity model. Asocial learning mechanisms have been incorporated into SIS-type models (Hill et al., 2010a,b) and a model for the conformist transmission of a cultural trait (Eriksson and Coultas, 2009). Including an asocial learning term would generalise the models, thus increasing their applicability, however the addition of an extra term is likely to increase the calculational difficulty. Again, numerical methods may be the best option for solving such systems. Alternatively, it may be possible to obtain analytic results if such systems were simplified in other ways, such as reducing the number of other variables. This would highlight different key features of the real-world situation, indicated by a different set of simplifying assumptions.

In chapter 4 the spread and persistence of two competing languages is modelled by a reaction-diffusion system, where the language frequency is dependent upon both space and time. The implementation of different mathematical techniques was necessary to handle the increased complexity arising from a PDE, rather than ODE, system. The four constant equilibria of the system were found, and their stability analysed. The language coexistence state was of particular interest and was found to be globally stable. Analysis revealed that the coexistence of languages, where one is dominant, is a stable equilibrium state, subject to the parameter restriction $a_2 b_1 > a_1$ and sufficiently small initial populations of u and v speakers such that $\alpha > 0$ in inequality (4.63).

This is an extension of the work in previous chapters as the model incorporates both a spatial and temporal dependence, thus increases the mathematical complexity. The model does not exhibit the form of an SIR-type model with diffusion,

however its formulation again stems from dynamical systems models in biology. In particular it contains a logistic growth term used in ecology and the model itself is similar to a Lotka-Volterra competition model with diffusion (see Murray (2003); Cantrell and Cosner (2003)). As with the SARS alcohol model, the reaction-diffusion language model may be applied to other cultural traits which are in competition. For example, it could represent the spread of religious attitudes. For some traits it may be of interest to consider the effect of a conformist bias, where the strength of the status advantage of u is dependent on the proportion of u speakers within the population. This is discussed further in section 5.2.

The main aspect of the research which warrants improvement is the use of data to empirically verify the model assumptions and thus achieve accurate predictions. Whilst some parameter estimates are made for the SARS model in section 2.2.5, the social influence parameter β could not be approximated. To increase the utility of the models, appropriate data sets are required. It may be possible to obtain data from existing studies, as was the case with the SIS obesity model by Hill et al. (2010b) which referred to the Framingham Heart Study Network. Alternatively, an experiment could be designed to test the model by enabling appropriate data to be collected. This could then be fit to the model parameters to test if the model predictions match the experimental outcome. This is what was done by Efferson et al. (2008).

As discussed by Morgan et al. (2011), many models assume that only one social learning bias is in operation which may not be an accurate representation of the real world situation. This issue was addressed in chapter 3 by developing a model for both conformist and content biased transmission. The type of models discussed assume homogeneous mixing, where each individual has an equal chance of interacting with any other, and the influence exerted by each individual on another is equal. Thus including conformity or content biases, which may be assumed to have a population-wide influence, is a natural development. This method does not lend itself to representing model-based biases however, as this requires treating at least one individual (the model) differently.

In chapter 2 the concept of a recovery champion class (R_T) allows for success and

similarity biases by assuming that all recovery champions have an equal influence over individuals in treatment. Generally the creation of another class may not provide an accurate enough model. In such cases an alternative method may be required, perhaps considering individuals to be connected on a network. In this instance only connected individuals could influence each other. A model-based bias could be represented by allowing one individual to have a greater influence over other individuals than anyone else. The degree of influence could be proportional to the number of contacts the model individual has, assuming that the having more contacts indicates increased status within the social network.

5.2 Future Work

Future work will focus on extending the competing languages model of chapter 4 to include a conformist social learning bias. Kandler and Laland (2009) constructed a reaction-diffusion model for n competing cultural variants to investigate the effect of innovation on the level of cultural diversity within a population. Within their investigation they consider how a conformist influence compares with an unbiased learning model. They find that a low to moderate conformity strength decreases the cultural diversity at equilibrium. Their model formulation may be viewed as an advancement of the language model in chapter 4 as the system has been generalised to represent n cultural variants and includes more mathematically complex interaction terms. However, the focus of the model was to determine how innovation affects cultural diversity and thus the mathematical formulation contains specific functions to represent this. As the proposed future work does not investigate the effect of innovation, the model is different to that of Kandler and Laland (2009) so their results cannot be assumed to apply to the language model with conformity, which is outlined below.

As discussed in chapter 3, section 3.2, a cubic polynomial term may be used to represent a conformist influence. The interaction term uv in system (4.2) may be replaced with

$$uv(1 + k(2u - \eta)(1 - u)), \quad (5.2)$$

which includes a conformity function of the same form as c_2 (equation (3.9), section 3.3). In this instance k represents the strength of conformity and η , representing a possible content bias, controls the frequency for which trait uptake is equal to that of the linear frequency-dependent case. The resulting model for language competition with conformity is therefore

$$\begin{aligned}\frac{\partial u}{\partial t} &= \Delta u + a_1 u - b_1 u^2 + uv(1 + k(2u - \eta)(1 - u)), \\ \frac{\partial v}{\partial t} &= d\Delta v + a_2 v - b_2 v^2 - uv(1 + k(2u - \eta)(1 - u)).\end{aligned}\tag{5.3}$$

The constant equilibria occur when $(u_t, v_t) = (0, 0)$ and $(\Delta u, \Delta v) = (0, 0)$ so may be found by solving

$$\begin{aligned}0 &= a_1 u - b_1 u^2 + uv(1 + k(2u - \eta)(1 - u)), \\ 0 &= a_2 v - b_2 v^2 - uv(1 + k(2u - \eta)(1 - u)).\end{aligned}\tag{5.4}$$

Three constant equilibria which can easily be found are

$$\begin{aligned}(\bar{u}, \bar{v}) &= (0, 0), \\ (\bar{u}, \bar{v}) &= \left(\frac{a_1}{b_1}, 0\right), \\ (\bar{u}, \bar{v}) &= \left(0, \frac{a_2}{b_2}\right).\end{aligned}$$

These are also solutions to the system without conformity, equations (4.2). The analytic approaches which have been used in previous chapters to find all equilibria cannot be utilised in this case. Future work will focus on using numerical methods to find equilibria of system (5.3) and determining which equilibrium the system reaches for certain parameter sets. These results may then be compared with the findings of chapter 4 to try and obtain a greater understanding of how future predictions are affected by assuming a conformist bias by looking for qualitative differences in system behaviour.

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Appendix A

A.1 The Monty Hall problem

A gameshow contestant, wishing to win a car, is given the choice of three doors: A, B or C. The car is behind one door, and goats behind the other two. The contestant chooses a door then the gameshow host opens one remaining door to reveal a goat. The contestant then has the opportunity to switch from their door to the other unopened door. Should they stick or switch?

Intuitively the result appears to be that the probability of winning the car is $1/2$ if you stick or switch, so neither option increases the chance of winning the car. However, the chance of winning the car when switching is $2/3$ and $1/3$ for sticking, hence it is always beneficial to switch. This arises as the host is restricted in his choice of door as he must always reveal a goat.

Assume that the contestant picks door A. If the car is behind door A, which has probability $1/3$, then the probability of winning the car when sticking is 1 and 0 when switching. If the car is behind B or C (probability $2/3$), then one goat is behind door A and the host is forced to reveal the second goat from behind either B or C. The car is behind the door that the host does not open so the probability of winning the car is 0 when sticking and 1 when switching. The probability of winning the car when not switching is therefore $1 \times 1/3 = 1/3$ and the probability of winning when switching is $1 \times 2/3 = 2/3$. Thus, the contestant should switch doors to maximise their chance of winning the car.

Appendix B

B.1 Proof of $y_1 > 0$ implies $x_1 > 0$

We need to show that $x_1 > 0$ always holds provided $y_1 > 0$, where

$$\begin{aligned}x_1 &= \varphi + \rho + 2\mu + \gamma - \beta, \\y_1 &= -\rho\varphi + (\rho + \mu + \gamma)(\varphi + \mu - \beta).\end{aligned}$$

We first consider the inequality $y_1 > 0$ which can be written in terms of the parameters as

$$\begin{aligned}0 &< -\rho\varphi + (\rho + \mu + \gamma)(\mu + \varphi - \beta) \\ \Leftrightarrow 0 &< -\beta(\rho + \mu + \gamma) + \mu(\rho + \mu + \gamma + \varphi) + \gamma\varphi \\ \Leftrightarrow \beta(\rho + \mu + \gamma) &< \mu(\rho + \mu + \gamma + \varphi) + \gamma\varphi.\end{aligned}\tag{B.1.1}$$

We now consider the necessary condition for $x_1 > 0$ by rewriting this inequality in terms of the parameters,

$$\begin{aligned}0 &< \varphi + \rho + 2\mu + \gamma - \beta \\ \Leftrightarrow \beta &< \varphi + \rho + 2\mu + \gamma.\end{aligned}\tag{B.1.2}$$

We now multiply inequality (B.1.2) by $(\rho + \mu + \gamma)$ so that it may be directly compared with (B.1.1), which results in

$$\begin{aligned}\beta(\rho + \mu + \gamma) &< (\varphi + \rho + 2\mu + \gamma)(\rho + \mu + \gamma) \\ \Leftrightarrow \beta(\rho + \mu + \gamma) &< \mu(\rho + \mu + \gamma + \varphi) + \gamma\varphi + \mu^2 + \rho\varphi \\ &\quad + (\rho + \gamma)(2\mu + \rho + \gamma).\end{aligned}\tag{B.1.3}$$

By comparison, we see that inequality (B.1.1) imposes a stronger condition on $\beta(\rho + \mu + \gamma)$ than inequality (B.1.3). From this we conclude that $y_1 > 0$ implies $x_1 > 0$.

B.2 Endemic equilibrium solution calculations

B.2.1 Proof of $y_2 > 0$ implies $x_2 > 0$

We need to show that $x_2 > 0$ always holds provided $y_2 > 0$, where

$$\begin{aligned} x_2 &= 2\beta\bar{a} + \beta\bar{r} + \varphi + 2\mu + \rho + \gamma - \beta, \\ y_2 &= \varphi(\beta\bar{a} - \rho) + (\rho + \mu + \gamma)(2\beta\bar{a} + \beta\bar{r} + \varphi + \mu - \beta). \end{aligned}$$

We can write y_2 in terms of x_2 as

$$\begin{aligned} y_2 &= \varphi(\beta\bar{a} - \rho) + (\rho + \mu + \gamma)([2\beta\bar{a} + \beta\bar{r} + \varphi + 2\mu + \rho + \gamma - \beta] - [\rho + \mu + \gamma]) \\ &= \varphi(\beta\bar{a} - \rho) + (\rho + \mu + \gamma)(x_2 - [\rho + \mu + \gamma]), \end{aligned} \tag{B.2.4}$$

and, from (2.8), the equation for \bar{a} in terms of y_2 as

$$\bar{a} = \frac{y_2}{\beta(\rho + \mu + \gamma + \varphi)}. \tag{B.2.5}$$

Substituting equation (B.2.5) into equation (B.2.4) gives

$$y_2 = \frac{\varphi y_2}{\rho + \mu + \gamma + \varphi} - \rho\varphi + (\rho + \mu + \gamma)(x_2 - [\rho + \mu + \gamma]),$$

from which we find the equation for x_2 ,

$$x_2 = \frac{y_2}{\rho + \mu + \gamma + \varphi} + \frac{\rho\varphi}{\rho + \mu + \gamma} + \rho + \mu + \gamma. \tag{B.2.6}$$

From equation (B.2.6) we see that $x_2 > 0$ is always true if $y_2 > 0$.

B.2.2 Simplification of y_2

We have

$$y_2 = \varphi(\beta\bar{a} - \rho) + (\rho + \mu + \gamma)(2\beta\bar{a} + \beta\bar{r} + \varphi + \mu - \beta)$$

and we want to write this equation in terms of the model parameters only. We begin by substituting for \bar{r} using

$$\bar{r} = \frac{\varphi}{\rho + \mu + \gamma} \bar{a},$$

which follows from equations (2.8). This gives

$$\begin{aligned} y_2 &= \varphi(\beta\bar{a} - \rho) + (\rho + \mu + \gamma) \left(2\beta\bar{a} + \frac{\beta\varphi}{\rho + \mu + \gamma} \bar{a} + \varphi + \mu - \beta \right) \\ &= \varphi(\beta\bar{a} - \rho) + (\rho + \mu + \gamma)(\varphi + \mu - \beta) + \beta\bar{a}(\rho + \mu + \gamma) \left(2 + \frac{\varphi}{\rho + \mu + \gamma} \right) \\ &= \varphi(\beta\bar{a} - \rho) + (\rho + \mu + \gamma)(\varphi + \mu - \beta) + \beta\bar{a}(2\rho + 2\mu + 2\gamma + \varphi) \\ &= 2\beta\bar{a}(\rho + \mu + \gamma + \varphi) - \beta(\rho + \mu + \gamma) + \mu(\rho + \mu + \gamma + \varphi) + \gamma\varphi. \end{aligned}$$

Using equation (2.8) for \bar{a} , we write y_2 in terms of the parameters only as

$$\begin{aligned} y_2 &= 2\beta(\rho + \mu + \gamma) - 2\mu(\rho + \mu + \gamma + \varphi) - 2\gamma\varphi - \beta(\rho + \mu + \gamma) \\ &\quad + \mu(\rho + \mu + \gamma + \varphi) + \gamma\varphi \\ &= \beta(\rho + \mu + \gamma) - \mu(\rho + \mu + \gamma + \varphi) - \gamma\varphi. \end{aligned}$$

B.3 Positive invariant region

We show that provided we always take our initial conditions to lie in D , the solution will always be in D . We do this by considering the direction field at the boundary, ∂D , which is the triangle in the ar -plane with vertices $(0, 0)$, $(1, 0)$ and $(0, 1)$. We want to show that the direction field at ∂D always enters D . This ensures that any trajectory starting in D remains in D .

The boundary will be considered as the union of six sets: each of the three vertices, and each of the three edges minus the vertices. Firstly we shall look at the direction field across the line $r = 0$ for $a \in (0, 1)$. To determine the direction field along this boundary line we consider equations (2.3) along $r = 0$. This gives

$$\begin{aligned} \dot{a} &= -\beta a^2 + (\beta - \varphi - \mu)a, \\ \dot{r} &= \varphi a. \end{aligned} \tag{B.3.7}$$

As $a > 0$ along the boundary, equation (B.3.7) determines that $\dot{r} > 0$ along the boundary line $r = 0$. This is sufficient for us to determine that the direction field

arrows at the boundary line always point into D . Similarly we now evaluate equations (2.3) along the boundary line $a = 0$ with $r \in (0, 1)$ to obtain

$$\begin{aligned}\dot{a} &= \rho r, \\ \dot{r} &= -(\rho + \mu + \gamma)r.\end{aligned}\tag{B.3.8}$$

As $r > 0$ we can conclude from equation (B.3.8) that $\dot{a} > 0$, hence all direction field arrows along this boundary line point into the region D .

The final boundary line is $a + r = 1$ for $(a, r) \in (0, 1)^2$. Along this line we can write equations (2.3) in terms of one variable by using $r = 1 - a$ which gives the equations

$$\begin{aligned}\dot{a} &= -(\rho + \mu + \varphi)a + \rho, \\ \dot{r} &= (\rho + \mu + \gamma + \varphi)a - (\rho + \mu + \gamma).\end{aligned}$$

To determine the direction that arrows cross the boundary line $a + r = 1$ we use the vector dot product. The vector dot product for two vectors \mathbf{x} and \mathbf{y} is

$$\mathbf{x} \cdot \mathbf{y} = |\mathbf{x}||\mathbf{y}| \cos \theta,\tag{B.3.9}$$

where θ is the angle between the two vectors. We consider the vector $(1, 1)^T$, which is orthogonal to the boundary line, and dot this with the vector $(\dot{a}, \dot{r})^T$. We find that

$$\begin{aligned}\begin{pmatrix} \dot{a} \\ \dot{r} \end{pmatrix} \cdot \begin{pmatrix} 1 \\ 1 \end{pmatrix} &= -(\rho + \mu + \varphi)a + \rho + (\rho + \mu + \gamma + \varphi)a - (\rho + \mu + \gamma) \\ &= -\mu - (1 - a)\gamma.\end{aligned}\tag{B.3.10}$$

As $(1 - a) > 0$, the right hand side of equation (B.3.10) is negative. By applying the vector dot product formula (B.3.9) we conclude that $\cos \theta < 0$ so $\theta \in (\pi/2, 3\pi/2)$. For these values of θ , the direction field always crosses the boundary line $a + r = 1$ in a direction which enters the region D .

We now consider the direction field at each of the vertices of the boundary triangle. No trajectories can pass through the point $(0, 0)$ as $(\dot{a}, \dot{r}) = (0, 0)$. At the

point $(1, 0)$,

$$\begin{aligned}\dot{a} &= -(\varphi + \mu), \\ \dot{r} &= \varphi.\end{aligned}$$

The equation of the line passing through $(0, 1)$ for which (\dot{a}, \dot{r}) is the direction vector is $r = -\varphi(1 - a)/(\varphi + \mu)$ which has a gradient less than that of the boundary line $a + r = 1$, hence trajectories passing through the boundary point $(1, 0)$ will always enter D . At the point $(0, 1)$,

$$\begin{aligned}\dot{a} &= \rho, \\ \dot{r} &= -(\rho + \mu + \gamma).\end{aligned}$$

The equation of the line passing through $(0, 1)$ corresponding to the direction vector (\dot{a}, \dot{r}) is $r = -(\rho + \mu + \gamma)a/\rho + 1$. As the magnitude of the gradient of this line is greater than that of the boundary line $a + r = 1$ we can conclude that all trajectories passing through the point $(0, 1)$ will always enter the feasible region.

B.4 Endemic equilibrium solution comparison

We show that if the inequality $R_0 > 1$ is satisfied then the inequality $F < 0$ must also be true. We begin by considering the inequality $F < 0$, which gives

$$0 > -\beta(\rho + \mu)(\rho + \mu + \gamma) + \mu(\rho + \mu)(\rho + \mu + \gamma) + \mu\varphi(\mu + \gamma) - \rho\varphi(\rho + \varphi).$$

This rearranges to

$$\beta(\rho + \mu)(\rho + \mu + \gamma) > \mu(\rho + \mu)(\rho + \mu + \gamma) + \mu\varphi(\mu + \gamma) - \rho\varphi(\rho + \varphi). \quad (\text{B.4.11})$$

We now look at the constraints on the parameter values which come from $R_0 > 1$.

This can be written as

$$\beta(\rho + \mu + \gamma) > \mu(\rho + \mu + \gamma + \varphi) + \gamma\varphi. \quad (\text{B.4.12})$$

By multiplying both sides of inequality (B.4.12) by $(\rho + \mu)$ we get

$$\beta(\rho + \mu)(\rho + \mu + \gamma) > \mu(\rho + \mu)(\rho + \mu + \gamma) + \mu\varphi(\mu + \gamma) + \rho\varphi(\mu + \gamma), \quad (\text{B.4.13})$$

which has the same left hand side as inequality (B.4.11). We now compare inequalities (B.4.11) and (B.4.13) and find that inequality (B.4.13) imposes the greatest lower bound on the expression $\beta(\rho + \mu)(\rho + \mu + \gamma)$. From this we conclude that if the parameter values satisfy $R_0 > 1$ then they will satisfy $F < 0$.

Appendix C

C.1 Exact solutions to $f_1(a) = 0$ and $f_1(a) = 0$

Following the method described by Murray (2003, appendix 2.3), let

$$x = \frac{D+6}{36D}, \quad y = \frac{\beta(D+9) - 54\rho}{108\beta D}, \quad z = -\frac{5}{6}.$$

Then, for $\rho < g_1(a_-^1)$, the exact solutions to $f_1(a) = 0$ are

$$a = 2x^{\frac{1}{2}} \sin \phi - z, \quad a = -2x^{\frac{1}{2}} \sin \left(\frac{\pi}{3} + \phi \right) - z, \quad a = 2x^{\frac{1}{2}} \sin \left(\frac{\pi}{3} - \phi \right) - z, \quad (\text{C.1.1})$$

for $\phi = \sin^{-1}[y/2x^{\frac{3}{2}}]/3$, $|\phi| \leq \pi/6$. For the model with varying conformity threshold frequency, the solutions to $f_2(a) = 0$ for $\rho < g_2(a_-^2)$ are given by equations (C.1.1) with

$$x = \frac{6 + D(2 - \eta)^2}{36D}, \quad y = \frac{\beta(9(2 - \eta) + D(2 - \eta)^3) - 54\rho}{108\beta D}, \quad z = -\frac{4 + \eta}{6}.$$

C.2 Justification of the linear reversion term γA for small γ

Consider the two functions

$$r_1 = \gamma a,$$

$$r_2 = \gamma as[1 + \hat{D}(2s - 1)(1 - s)],$$

representing reversion from type A back to type S. The function r_1 assumes no social influence, whereas r_2 assumes a conformist influence of the same form as c_1

(used in section 3.2) with conformity coefficient \hat{D} . The difference between these two functions can be calculated by subtracting r_2 from r_1 , resulting in

$$d_\gamma(a) = \gamma a^2(-2\hat{D}a^2 + 3\hat{D}a + 1 - \hat{D}).$$

The turning points of this function occur at $a = 0$ and

$$a = \frac{9}{16} \pm \frac{1}{16} \sqrt{17 + \frac{64}{\hat{D}}}.$$

By considering these points as $\hat{D} \rightarrow 0$ it can be determined that, for all values of \hat{D} , the function d_γ is strictly monotonically increasing on $(0, 1)$, therefore attains its maximum within $[0, 1]$ at $a = 1$. By direct calculation, $d_\gamma(1) = \gamma$ so the maximum error magnitude which can arise from using the linear function r_1 over the conformity function r_2 is γ . As stated in section 3.2 we assume γ to be very small, and much smaller than β , therefore using r_1 is appropriate owing to the small magnitude of the error.