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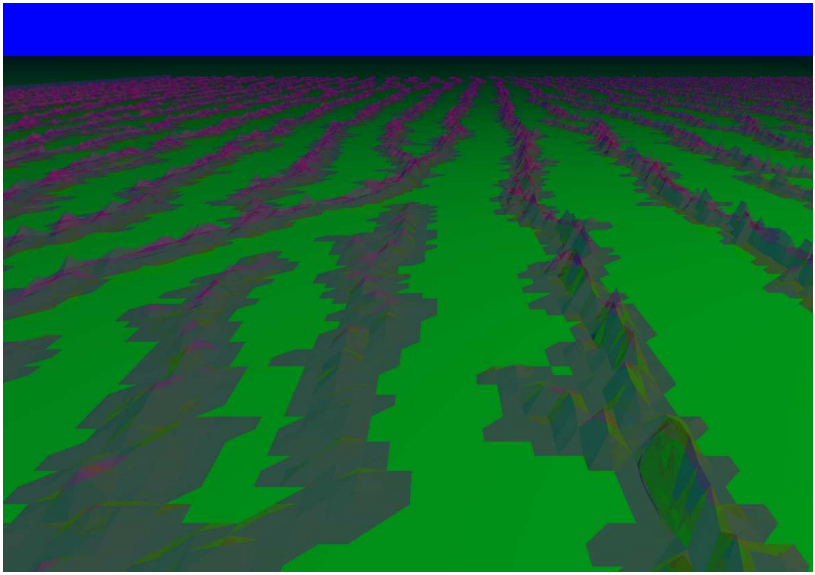

Simulations on evolutionary phenomena with ageing models

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Deutsche Zusammenfassung

Diese Arbeit beschreibt biologische und linguistische Systeme anhand von Methoden, die auch bei der Erklärung von Phänomenen in der statistischen Physik erfolgreich waren. Kritisches Verhalten wurde nicht nur in physikalischen Systemen gefunden, sondern auch in vielen nichtphysikalischen, wie z.B. in der Soziologie, der Biologie, der Geologie, und anderen. Eine adäquate Untersuchung und die Vorhersage des zukünftigen Verhaltens derartiger Systeme setzt in der Regel voraus, daß agenten-basierte (in biologischen Systemen individuen-basiert genannt) Simulationen durchgeführt werden. Die prinzipiellen mikroskopischen Eigenschaften der einzelnen Komponenten als auch deren Wechselwirkungen sind von fundamentaler Bedeutung, und können zu komplexem makroskopischem Verhalten führen. Die Anwendbarkeit analytischer Methoden macht Modelle auf dem Computer unersetzlich. In allen drei in dieser Arbeit behandelten Gebieten evolutionärer Systeme – Alterung, biologischer Artbildung und Konkurrenz von Sprachen – wird kritisches Verhalten gefunden.

Die Ursachen für unser Altern, als auch für das der Tiere, sind bis zur heutigen Zeit ziemlich unklar. Modelle zur Seneszenz beschäftigen sich mit der Fragestellung wie und wann Organismen sterben. Sowohl intrinsische Faktoren (z.B. wenn eine oder mehrere Körperfunktionen aufhören zu funktionieren) als auch externe Faktoren (Raubtiere, Hungertod oder andere feindliche Bedingungen) können für den Tod verantwortlich sein. Die menschliche Spezies war ziemlich erfolgreich bei der Begrenzung des durch externe Faktoren verursachten Tods. Bei Tieren kann man externe Einflüsse dadurch verringern, indem man sie im Zoo oder im Labor hält. Gemäß der Mutations–Akkumulations–Hypothese bedrohen umso mehr potentielle Erbkrankheiten unser Leben desto älter wir werden. Dabei werden Krankheiten, die vor der Geschlechtsreife auftreten, unterdrückt, da sie nicht weitergegeben werden können. Demgegenüber werden Erbkrankheiten, die erst im hohen Alter vorkommen, an die Nachkommen weitergegeben, und verhindern, daß sehr alte Individuen mit den jüngeren um natürliche Ressourcen konkurrieren. Experimente zeigen, daß Menschen, Tiere und auch viele simple Organismen wie Fliegen oder sogar Gegenstände wie z.B. Autos ähnliche Alterungsprozesse aufweisen (Vaupel, 1997). Die Sterblichkeitsrate

ist durch die Wahrscheinlichkeit definiert, in einem Altersintervall zu sterben. Sie steigt exponentiell mit dem Alter an, wie Gompertz schon 1825 herausfand (Gompertz, 1825). Bei vielen Spezies wurde beobachtet, daß sich die Sterblichkeitsrate bei sehr alten Individuen verlangsamt (bei Menschen wird dies noch kontrovers diskutiert). Dieses Phänomen heißt "oldest old"-Effekt (Vaupel et al., 1998). Es gibt die Theorie, daß dieser Effekt darauf zurückzuführen ist, daß die Wahrscheinlichkeit, wegen einer Erbkrankheit zu sterben, nicht abrupt ansteigt (Coe et al., 2002). Das Penna-Modell, welches auf der Mutations–Akkumulations–Hypothese aufbaut, war nicht nur erfolgreich in der Darstellung des Gompertz–Gesetzes (Penna, 1995), sondern auch bei der Erklärung von Phänomenen wie z.B. der katastrophischen Vergreisung des pazifischen Lachses (Penna et al., 1995), des Eva–Effekts (de Oliveira et al., 1997) und der Selbstorganisation der Menopause (Wechseljahre) (Moss de Oliveira et al., 1999a). Dieses Modell liefert eine realistische und stabile Alterstruktur von Populationen verschiedenster Organismen, und ist damit ein effizientes Werkzeug, um Computermodelle von biologischer und kultureller Evolution zu erstellen.

Der genetische Code aller lebenden Organismen ist aus vier Nukleotiden aufgebaut. Diese bilden das Alphabet unserer Gene. Eine direkte Übersetzung des genetischen Codes in das binäre System von Computern ist möglich. Zur Vereinfachung wird im Penna Modell die Aktivität einer Erbkrankheit, die vererbt wird oder durch schädliche Mutationen entsteht, durch nur ein Bit beschrieben. Das chronologische Genom jedes Individuums setzt sich aus einer Folge von Bits zusammen, von welchen jedes die Aktivität einer anderen Erbkrankheit beschreibt. Jedes Bit entspricht außerdem einem bestimmten Alter, ab dem die jeweilige schädliche Erbanlage aktiv wird, falls das Bit gesetzt ist. Sobald ein Individuum die Schwelle von T aktiven Krankheiten überschreitet, stirbt es. Schädliche Mutationen kommen in der Natur viel häufiger vor als nützliche. Daher werden nur erstere berücksichtigt. Damit die Population nicht ungehindert wachsen kann, muß noch ein zusätzlicher, populationsgrößenabhängiger Verhulst–Faktor eingeführt werden, oder die Geburtenrate während jeder Iteration entsprechend angepaßt werden. In der sexuellen Variante des Penna Modells besitzt jedes Individuum zwei Bitfolgen (Moss de Oliveira et al., 1999b). Sich sexuell reproduzierende Organismen verfügen über zwei Chromosomensätze, wobei je einer von einem Elternteil stammt. Ähnlich dem realen Prozeß der Meiose (Reifeteilung) bei der Produktion eines Nachkommen werden im sexuellen Penna Modell die Bitfolgen rekombiniert.

Das Penna Modell kann in seiner ursprünglichen Form sowohl in der asexuellen als auch in der sexuellen Variante den oben erläuterten "oldest old"–Effekt nicht reproduzieren. Dieses Plateau der Sterblichkeitsrate bei hohem Alter konnte jedoch durch eine einfache Erweiterung des asexuellen Penna Modells erklärt werden (Coe et al., 2002). In dieser Arbeit wird diese Erweiterung in die sexuelle Version implementiert, um deren Gültigkeit sowie deren Einfluß auf die anderen Populationseigenschaften zu untersuchen (Schwämmle and Moss de Oliveira, 2005). Ein Individuum stirbt nun

nicht exakt zu dem Zeitpunkt, an dem es zum ersten Mal T aktive Erbkrankheiten aufweist. Die Wahrscheinlichkeit, wegen einer bestimmten Anzahl aktiver schlechter Erbanlagen zu sterben, wird durch eine glatte Funktion beschrieben. Diese glatte Funktion ist eine Näherung der Treppenfunktion (z.B. eine Fermi-Funktion), die ihren Wert bei T von Null nach eins wechselt. Die Verwendung einer solchen Sterbewahrscheinlichkeit führt dazu, daß viele junge Individuen vor der Geschlechtsreife sterben, und somit die Geburtenrate stark erhöht werden muß, um das Verschwinden der gesamten Population zu vermeiden. Ein Plateau der Sterblichkeit kann aber trotz der Kompliziertheit der Messung, verursacht durch schwer zu kontrollierende chaotische Fluktuationen der Populationsgröße, beobachtet werden. Eine komfortablere Methode, den gesuchten Effekt zu untersuchen, besteht darin, die Sterbewahrscheinlichkeit für Individuen mit weniger als T aktiven Erbkrankheiten zu vernachlässigen. Dadurch muß die Geburtenrate nicht angepaßt werden. Die Ergebnisse zeigen, daß das Plateau sich unabhängig von der Art der Näherung der Treppenfunktion ausbildet, die Länge des Plateaus aber von der Stärke der Näherung abhängt. Ohne die Vernachlässigung der Sterbewahrscheinlichkeit für Individuen mit weniger als T aktiven Erbkrankheiten kann das chaotische Verhalten des Modells durch die automatische Anpassung der Geburtenrate vermieden werden. Die nun konstant gehaltene Anzahl der Individuen zeigt, daß eine glatte Sterbewahrscheinlichkeit starke Einflüsse auf andere Populationseigenschaften ausübt: Ein längeres Plateau durch eine weniger abrubt ansteigende Sterbewahrscheinlichkeit führt nicht nur zu einer höheren Geburtenrate, sondern auch zu einem Vorzeichenwechsel in der Krümmung der Altersverteilung in der Population. Es gibt in diesem Fall sehr wenige Individuen, die sehr alt werden.

Die Ergebnisse der verschiedenen Implementationen einer Sterbewahrscheinlichkeit, die nicht abrupt von Null nach eins ansteigt, legen dar, daß das Plateau der Sterblichkeitsrate sehr alter Individuen unabhängig von der jeweiligen Implementation reproduziert werden kann. Experimentelle Ergebnisse, die einen großen “oldest old”-Effekt für simple Organismen wie z.B. Fliegen beobachten, unterstützen die hier vorgestellten Simulationsresultate. Einfache Organismen haben sehr hohe Geburtenraten und ein Plateau kann nur beobachtet werden, wenn z.B. die Sterberate von mehr als einer Million Fliegen (Drosophilae) untersucht wird (Vaupel et al., 1998). Von uns Menschen gibt es umfangreiche Statistiken, die die der Fliegen um ein weites übertreffen. Es wurde bisher kein ausgeprägtes Plateau der Sterblichkeitsrate gefunden. Dies sollte auch für andere höher entwickelte Organismen zutreffen.

Der Prozeß, der zu der Bifurkation von einer biologischen Spezies in zwei oder mehr neue führt, heißt Speziation oder Artbildung und ist verantwortlich für die hohe Biodiversität auf unserem Planeten. Eine Spezies ist dadurch definiert, daß sie von allen anderen reproduktiv isoliert ist, d.h. deren Individuen können sich nur mit Individuen der gleichen Spezies fortpflanzen (Mayr, 1942). Bei der Beschreibung von biologischer Artbildung müssen im Vergleich zu Alterungsmodellen auch die Wechselwirkungen zwischen den Individuen und deren Anpassung an die Umgebung be-

rücksichtigt werden. Die Frage, wann und unter genau welchen Bedingungen Speziation vorkommt und wie sich eine Population während dieses Prozesses verhält, blieb bisher weitgehend unbeantwortet. Speziation kann nicht einfach anhand von Experimenten im Labor nachvollzogen werden, da die Zeitskala für solch einen Prozeß zu groß ist. Daten, die aus dokumentierten fossilen Funden oder dem Vergleich des genetischen Codes verschiedener Individuen gewonnen werden, liefern dagegen Anhaltspunkte, wie Speziation stattfinden könnte. Obwohl mittlerweile eine große Anzahl an Modellen zur Artbildung vorliegt, führten sie nicht zu einer allgemeinen Theorie dieses Prozesses. Der Einfluß von kritischem Verhalten in solchen Systemen wurde bisher vernachlässigt. Die meisten Modelle basieren auf Mean-field-Näherungen oder bauen auf sehr kleinen Populationen von weniger als 1000 Individuen auf. Es gibt zwei Hauptformen der Artbildung: allopatrische und sympatrische Speziation. Bei der allopatrischen Artbildung handelt es sich um die genetische Divergenz zweier Subpopulationen, die durch eine geographische Barriere getrennt sind. Natürliche Selektion, Mutation und Gendrift führen nach einer längeren Zeitspanne zu reproduktiver Isolation: auch bei späterem Kontakt können sich nun Individuen der verschiedenen Subpopulationen nicht mehr paaren, und formen somit zwei neue Spezies. Bei der sympatrischen Speziation divergiert eine Art, obwohl keine Populationen geographisch voneinander getrennt werden. Diese Form der Artbildung ist offensichtlich schwieriger. Nichtsdestoweniger muß kein geologisches Ereignis vorausgesetzt werden, das groß genug ist, eine Teilpopulation von der restlichen Spezies für lange Zeit zu isolieren. Es wurde schon immer diskutiert, ob Speziation unter sympatrischen Bedingungen überhaupt möglich sei. Neuere experimentelle Ergebnisse zeigen, daß ein solches Szenarium durchaus möglich sein sollte. Z.B. lebt eine große Anzahl von nah verwandten Buntbarscharten in den vulkanischen Seen in Westafrika. Diese Fischarten waren nie durch eine physikalische Barriere voneinander getrennt (Seehausen and van Alphen, 1999). Verschiedene Indizien existieren, die darauf hinweisen, daß diese Spezies nur in sympatrischen Bedingungen entstanden sein konnten. Zwei Hauptmechanismen werden für die Divergenz unter sympatrischen Bedingungen verantwortlich gemacht: Disruptive Selektion und sortengleiche Paarung (sexuelle Selektion). Bei ersterer handelt es sich um die Anpassung der Individuen an zwei verschiedene Nischen. Bei sexueller Selektion paaren sich z.B. Weibchen bevorzugt mit Männchen, die bestimmte Merkmale aufweisen.

Ein Modell, welches sympatrische Speziation untersucht, wird in dieser Arbeit auf dem Penna Modell aufgebaut (Luz-Burgoa et al., 2006). Eine zusätzliche Bitfolge stellt den Phänotyp dar, der gleichzeitig für die Spezialisierung auf bestimmte Nahrungsressourcen und für die Art der sortengleichen Paarung verantwortlich ist. Ein zusätzliches Bit entscheidet, ob ein weibliches Individuum sexuell selektiv ist oder nicht. Diese Bits werden von Generation zu Generation weitergegeben und dabei durch gelegentliche Mutationen verändert. Die Population wird entsprechend dem Phänotyp in drei Subpopulationen eingeteilt. Für die Individuen gelten je nach Subpopulation verschiedene Sterbewahrscheinlichkeiten. Der Kontrollparameter in die-

sem System ist das Ausmaß, mit welchem die mittlere Subpopulation mit den beiden anderen um natürliche Ressourcen konkurriert. Die äußeren Phänotypgruppen haben sich derart spezialisiert, daß sie nicht miteinander konkurrieren. Die Ergebnisse dieses Modells zeigen, daß die Teilung einer Spezies in zwei neue direkt von dem Kontrollparameter abhängt. Ein scharfer Phasenübergang im Nichtgleichgewicht trennt den Zustand des Polymorphismus innerhalb einer Spezies von dem zweier Spezies, die wegen fehlendem Genfluß reproduktiv voneinander isoliert sind. Der Ordnungsparameter, definiert durch die Häufigkeit sexuell selektiver Individuen in der Population, zeigt ähnliche Eigenschaften wie der eines physikalischen Systems im thermodynamischen Gleichgewicht. Die Fluktuationen des Ordnungsparameters divergieren am kritischen Punkt mit einem Potenzgesetz. Abbildung 1 illustriert diesen Übergang. Zusätzlich führen größere Werte eines anderen Parameters – die Anzahl der Männchen, aus denen ein passender Fortpflanzungspartner gewählt wird – zu einem schärferen Übergang. Diese Analogien helfen, den kritischen Charakter der Eigenschaften einer Population, die sich im Prozeß der Artbildung befindet, zu verstehen. Zu dem beschriebenen Modell konnte ein Mean–field–Modell entwickelt werden, das die makroskopischen Eigenschaften des Phasenübergangs im Computermodell qualitativ sowie quantitativ reproduziert (Schwämmle et al., 2005a). Die prinzipiellen Mechanismen des Computermodells wurden implementiert und das resultierende dreidimensionale Differentialgleichungssystem konnte auf ein eindimensionales vereinfacht werden, ohne die Ergebnisse stark zu verfälschen. Die Ergebnisse des Modells eignen sich, um diese an Statistiken von realen Populationen zu testen, und herauszufinden, ob sich eine Population im Prozeß der sympatrischen Speziation befindet.

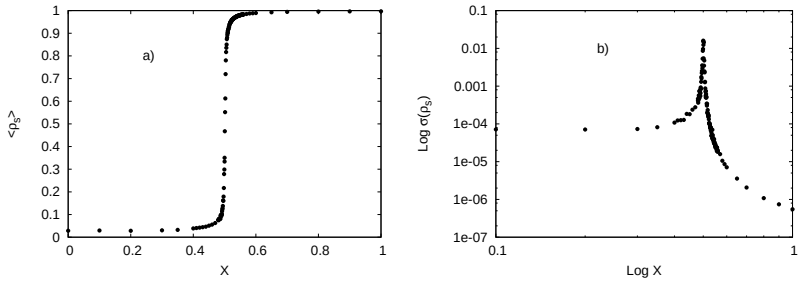


Abbildung 1: a) Der Mittelwert der Häufigkeit sexuell selektiver Individuen, $< \rho_s >$, in Abhängigkeit vom Kontrollparameter X, dem Ausmaß an Konkurrenz um natürliche Ressourcen. b) Logarithmischer Plot der Standardabweichung von $< \rho_s >$ versus X.

Die parapatrische Speziation kann als eine Mischform der allopatrischen und der sympatrischen betrachtet werden, wobei sich eine Hybridzone in der Region ausbil-

det, in welcher die Individuen zweier auf verschiedene Nischen angepaßter Subpopulationen einer Spezies in Kontakt stehen. Die Ursache der Anpassung an geographisch getrennte Gebiete ist normalerweise ein kontinuierlicher Nahrungsgradient, verschiedene Lebensbedingungen durch Höhenunterschiede oder andere äußere Einflüsse. Diese Art der Speziation wurde bisher wenig erforscht. In einem auf diese Art der Speziation konzentrierten und auf dem Penna Modell basierenden Modell spielen Fluktuationen und genetische Diversität als intrinische Eigenschaften des Modells eine wichtige Rolle (Schwämmle et al., 2005b). Ähnlich dem Modell zur sympatrischen Artbildung stellt in diesem Modell eine Bitfolge den Phänotyp eines Individuums dar. Die geographische Verteilung der Population wird durch ein quadratisches Gitter modelliert, worauf sich die Individuen bewegen können. Die disruptive Selektion ist hier abhängig von der geographischen Position und dem Phänotyp der Individuen und unterstützt deren Anpassung an geographisch voneinander getrennte Nischen. Diese Selektion wird durch eine Funktion, die die ökologischen Bedingungen beschreibt, ausgedrückt. Individuen mit hohen oder niedrigen Werten ihres phänotypischen Merkmals überleben besser auf den Rändern des Quadratgitters, während solche mit dazwischenliegenden Merkmalen überall auf dem Gitter selektiv unterdrückt werden. Diese Funktion wird erst nach einigen Tausend Simulationsschritten eingeschaltet. Dadurch wird die Eroberung eines neuen Gebiets oder eine abrupte Änderung der ökologischen Bedingungen simuliert. Diese kann eine Population zur Artbildung führen (Abbildung 2) oder nicht. Die Ergebnisse des Modells zeigen, daß parapatrische Speziation einem schnellen Prozeß entspricht, der von der Mutationsrate der Gene, die den Phänotyp kodieren, unabhängig ist. Bei starker disruptiver Selektion wird das System durch die Fluktuationen in der Phänotypverteilung in eine der beiden Nischen gezwungen. Einer der beiden extremen Werte des phänotypischen Merkmals dominiert dann auf dem ganzen Gitter. Bei schwacher Selektion bleibt das System in seinem ursprünglichen Zustand. Parapatrische Speziation tritt bei mittleren Werten mit bis zu 50% Wahrscheinlichkeit auf. Die Wahrscheinlichkeit, Artbildung zu erhalten, steigt mit der Größe des Gitters und der Anzahl der Individuen. Es konnte gezeigt werden, daß der Genfluß zwischen Individuen, die an verschiedene Nischen angepaßt sind, klein genug ist, um von Artbildung auszugehen. Die Hybriden sterben, bevor sie das Fortplanzungsalter erreichen. Das Modell kommt ohne sexuelle Selektion aus. Andere Modelle gingen dagegen davon aus, daß bei zufälliger Auswahl der Paarungspartner keine Speziation stattfinden kann.

Dokumentierte fossile Funde können uns näherungsweise mitteilen, wann und wie oft neue Arten entstanden und ausgestorben sind. Die aus diesen Daten gewonnenen Statistiken geben Hinweise auf die Funktionsweise eines makroevolutionären Systems. Die Theorie des Punktualismus sagt unter anderem aus, daß es ausreicht, verschiedene Spezies als Ganzes zu betrachten, ohne auf die mikroskopischen Eigenschaften ihrer Individuen Rücksicht zu nehmen (Gould and Eldredge, 1977). Diese Theorie ist noch heute umstritten, da die natürliche Selektion nach der Darwinistischen Theorie auf die Individuen und nicht auf eine ganze Spezies wirkt. Es wird ein

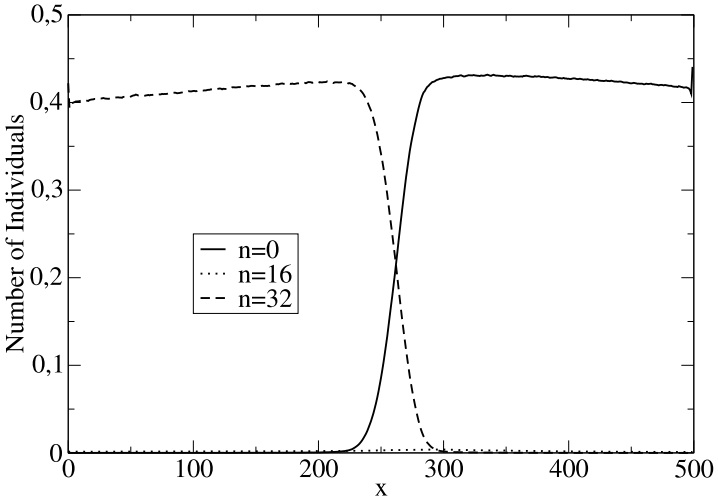


Abbildung 2: Häufigkeit der Individuen eines bestimmten Phänotyps versus geographische Position x auf dem Gitter. Die Werte sind über 10,000 Iterationen gemittelt.

simples, individuen–basiertes Modell konstruiert, welches nur die prinzipiellen Mechanismen von Speziationsereignissen beinhaltet: Mutationen und natürliche Auslese auf der Ebene der Individuen (Schwämmle and Brigatti, 2005). Dies ermöglicht die Untersuchung eines makroevolutionären Systems vieler Spezies, die sich in neue Spezies aufteilen oder aussterben, abhängig von deren Konkurrenz um Ressourcen. Andere Modelle der Makroevolution betrachten nur die Dynamik, die vom Aussterben von Spezies ausgelöst wird, und sind damit nicht imstande, zu untersuchen, ob Mikro- von Makroevolution getrennt werden kann. Hier wird anhand der Verwendung bekannter Mechanismen, die zur Artbildung führen, untersucht, ob so ein Bild ausreicht, die Makroevolution einer großen Anzahl von Spezies zu generieren. Die Ergebnisse werden dafür mit Statistiken aus dokumentierten fossilen Funden verglichen. Es wird die Funktion eines bekannten Modells (Dieckmann and Doebeli, 1999) verwendet, welches auf einer Wechselwirkung basiert, die von der aktuellen Frequenz der vorkommenden Phänotypen abhängt, und damit die Koexistenz von sich verzweigenden Spezies simulieren kann (Brigatti et al., 2005). Jedes Individuum besitzt eine ganzzahlige Variable, deren Wert die Anpassung seines Phänotyps an eine bestimmte ökologische Nische bestimmt. Die Konkurrenz um Nischen führt zu einer Dynamik,

die einen sich temporär verzweigenden Baum erzeugt. Die sich permanent ändernde Verteilung der Phänotypen der Population wird von Ansammlungen getrennter Populationen (Spezies) gebildet, die sich zu andereren für sie momentan günstigere Nischen bewegen, sich verzweigen oder aussterben (Bild 3). Sowohl die Mutationsrate des Phänotyps als auch der Parameter, der die Konkurrenz zwischen ähnlichen Phänotypen beschreibt, beeinflussen die Wahrscheinlichkeit einer Verzweigung (Spezies) und die Größe der einzelnen getrennten Spezies. Eine statistische Auswertung der Simulationsergebnisse legt dar, daß, wie auch in dokumentierten fossilen Funden beobachtet, die Verteilung der Lebenszeit der Arten über mehrere Dekaden einem Potenzgesetz mit dem Exponenten -2 folgt und dann exponentiell abfällt. Der exponentielle Abfall bei sehr langlebigen Spezies wurde auch mit einem anderen Modell zur Makroevolution gefunden (Chowdhury et al., 2003), kann aber wegen dem Fehlen von ausreichend realen statistischen Daten nicht bestätigt werden. Die Analyse der Zeitreihe der Anzahl an aussterbenden Arten stimmt nicht mit auf empirischen Daten stützenden Schätzungen überein. Es wurde kein intermittentes Verhalten gefunden, das charakteristisch für den Punktualismus wäre. Das Modell reproduziert erfolgreich die meisten statistischen Eigenschaften, die bei einem makroevolutionären System gefunden werden, kann aber z.B. keine Massensterben vorweisen. Eine zusätzliche Implementation verschiedener Ebenen von Nahrungsketten, von auf ganze Spezies wirkender natürlicher Auslese oder von externen Faktoren wie z.B. globalen geologischen Ereignissen könnte in diesem einfachen Modell Intermittenzen produzieren.

Organismen aller Arten – abgesehen von Prokaryoten (einfache Organismen wie z.B. Bakterien) – verändern ihre genetische Struktur nicht während ihres Lebens. Dagegen bekommen die Nachkommen eine neue genetische Struktur durch Mutationen und Kreuzung der Gene der Eltern. Das aktuelle Gebiet der Sprachenkonkurrenz, welches bis jetzt weitgehend unverstanden ist, zieht zunehmend Wissenschaftler vieler Gebiete an, und folgt einem anderen Konzept. Hier ändern die Individuen ihre Eigenschaften während ihrer Lebenszeit aufgrund von Lernprozessen, die von anderen Indviduen oder deren Umgebung gelenkt werden. Solch ein stark gekoppeltes System zeigt ein viel dynamischeres Verhalten als biologische Systeme. Trotzdem kann man auf die längere Erfahrung mit biologischen Systemen zurückgreifen, um das evolutionäre Verhalten eines multilingualen Systems zu verstehen. Schon Darwin zog damals Parallelen zwischen der Evolution von Spezies und der der Sprachen. Durch die Globalisierung und den zunehmenden Kontakt zwischen Personen verschiedenster Kulturen sterben mehr und mehr Sprachen aus. Sobald der letzte Sprecher einer Sprache stirbt, oder aufhört, diese zu benutzen, geht ein Kulturerbe mehr verloren. Sowohl das Aussterben als auch das Entstehen neuer Sprachen hat seine Analogie in dem oben beschriebenen Aussterben von Spezies, bzw. der biologischen Artbildung (Sutherland, 2003). Da in der heutigen Welt die Sprachkenntnis eines Menschen normalerweise keinen Einfluß auf dessen Überlebenswahrscheinlickeit hat, beinhalten Modelle zur Konkurrenz von Sprachen normalerweise nur die Interaktionen der Individuen, die

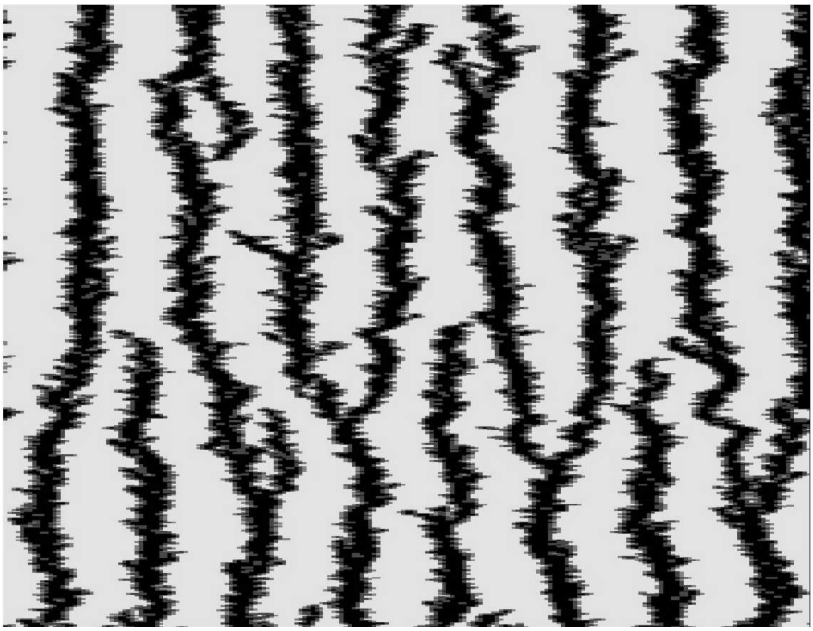


Abbildung 3: Zeitliche Entwicklung der Phänotypverteilung (von unten nach oben).

die Sprachenkompetenz verändern. Da die detaillierte Weise, mit welcher Sprecher verschiedener Sprachen miteinander interagieren, nicht bekannt ist, werden verschiedene Szenarien ausprobiert, um die bedeutendsten Mechanismen zu verstehen, die solch ein System beeinflussen.

Zwei Computer–Modelle werden in dieser Arbeit zur Untersuchung der Stabilität eines Systems mit mehreren Sprachen verwendet. Vom einfachsten Fall ausgehend, wird jede Sprache durch eine Einheit gekennzeichnet, ohne auf ihre komplexe innere Struktur einzugehen. Im ersten Modell lebt eine Population von Agenten (Individuen), die nach dem Schema des Penna Modells sterben und sich fortpflanzen, auf einem quadratischen Gitter (Schwämmle, 2005). Jeder Agent kann entweder Sprache 1, Sprache 2 oder beide sprechen, und aktualisiert seine Sprachkenntnis aufgrund einfacher Interaktionen mit seinen lokalen Nachbarn. Das Gitter wird so initialisiert, daß auf jeder Hälfte eine der beiden Sprachen vollständig dominiert. Die Stabilität des Systems wird in Abhängigkeit der Migrationsrate und der Stärke der Wechselwirkungen untersucht. Eine Grenze zwischen zwei Sprachen bleibt in diesem System desto länger in ihrem metastabilen Zustand je höher die Populationsdichte pro Gitterplatz ist. Dies ist ein Hinweis dafür, daß Sprachen in dicht bevölkerten Gebieten koexistieren können. Ein solches Szenarium kann man in vielen Städten beobachten.

Die Anzahl der Sprecher einer Sprache nimmt in der Kontaktzone exponentiell ab, während zweisprachige Personen mit gleicher Häufigkeit über das ganze Gitter verteilt sind (Abbildung 4). Ein weiteres Modell zur Sprachenkonkurrenz, das auch auf dem Penna Modell basiert, untersucht die Stabilität eines Systems von mehr als zwei Sprachen (Schwämmle, 2006). Die bestehende Alterstruktur wird ausgenutzt, um eine altersabhängige Lernroutine zu implementieren und deren Einfluß auf das kollektive Verhalten des Systems zu studieren. Ein Agent kann nur bis zu einem bestimmten Alter eine Sprache lernen oder vergessen. Bei der Geburt eines Agenten lernt dieser eine zufällig ausgewählte Sprache mit der Wahrscheinlichkeit m. Dieser der Mutationsrate in biologischen Systemen ähnliche Kontrollparameter m bestimmt, ob eine Sprache das System komplett dominiert, oder alle Sprachen gleichverteilt sind. Bei einem System mit mehr als zwei Sprachen kontrolliert dieser Parameter einen Phasenübergang erster Ordnung mit Hysterese. Dieser Phasenübergang ist vergleichbar mit dem zweier anderer Modelle zur Sprachenkonkurrenz (Stauffer and Schulze, 2005), bzw. Grammatik (Komarova, 2004), aber unabhängig von der Anzahl der Agenten. Die Verwendung anderer Werte der Parameter *maximales Lernalter* und *Anzahl der Wechselwirkungen pro Iteration* verursacht eine lineare Verschiebung des kritischen Punktes. Somit unterdrückt die Fähigkeit der Agenten, Sprachen bis zu einem höheren Alter zu lernen, die Möglichkeit einer Koexistenz mehrerer Sprachen. Das Modell liefert qualitativ die gleichen Resultate, wenn auf Alterstruktur und Geschlechtertrennung verzichtet wird und ist somit als Basis für viele Erweiterungen geeignet. Ein Ziel solcher Modelle ist es, die aktuelle Sprachenverteilung auf der Erde zu reproduzieren, und somit die zukünftige Entwicklung dieser Verteilung voraussagen zu können.

Die hier vorgestellte Dissertation untersucht verschiedene biologische und linguistische Modelle mit dem Ziel, mehr über die Auswirkungen mikroskopischer Eigenschaften auf das kollektive Verhalten derartiger Systeme zu verstehen. Kritisches Verhalten wurde in den meisten Fällen beobachtet und damit bewiesen, daß Fluktuationen in diesen Systemen nicht vernachlässigt werden können. Die Ähnlichkeit kritischen Verhaltens in physikalischen wie auch in den hier nicht–physikalischen Systemen unterstützt die Hypothese, daß beide Arten von Systemen durch ähnliche Methoden beschrieben werden können. Durch die Entwicklung geeigneter Computermodelle war es möglich, die Erklärung evolutionärer Phänomene wie Vergreisung, Artbildung und Sprachenkonkurrenz einen Schritt weiter in Richtung einer allgemeinen Theorie zu bringen. Die Resultate der vorgestellten Simulationen bieten eine Menge Daten, die mit experimentellen Ergebnissen verglichen wurden, bzw. noch verglichen werden können.

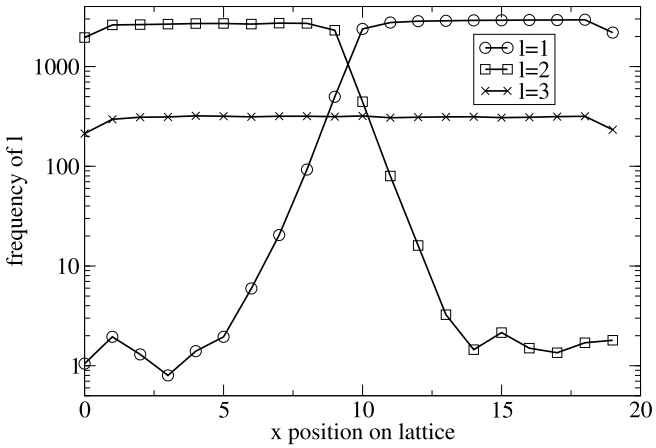
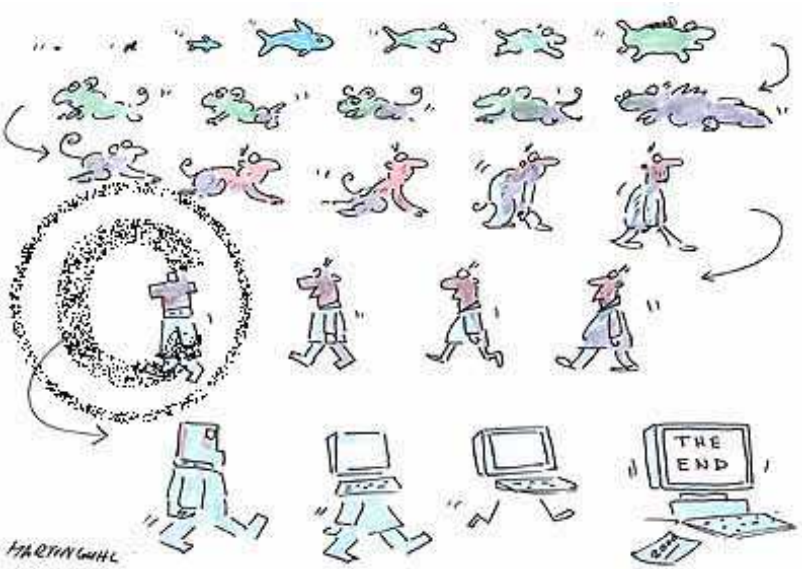


Abbildung 4: Verteilung einsprachiger ($l = 1$ und $l = 2$) und zweisprachiger ($l = 3$) Agenten auf einem Quadratgitter.

Chapter 1

Introduction



The comprehension of complexity in Nature still remains a great task for mankind: There is no consistent description of its behaviour. Fortunately, the research of many amazing phenomena gives more and more insight into the basic mechanisms of for instance evolutionary systems, and, as a consequence, complexity in fields like physics, biology, sociology and linguistics recently became more transparent. The richness in patterns and dynamics found in physical systems out of equilibrium results from

the microscopic interactions between the elements of the system. Mathematical, numerical and computational models are powerful tools to describe many of these features. The sometimes astonishing similarity of the behaviours between non-physical phenomena and the physical ones have lead physicists to extend their research to non-physical systems.

The idea of reducing the characteristics of highly complex systems to some basic features without losing the essential informations has been successful for the comprehension of not only physical systems. The experience in treating systems which consist of many elements that interact over different time scales as well as over wide distances, has opened many interdisciplinary fields to statistical physicists. Although animals and human beings behave in a much more complex way than for instance atoms or molecules, simple statistical models are able to reproduce many biological phenomena. The goal is to reproduce the collective behaviour of a large number of organisms: their individual properties are not of crucial importance. Due to the strongly increasing computer power along the last fifty years, computational models have gained more and more importance for research in many areas. The limitation of analytical models to describe accurately non-linear systems with critical behaviour makes computational models nearly irreplaceable.

The reasons for ageing in humans and in other organisms are still not understood completely (Stauffer et al., 2006). There are several theories among which mutation accumulation seems to be the most convincing one. Computer models of ageing are built on simple rules in order to understand how an age structure evolves by selection and mutation without the need to take into account the detailed interactions between the individuals and without focusing on external influences.

A step further, simulations on biological speciation concentrate on the interactions among the individuals and their adaptation to the surroundings. The formation of different species from a single one depends mainly on the mechanisms of assortative mating and disruptive selection by adaptation to different niches. Nevertheless, it remains unclear which mechanisms exactly drive a population to speciate, and how this occurs (Orr et al., 2004; Arnegard and Kondrashov, 2004). Computational models have the advantage of being able to predict critical behaviour in such scenarios. The comparison between the results given by such models and the real behaviour of physical systems can reveal strong similarities between the dynamics and structure of biological and physical systems, and thus help in their understanding.

Biological evolution is based on the concept that natural selection preserves the most fit individuals, that is, those that present the highest reproductive success. Individuals do not change their genetic structure during their lives but transmit them to their offspring. Mutations at birth change the structure of the offspring, and thus lead to diversity.

The new and until now not well understood field of language competition relies on

a somewhat different concept. Instead of relating a higher reproductive success to the better adapted individuals, the evolution of cultural elements is determined by the interactions between individuals capable to change their cultural characteristics during their lives. These even stronger inter-connected systems exhibit a faster temporal dynamics. However, it is possible to apply the large experience, gathered from thorough studies of biological evolution, to systems involving language competition, which recently began to draw the attention of many researchers (Nowak et al., 2001; Abrams and Strogatz, 2003; Wang and Minett, 2005).

Language competition can be considered as a part of sociophysics which began with the works of Schelling and Weidlich (Schelling, 1971; Weidlich, 2000) followed by Galam (reviewed in Galam (2004)). More recently many books about sociophysics have been written, as for instance (Helbing, 1995; Schweitzer, 2003; Arnopoulos, 2005; Stauffer et al., 2006).

Overview

The thesis is organised as follows:

The next section of this chapter discusses the relation between evolutionary phenomena and critical phenomena in statistical physics. It presents a variety of approaches which deal with the features of critical transients that are found in such systems. Section 1.2 briefly introduces the reader to the three main topics of the present work: biological ageing, biological speciation and language evolution. Section 1.3 presents a small overview of critical systems in general and phase transitions. Their relation to biological evolution is depicted in Section 1.4.

Chapter 2 is dedicated to biological ageing. First, the available experimental data and the theories that try to explain such phenomena are presented. The two following sections introduce the model of ageing used in this thesis, the so-called Penna model, one section presenting its asexual version and the following one its sexual version. The last section deals with the first work of the present author: The deceleration of mortality at very high ages is reproduced with a modified version of the Penna model, and the consequences of this "oldest old effect" are discussed.

Chapter 3 focuses on the topic of biological speciation. In the first section the theories that deal with this bifurcation of one species into two are introduced. The following section describes a work of the present author which shows that the process of sympatric speciation also exhibits a phase transition, like the ones observed in physical systems. In Section 3.3 a simple analytical model of the previously described computational model is derived. Simulations on parapatric speciation (section 3.4), where individuals are geographically distributed and can move on a lattice, show that fluctuations play a crucial role in this type of speciation where assortative mating is not

a necessary ingredient. The last section of Chapter 3 presents a model of macro-evolution. It considers only the fundamental mechanisms of the speciation process in order to produce an evolutionary tree. The results are compared with the experimental data obtained from the fossil record.

The experience one obtains in developing models to study biological evolution clarify the approaches to be followed in order to understand language evolution. The field of language competition is still in its very beginning, and is introduced in the first section of Chapter 4. The following section treats the problem of the stability of an interface between two languages. Section 4.2 analyses a model of competition of many languages where learning depends on the age of the individuals.

Finally, the conclusions and the outlook present the interpretation of the author's results and a possible continuation of his approach.

1.1 Evolutionary phenomena and Statistical Physics

The research on evolutionary dynamics concentrates on the problem of how natural selection drives a population to a better adaptation of its individuals, generation by generation. The search for efficient algorithms facing this problem and their applications opened the door to a wide field of computational tools in population dynamics, each of them encoding for a particular type of mechanism: *genetic algorithms* (Goldberg, 1989; Mitchell, 1996), *genetic programming* (Koza, 1992), *evolutionary strategies* (Bäck, 1996) and *evolutionary computation* (Jong, 1993) provide many ideas to create computer models in order to face a particular topic in biological evolution, as well as in other areas with similar characteristics. Evolution theory is built up on many evidences: fossils, genetic diversity, extinction of species, and so on. These observations can be explained by the picture of a world where species continuously evolve, and thus biological evolution is considered as a fact and not as a theory. Nevertheless, enormous difficulties exist to capture the exact behaviour of biological evolution. For instance, species change their shapes only very slowly, evolving over time periods much larger than experimentalists' life times. The large time scales of evolutionary processes enormously complicate the investigation of a system's behaviour under particular conditions, set by the researcher.

Experimental data are mostly obtained by extracting information from the fossil record as well as from the comparison of genetic sequence data of contemporary organisms (Li and Graur, 1991; Carporale, 1999). Experiments to confirm predictions suggested from the analysis of these data are in general not available, a situation similar to other scientific areas like astrophysics or geology.

The aim is to understand the vast diversity of the biological structure, its function

and its time evolution. A more thorough look reveals that in biological systems many components interact. The interplay of selection, genetic variation, morphological constraints, population dynamics, self–organising structures and environmental variations, to name some of the processes involved, creates the scenario we have when we look at our surroundings. Questions concerning how they interact and if some of these components result from some simpler internal dynamics need to be dealt with. Morphology and function of phenotypes, as well as cooperative interactions between individuals and between different species are now believed to be essential components of a biological evolving system.

Evolutionary systems are driven mainly by two mechanisms working on the individuals: selection and mutation. The first represents Nature's response to diversity, preserving the individuals that are better adapted to the environment. The second generates the diversity of the biological structures. A balance between these two mechanisms is needed to avoid extinction due to bad adaption of a before well adapted population, as a consequence of changes in the environmental conditions, or extinction due to the accumulation of too many mutations. The processes of mutation and selection, mediated by continuously evolving environments, allow the co-evolution of different species and prevent the static endpoint of a single perfectly adapted winner.

Complexity in Nature arises at the border of order and disorder: the interplay between a regular and a random process, between natural selection which alone would drive the system to a static state and mutations which alone would destroy adaptation.

Self–organisation is observed in a huge variety of non-equilibrium systems and until now there is no general theory to explain it. Haken (1977) and Nicolis and Prigogine (1977) gave strong arguments that biological evolution exhibits far–from–equilibrium self–organisation. Pattern formations in biological development (Meinhardt, 1982) present a famous example of self–organisation in biological systems.

Self–organisation is observed as well in many other simple systems. Simulational studies of critical phenomena in non-equilibrium systems, like the sand–pile's avalanches or the dispersion of forest fire, have shown that those systems exhibit power–law scaling. Neither a characteristic time scale nor a characteristic spatial scale are observed in critical systems. Additionally, these systems naturally evolve to this complex state: they are said to show *self–organised criticality* (Bak, 1997). Self–regulating and stable complexity is a feature which is also common for evolutionary dynamics, where biological structure and function are maintained. A simple model of self–organised criticality describes successfully the main features of the macro–evolution of many species (Bak and Sneppen, 1993).

Biological evolution expands over an impressively wide range of temporal and spatial scales, from time intervals of seconds and spatial extensions of nanometres at the molecular level to geological time scales and continents at the macro–evolutionary level. Theories have been developed to find a consistent description of the processes

on the different scales, trying to understand what is similar and what is different at distinct scales. Theoretical approaches borrow heavily from already developed theories in physics and mathematics: Phase transitions, critical phenomena and self–organisation are processes ubiquitous in statistical physics, as well as bifurcations, pattern formation and chaos as part of dynamical systems theory. Scientists of areas outside biology already investigated phenomena which turned out to be strongly related to biological evolution.

It has been learnt from disciplines different from biology that systems, where distinct elementary forces compete against each other, can create structural complexity as a consequence of the tension that arises from this competition. Several physical systems, related to equilibrium statistical mechanics, were among the first ones found to exhibit spontaneous formation of order. The Ising model is an amazingly simple model of a ferromagnet and considers only the spatial distribution of magnetic spins which interact locally. At low temperatures, an energetically favourable configuration of the spins dominates the structure of the magnet, creating a majority of spins pointing at the same direction. In contrast, thermal fluctuations break this local order at high temperatures, leading to a configuration of spins pointing at random directions. Peierls (1936) argued that there must exist a critical state at an intermediate temperature where order and disorder balance, and long–range correlations dominate the system. At this temperature the spins align forming clusters of all sizes and thus the system shows self–similarity. The complexity of physical systems exhibiting such a phase transition increases near the critical temperature. The competition between order and disorder completely controls the behaviour. Similar tensions are observed in evolutionary systems, as mentioned above.

The experience in treating critical systems has shown that research should also rely on simulations, not only to reproduce analytical results but also to understand regimes where analytical models fail to provide a realistic picture. Computational models promise deeper insights into evolutionary phenomena, by considering simplified interactions between the different biological agents.

Consequently, evolution is modelled as a system which contains stochasticity simulated through random variables, and non–linear deterministic processes. The individuals of a population are represented by a set of genotypes which diffuses through the space of all possible genotypes. The introduction of non–linear processes accounting for, for instance, competition and ecological fitness, constrains diffusion to be far from random. Genotype space can be defined to represent all configurations of gene sequences (Wright, 1932), of proteins (Maynard Smith, 1970) or, most appropriately, of polynucleotides (Eigen, 1971), the last being the elements which encode our DNA.

What are the effects of mechanisms which work on the microscopic scale, to the macro–evolutionary dynamics? The distribution of phenotypes seems to keep its structure for long time periods while at the microscopic scale the genotypes con-

stantly change and diffuse in genotype space. Evolutionary systems basically operate on two time scales: the macro–evolutionary slow scale and the fast scale of microscopic development. Besides the difficulties to consider a system which contains stochastic and deterministic components at extremely different time scales, one has to analyse the pattern formation, that occurs during development, in order to identify the most important features which are necessary to describe accurately the system.

Individual–based computational models need to consider the particular aspects of each individual: its position in genotype space, its phenotype, its geographic position and so on. In order to be able to obtain results within a reasonable computational time, simplifications play a crucial role to adapt the model to a certain problem without the loss of its validity. Fortunately, the experience in making simulations of critical systems tells that the behaviour of evolutionary systems can be simplified to some basic rules if one develops a simple architecture which accounts only for the fundamental processes involved.

Models which originally were invented to describe evolutionary dynamics, can be applied to many other problems that are not related to biological evolution. The design of adaptive and evolutionary algorithms used in, for instance, engineering (Holland, 1986; Bäck, 1996), tries to profit from strategies that Nature has successfully applied for millions of years.

Evolutionary phenomena can also be found in social systems. The field of sociophysics tries to better understand many different processes like opinion dynamics, self–organisation of hierarchies, traffic jams and social networks. Social systems have the property that their macroscopic behaviour is a consequence of a large number of microscopic agents interactions. Although social scientists look back to their own tradition in making computer models (Gilbert and Troitzsch, 1999), the methods of statistical physics have had great impact on the analysis and predictions of social collective behaviour.

Why do some people have much higher social positions than others? The formation of social hierarchies has been investigated ten years ago by Bonabeau and more recently by Stauffer (Bonabeau et al., 1995; Stauffer, 2005). In the Bonabeau model a phase transition was observed from social equality to inequality by, changing the population density on the lattice.

The research on opinion dynamics became popular during the last years. The question of how opinions can propagate through a population and why they persist is treated by means of computational and analytical models. There are several approaches to describe such a system. Ising–like models are a convenient tool to simulate the formation of opinions and are reviewed in Holyst et al. (2001). One of them is based on the so–called “social impact theory” (Nowak et al., 1990) while a random–field Ising model bases on real experiments (Galam, 1997). The voter models (Liggett, 1985), where the agents change their opinion to the majority one in their neighbourhood,

are another approach similar to Ising–type models. The restriction that the agents are usually located on a rigid topology can be avoided by the use of models with Brownian agents (Schweitzer, 2003). Providing a social hierarchy, majority decisions are given to a representative agent through several hierarchy levels (Galam, 1999). With this approach it is possible to explain why a dictatorship can remain in power even if it is not supported by the majority.

Three recent models are based on the concept of bounded confidence (Sznajd-Weron and Sznajd, 2000; Deffuant et al., 2001; Hegselmann and Krause, 2002). There, interactions occur only between agents with similar opinions. The range of confidence is defined through a parameter. Although these models differ in their basic rules, the results are rather similar. There exist three final states: consensus (one opinion dominates the system), polarisation (two opinions dominate) and fragmentation (the opinions are uniformly distributed). While in Deffuant et al. (2001) an interaction makes the opinions of the interacting agents to become closer, in Hegselmann and Krause (2002) an agent changes its opinion according to the mean value of the opinions of all agents which are inside the confidence boundary. The Sznajd model relies on a somewhat different concept: if two neighbours have the same opinion, they convince the other neighbours to share it as well. This approach became popular rather recently and the works applying this model are reviewed in Sznajd-Weron and J. Sznajd (2005). By putting the agents on a Barabási–Albert scale–free network, the election results of Brazil and India could be reproduced (Gonzalez et al., 2004). Even different models can reproduce the many properties of the Sznajd approach (Behera and Schweitzer, 2003; Galam, 2005) and thus support the idea that the detailed mechanisms in such models do not contribute significantly to reproduce the observed macroscopic behaviour. The stability of cultural diversity is investigated in the Axelrod model (Axelrod, 1997). Here, an agent can have several opinions (they are simulated by more than one variable). This model gave rise to other multi–opinion models (Sznajd-Weron and J. Sznajd, 2005; Jacobmeier, 2005; Fortunato et al., 2005) based on the works of Sznajd-Weron and Sznajd (2000), Deffuant et al. (2001) and Hegselmann and Krause (2002), respectively.

Social networks often cannot be represented by regular networks like one–dimensional chains or square lattices. The fact that, for instance, some of us have much more friends than others makes networks, where the agents have different numbers of neighbours (connections), more realistic. In general, living beings form complex networks. Social networks have been studied in detail during the last decade, leading to plenty of publications on this topic. The approach of modelling social connections through random graphs was not very successful, so that other strategies needed to be developed. The first attempt was the model of Watts and Strogatz (1998), in which the sites of a regular network are reconnected up to some degree. Other more realistic models of social networks are the ones of Barabasi and Albert (1999). These networks are scale–free and were able to reproduce not only the social connections

between human beings, but also the organisation of the Internet and the connections of the current power grid, among others (see the books of Barabasi (2002) and Albert and A.-L. (2002)).

Another field showing critical phenomena and that was inspired by sociophysics is the evolution of languages. Computational models developed in order to understand some linguistic phenomena are relatively new (Briscoe, 2002; Healey et al., 2002; Cangelosi and Parisi, 2002; Minett and Wang, 2005). Language evolution poses many open questions. Where on Earth did language have its beginnings and how did it evolve (Steels, 1997)? Language evolution is strongly connected to biological evolution. It is possible, for instance, to investigate how the language capability evolved. On the other side, language is strongly related to culture (Hurford, 1999). Models that intend to explain the many aspects of language evolution are supposed to rely on the concepts of biological evolution but also have to take into account the different dynamics of cultural progress.

The emergence of several of these aspects of language evolution has been analysed by means of computational models. The emergence of lexicon (Hurford, 1989; Ke et al., 2002; Smith et al., 2003) and grammar (Kirby, 2000; Cangelosi and Parisi, 2002; Gong and Wang, 2005) enables us to use language to exchange complex information. Linguistics discusses if language evolved as a consequence of informing other persons about some significant feature of the environment (Bickerton, 2002) or if social reasons (social interactions and coordinations) lead to this so important trait (Knight et al., 2000). Informations about the environment can be useful for the hearer. In contrast, the speaker in general does not have any advantage of communicating and thus this form of passing information can be seen as an altruistic behaviour of the speaker. Providing that genetic selection favours individuals that tend to learn from others, communication can emerge if the cost of being a speaker is lower than the benefit of the hearer (Simon, 1990). Simulations have been carried out to prove this theory (Mirolli and Parisi, 2005) and led to the conclusion that a social structure is necessary for the evolution of a language which is able to inform others about the environment.

Child development is a crucial factor in language evolution. It can be investigated how children learn to accurately communicate with their environment and how experience changes this behaviour. In the research on cognitive science also computational models play a role (Parisi, 1996). Learning and development of languages (language acquisition) has been studied including perceptual categorisation (Christiansen et al., 1998), syntax (Elman, 1993; Plunkett and Marchman, 1991; Rumelhart and McClelland, 1986), semantics and lexical growth (Plunkett and Sinha, 1992; Regier, 1996) and, more generally, the development of a shared communication system (Cangelosi and Parisi, 1998; Steels, 1998). For instance, it can be shown that many language acquisition phenomena depend on the statistical properties of the training set.

The social networks of language interaction can be analysed (Henrich and Boyd, 2002) and network theoretical approaches have been used (Girvan and Newman, 2002; Lieberman et al., 2005). Nettle (1999) and Gong and Wang (2005) investigated the impact of social structure on language change.

Language consists not only of words and grammar but also of sound. The phonetic representation of a language usually shows geographic diversity. A language evolves in time: its written and phonetic representation mutates rather fast making it sometimes almost impossible to read, for instance, literature written several centuries ago. Following Cavalli-Sforza and Feldman (1981), the invasion of a linguistic innovation behaves similar to the invasion of a virus. Sound changes invade the lexicon at different rates: word–to–word diffusion and speaker–to–speaker diffusion (Shen, 1997). Attempts to model such an invasion have been carried out by Wang et al. (2004) and Yang (2000) who predict that an innovation spreads through following generations following an S-shaped curve.

Theoretical approaches to understand language competition, leading to the actual risk of many languages to disappear, have less tradition (Nowak et al., 2001; Abrams and Strogatz, 2003; Patriarca and Leppänen, 2004; Mitchener and Nowak, 2004; Stauffer and Schulze, 2005; Kosmidis et al., 2005; Schwämmle, 2005), reviewed in Schulze and Stauffer (2005b). Chapter 4 concentrates on this process.

1.2 From ageing and speciation to language competition

This section provides a general overview of the different evolutionary phenomena treated in this thesis, and discusses their mutual relations. In all three fields - biological ageing, speciation, and language competition - critical behaviour can be found, and the corresponding systems can be called evolutionary systems.

Quantitative models of senescence or biological ageing deal with questions like how and when organisms die. Observations showed that many organisms age similarly: primitive ones like flies or even non-living objects like cars up to humans (Vaupel, 1997). Ageing models generally neglect death by non-genetic reasons, like infectious diseases, in order to understand the principal mechanisms which produce the behaviour observed in real systems. Experimental data is collected in industrialised countries for humans or for animals living in favourable conditions like in the zoo or in the laboratory. Several quantities can be measured: the probability to survive up to some age, the population age distribution, the mortality function, and others. The last one, the mortality function, defines the probability of an individual to die in an age interval. This quantity increases exponentially with age, as found by Gompertz

a long time ago (Gompertz, 1825). It was found that for some species the mortality deviates from this exponential growth for very old ages (Vaupel et al., 1998). This effect of the oldest old is still not well understood, but there exists the idea that in asexual populations it results from a relatively smooth death probability by deleterious mutations (Coe et al., 2002).

Several models, computational ones as well as analytical ones, have been developed in order to reproduce the empirical data of ageing. For an overview of the theories of biological ageing see Kirkwood (2005), Cebrat and Laszkiewicz (2005), Moss de Oliveira et al. (1999b), Stauffer et al. (2006), or Chapter 2 of this thesis. Still, no general theory of biological ageing exists. Additionally, some theories are not inconsistent with each other. That means that we can probably die due to several different reasons, none of the theories being predominant. A particular computational model, the Penna model (Penna, 1995), has been successful not only to describe the exponential increase of mortality with age but also to reproduce phenomena like, for instance, the catastrophic senescence of the pacific salmon (Penna et al., 1995), the Eve-effect (de Oliveira et al., 1997) and the self-organisation of menopause (Moss de Oliveira et al., 1999a), to mention only a few of the applications of the Penna model. This model relies on the theory of mutation accumulation to explain ageing. In the model an individual dies as soon as it accumulates a certain number of active inherited diseases. The genome of an individual is represented by a string of bits zeroes and ones. The book of Moss de Oliveira et al. (1999b) and the one of Stauffer et al. (2006) provide a detailed discussion of most of the extensions and applications of the Penna model. It yields a realistic and stable age structure and is thus a powerful tool to carry out simulations of biological and cultural evolution, as we intend to show.

Biological speciation corresponds to a process which does not occur very frequently in the time scale of a human's life. That is why speciation cannot be observed easily in laboratory experiments, and the researcher needs to investigate the fossil record, or compare the genetic structures of different species, in order to know how much distinct species are related to each other. However, the accumulation of a large number of speciation processes is responsible for the high diversity of our flora and fauna. The branching event that leads to the emergence of the humans and apes from a common ancestor represents an example. Modelling biological speciation needs a step further than modelling pure biological ageing: additionally to the intrinsic properties of an individual it is necessary to consider precisely how it interacts with the environment, in order to understand and simulate the selection mechanism.

There are two principal types of speciation: allopatric speciation and sympatric speciation. The first type deals with two populations of an ancestral species which become geographically separated. This happens for instance when a sub-population invades a new island. The absence of contact between this sub-population and the main one can lead to the situation where each one develops so many different genetic features, in order to adapt to its own environment, that finally the individuals of the differ-

ent populations cannot interbreed anymore. This type of speciation is very slow, but rather well understood and accepted by the scientific community (Mayr, 1942). The other type of speciation, the sympatric one, deals with the splitting of a population in the absence of any geographical barrier. Now the gene flux between all individuals needs to be in some way interrupted in order to provide a possible separation of one species into two new ones. Two main mechanisms are thought to be responsible for speciation in this scenario: assortative mating and disruptive selection. The former leads to sexual selection where mating only occurs if the two mating partners share some characteristic trait. The latter means that there exist two different niches which provide a better survival of this or that individual, depending on its phenotype. Parapatric speciation is in between the two previously described types of speciation, where the individuals live on a geographical gradient of, for instance, temperatures. In this case, the initial gene flux between arbitrary individuals is lower than in the case of sympatry.

Recently, literature that focuses on sympatric and parapatric speciation has been published. Experimental evidences are reviewed in the book of Coyne and Orr (2004), computational and analytical models discussed from a biological point of view can be found in Gavrilets (2004) and Dieckmann et al. (2004), while the physical aspects of the speciation process are presented in Stauffer et al. (2006). Models of speciation try to explain which are the main factors responsible for the occurrence of such an event, but a general theory is still not available. The role of statistical fluctuations and criticality in such scenarios, in most models neglected until now, needs to be focused on.

The fossil record enables us to obtain a rough estimate of the speciation and extinction events occurred in the past. Statistics have been made in order to understand how macro-evolution occurs (Solé et al., 1997). The theory of punctuated equilibrium says that it is enough to focus only on entire species and their interactions, instead of considering all individuals of the different species (Gould and Eldredge, 1977). The model of Bak and Sneppen (1993) gave reasonable results using this theory. Nevertheless, the idea of punctuated equilibrium is still controversial, regarding the fact that, according to Darwin, natural selection works on the individual level and not on the entire species. The shape of the phylogenetic tree, which depicts the speciation and extinction events, needs to be exactly reproduced, in order to establish which theory is the most appropriate one.

Although the evolution of language seems to be very different from the evolution of biological systems, Darwin had already revealed parallels between languages and species (Darwin, 1859; Mesoudi et al., 2004), and had used these analogies to explain his new theory of biological evolution. For instance, the phylogenetic tree, the tree of the evolution of species, has its analogy in the linguistic tree, which depicts the connections of the nowadays spoken languages to their ancestor ones. Sereno (1991) reviews the different analogies that can be made between the two types of

systems - as for instance the focal comparisons: *species/language, genes/culture, organism/concept* - and determines the restrictions, that have to be kept in mind, if techniques that were successfully used to describe a system of one of the two fields are applied to the other field.

Many attempts have been made to categorise today's languages and to find their ancestors by grouping them into language families (Greenberg, 1966, 1987; Campbell, 1997). The search for systematic patterns of word changes across several languages led to some agreement, but language classification is still strongly discussed. Critics say that languages have been evolving for too long to be able to trace back their origin (Ringe, 1993). In order to obtain better results, genetic analysis could offer a useful tool to find ancient family trees (Cavalli-Sforza, 1997), although genes and words do not follow the same time scale. More sophisticated methods, used successfully to obtain phylogenetic trees of biological species can be used to determine the relations between different languages (Searls, 2002).

The even more difficult task to find the geographic origin of European languages leads to the attempt to recreate our ancestor language *Proto–Indo–European* (Drews, 2000) and to trace back the genetics of modern Europeans (Gray and Atkinson, 2003). These methods gave some insight into the origins of European languages. While the first method supports the idea that Proto–Indo–European appeared first in the Kurgan homeland (located in the Ukraine), the second one suggests that language began together with the early days of farming in Anatolia.

From roughly 6000 languages spoken now, about 90% are thought to become extinct during the next century (Sutherland, 2003). Nevertheless, global diversity will be more or less maintained by the development of new urban hybrid forms. In cities language change is faster and thus their recent growth helps to form new dialects and mixtures of different languages. For instance, English is known to have hundreds of new forms around the world (Crystal, 2003). The principal use of English in science and trade has the consequence that influences from very different cultures will produce a change of its form. The use of alternative expressions generate its current diversity (Montgomery, 2000).

In the past, national identity was strongly related to the language somebody spoke. Nowadays, new forms of communication and globalisation – economical, cultural and political – lead to a new trend of bilingualism. People tend to speak more than one language in order to be able to participate in today's multilingual society. Especially English became very popular, being the most studied foreign language. The contact between speakers of different languages increases crucially and should be taken into account in new models of language competition.

In biological evolution, natural selection eliminates the poorly fit individuals, preserving those that present a larger reproductive success. However, natural selection does not change the genetic structure of the individuals during their lives. In order to

be able to consider cultural traits, such as languages, the concept of selection needs to be widened. The agents now can change their cultural traits during their life times, making these traits vulnerable to learning and forgetting processes. The dynamics of such evolutionary processes is much faster. Considering the special case of language competition, the influences of the language traits on the fitness of an individual generally can be neglected. Nevertheless, the large number of interactions makes the simulations rather time consuming. Detailed knowledge about the principal interactions between speakers of different languages is still missing. Different scenarios need to be tested to understand better the principal mechanisms driving such a system and some will be presented in Chapter 4.

1.3 On the edge of chaos

The description of the time evolution of a system can be obtained by many different approaches. Linear differential equations present the simplest way to calculate the future (and past) trajectory of a system. But, non–linear correlations between atoms, molecules, agents, individuals, ... need to be considered in order to obtain valuable results for the evolution of many systems.

Dissipative non-linear systems: These systems are often described by non-linear differential equations, whose set of initial conditions in phase space shows a contraction through time. In other words, the initial volume decreases during the evolution of the system towards an attracting set which is usually called an attractor. Attractors can be as simple as periodic orbits (for instance fixed points or limit cycles) or as complicated as strange attractors. Strange attractors are found in systems with chaotic behavior, and have typically a nonperiodic dynamics and a fractal dimension yielding very complicated shapes in phase space (see Figure 1.1).

We focus now on the speed with which the final state is reached, starting from an arbitrary initial point in phase space. In order to measure this speed, we compare the trajectories beginning at two slightly different initial conditions. In “regular” systems (the system evolves towards a periodic attractor) the distance between these trajectories in general decays exponentially. The trajectories, beginning at slightly different initial points, converge fast to the same final trajectory. Hence, if we wait sufficiently long, the system forgets its initial state and so presents short–term memory.

In chaotic systems, trajectories initially separated by a very small distance in phase space diverge exponentially in time converging to the final attractor in completely different ways and therefore showing different dynamics. In other words, after some time it is not possible any more to ascertain how close they were in the beginning. A famous example for this effect is the movement of a butterfly in Hong–Kong being

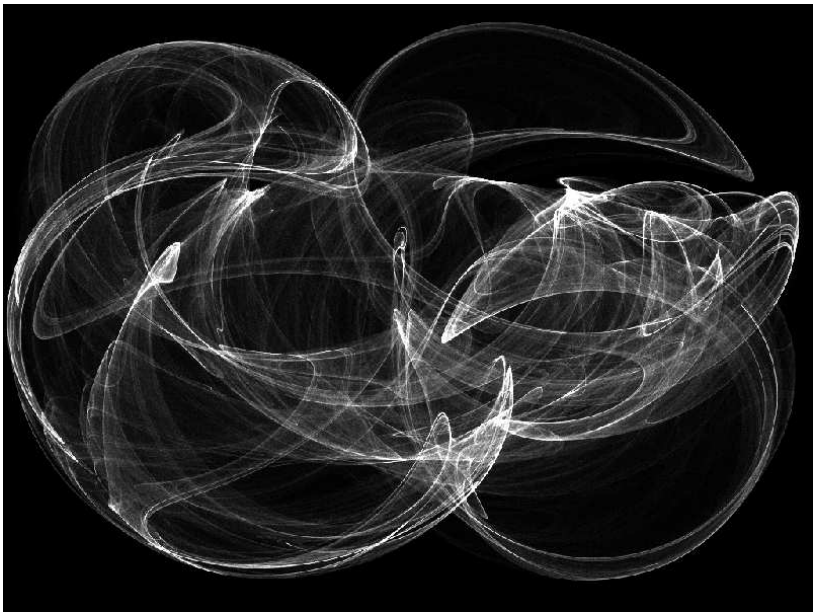


Figure 1.1: Shape of a strange attractor. Its fractal dimension can lead to “attractive” visualisations (from http://www3.sympatico.ca/olanglois/fractal/vstr.jpg).

able to influence the weather in such a way that it decides if a hurricane will emerge in the Caribbean Sea or not. As also observed for regular systems, chaotic ones can be characterised by a short memory. The system forgets its initial state after some time interval.

Critical systems: Now, we focus on the special case of systems on the edge of chaos. These are systems, for which the distance between initially very near trajectories varies polynomially in time. They can follow critical dynamics and exhibit a behaviour different from the one of regular and chaotic systems. Critical dynamics is associated with long–term memory in contrast to the short–term memory of regular and chaotic systems, presented above.

Consider a population where the offspring inherit their genomes from their parents. The genes pass from generation to generation and not from two or more generations before. Even so, the individuals exhibit a high diversity of genomes in order to be always sufficiently fit to guaranty the population’s survival. For the sake of simplicity, let us assume that the entire population reproduces at the same time. The system state is defined through the distribution of genes present in the population. At each

generation the state of the system evolves according to some rules we are not particularly interested in. Let the perturbation of the system be a mutation which gives to an individual a highly beneficial gene. Then the descendants of this individual benefit from their evolutionary advantage and the frequency of this gene in the population increases strongly. One single mutation generates a gene distribution with a large part of the population carrying now this particular gene, whereas without that mutation the entire population would lack it. This example demonstrates how evolutionary dynamics depends on the past. The pattern of evolution can strongly depend on events which happened a long time before. Long–term memory is found as well in many physical systems.

The Eve effect provides a nice example of such a "founder effect". Beside the name the Eve effect relies on genetic experiments and not on the Bible. We transfer our genes to our offspring after mixing them during conception. The genes of the mitochondria, the mitochondrial DNA (mt-DNA), in our cells play a particular role. The mitochondria are "factories" which are responsible to supply energy to our cells. The evolution of their genes is completely independent from the one of the nuclear DNA of our reproductive cells. The mt-DNA does not undergo reshuffling. The mitochondria that are originally present in every cell except of sperm cells. Sperm cells do not contain mitochondria, and thus the egg cell is the only gamete to contribute mitochondrial DNA to the offspring. Only mutations change the genetic material of the mitochondria. The mt-DNA provides a unique tool to do research on our lineage. An almost exact copy is transmitted from grandmothers to mothers and daughters, thus enabling us to trace back family branches. The comparison between human mt-DNAs shows that Europeans have seven different types of mt-DNA, coming from seven so–called ancient mothers. Every European is descendant of one of seven ancient mothers who lived about 30.000 years ago! If only six of them had contributed to our mitochondrial genes, our actual genetic distribution would be rather different.

Evolutionary systems evolve in trees, they never cover the entire space of possible configurations of the system. Bifurcations can drive the system to very different states, but still a vast number of possible non-visited configurations remains. Critical systems explore the space slowly, usually with a power law instead of an exponential dynamics, without occupying the entire number of possible configurations. Most of the possible states, for instance those corresponding to genes which were not favoured by selection are left unoccupied.

Criticality and power laws: Exponential and power law decays are very different. While the first one implies a finite life time, the second one corresponds to an infinite one. Real (physical, biological, ...) systems have finite sizes and thus finite life times. Nonlinearities in size appear due to correlations between the many components (atoms, molecules, individuals, agents) of a system. Even in systems with short–range interactions, long–range correlations can emerge out of a series of interactions

of neighbouring components, one influencing the other. The larger the correlation range, the longer the time a fluctuation needs to propagate along the system. The system size, that is the number of components, limits the correlations from exceeding certain maximum values. Real systems are of finite size and thus generate a cutoff on a certain life time. The quantities deviate from the power law and decay to zero at large times. For example, the magnitude of earthquakes is restricted to the size of the active region. Differential equations describe systems infinite in time and size. Nevertheless, the use of non–linear differential equations is appropriate under the constraint that they provide consistent results only for scales within the ones of the real system.

Power laws are observed in a vast number of phenomena and are not restricted to physical systems: phase transitions, earthquakes, avalanches in sand piles, extinction life times of biological species, neuronal activity, and so on. Critical behaviour is found in systems where the components interact in such a way that spatial long–range correlations are generated. Apparently non-related components can influence each other over large distances such that small changes can have great impact on the macroscopic behaviour. One single sand grain can cause an avalanche, or, the extinction of a small fish can lead to the extinction of an entire bio-system feeding on it.

Complexity: Complex systems can exhibit critical behaviour and phase transitions (Bak, 1997; Christensen and Moloney, 2005). The concept of complexity will be provided only shortly here, also because until now no exact definition has been accepted by the scientific community. For a much richer analysis see Parisi (1999).

A simple classification of a system presenting complexity is the following: the system is composed of many microscopic components. The interactions between these components evolve in time and generate the macroscopic behaviour of the entire system. Interactions play the crucial role, enabling each component to influence directly its neighbouring ones, and vice versa. Such a system of a large number of components generates long–range behaviour if the components, which are not directly connected, can interact through the intricate network of influences. Several processes act on one component and can generate conflicts. A complex system displays collective properties which look qualitatively different from a simple superposition of the elementary effects of its components.

This definition seems to be valid in most cases. Complex systems are observed in a vast set of subjects: evolution, speciation, genetic regulation, epidemics, neuroscience, earthquakes, forest fires, astrophysics, economic market, networks, adaptive learning and so on.

In order to understand the critical behaviour of complex systems, one can analyse

physical phase transitions. Phase transitions show criticality and have been studied for a century now. The analysis of a phase transition in a system in thermodynamic equilibrium facilitates the description of its behaviour due to the fact that the system does not evolve macroscopically. Nevertheless, the features can mostly be applied to complex systems out of equilibrium.

Physical phase transitions: Phase transitions have been thoroughly studied since the last century (Stanley, 1971). They show how materials change their phase with respect to physical quantities like pressure, temperature, volume, magnetic field, and so on. We distinguish not only between the phases solid, liquid and gas but also between different crystal structures, or, more general, different macroscopic behaviours of a material. Distinct phases generally differ by presenting different microscopic symmetries.

Critical behaviour is shared by many systems. Various physical quantities, from now on called order parameters, respond strongly to small increments of other quantities, called control parameters, providing that the system approaches some critical point. Critical behaviour is related to the correlation length divergence at the critical point. For this reason one cannot analyse the behaviour of a critical system by dividing it into small isolated pieces. The various components do not behave independently from each other. On the contrary, the macroscopic system must be treated as a whole.

In order to understand the results of experiments and observations in Nature, models must be able to reproduce these results and be built on plausible assumptions. As seen above, the application of analytical methods is restricted to some particular, very simple cases and thus does not provide the right tool even for macroscopically static systems. One has to perform simulations with agent–based computer models in order to obtain reasonable results for the dynamic evolution. The individual features of the agents are updated with respect to certain rules, corresponding to the problem with which the model deals. Agents interact with each other in such a way to produce long–range correlations. Additionally, the model can be extended to account for fluctuations: random numbers determine which action an agent carries out. Simulations run with different parameters and various initial conditions explore the possible final situations the model can produce.

The structure of computational agent–based models is usually rather simple in order to be able to study the system's behaviour for long time periods and for a large number of agents within tolerable computational times. Simulations performed over a small number of time steps or with a small number of agents cannot account for long–term memory or long–range correlations, respectively. We can not break the system into small pieces in time or space, for which an exact solution exists, with the purpose to obtain the behaviour of the whole by summing up the results of the pieces. The non–linear character makes size and time scalings non–trivial.

Simple computational models are usually criticised for discarding many microscopic features of the systems they try to describe, and thus are thought to be too simple to reproduce their real behaviour. However, the concept of universality allows us to neglect many mechanisms if we focus on systems with critical behaviour. The trick lies in the reduction of complicated systems to a simple model, which only accounts for the very general characteristics determining its universality class (for instance dimension of space and the order parameter). Unfortunately, these characteristics still are not very well understood, particularly for systems out of equilibrium, and thus the researcher will need to test to which level of simplicity the results of the model still remain reasonable.

Mean–field theories: We already realised that critical systems cannot be analysed by the superposition of reduced pieces. Mean–field models are those where the individual interactions between the components/agents are simplified to produce one general force equally acting on all of them. This strategy can, in general, give some insight into the qualitative behaviour of the system, helping to understand the problem itself by means of analytical methods. However, mean–field theories do not only predict quantitatively wrongly the values of the critical exponents but also other important relations. Even the qualitative behaviour can be entirely different. Although mean–field approaches are widely used in population dynamics, they can lead to wrong predictions when we have a system with fluctuations leading to long–range correlations. To find out if such an approach presents reliable results, one needs to check its results using an appropriate agent–based model. Hence, a more accurate approach to understand a critical system suggests that first simulations with a computational model should be performed, and afterwards a mean–field model should be developed to test if the simulation results can be reproduced. A mean–field theory which provides qualitatively correct results can be very useful, especially to explore a large parameter space and to capture different dynamics.

Fluctuations are completely neglected when we replace local interactions by the average of, for instance, all microscopic magnets. Small perturbations generate large responses of the system near the critical temperature and thus fluctuations propagate through large areas. The mean–field approach does not take into account long–range correlations.

Biological speciation can be seen as a bifurcation from one species to two coexisting ones. We could replace the temperature and the magnetisation by time and some mean genetic difference between the genomes of the individuals, respectively. In fact, Section 3.2 presents a model where the process of speciation exhibits a phase transition. Fortunately, a mean–field approach (Section 3.3) is rather successful to describe the main features of this transition, but is not able to reproduce many of the results of the computational model. Fluctuations normally cannot be neglected in population dynamics, and thus we should never trust too much in mean–field models.

1.4 Biological evolution

The idea that flora and fauna on Earth evolve in time, was proposed first by Jean–Baptiste Lamarck at the beginning of the 19th century (Lamarck, 1802). For him, the currently living species are not static, but change their structure and morphology, and interact in a complex framework. Species are continuously modified by very slow processes making them what they are now, differing from what they will be in the future. Lamarck stated two major "laws" in his book *Philosophie zoologique*:

1. The individuals of a species adapt themselves to their environment during their lives. A change in the environmental living conditions leads to another behaviour and finally to a transformation of the individual's structure, as for instance, the increase or decrease of the function of an organ after some generations.
2. These adaptations to the new environment are inherited.

Lamarckian evolution states that species react to changes of their environment by adapting themselves during the life time of the individuals.

Although this theory is wrong, it pronounced the dynamic slow character of evolution, at a time where religious dogmas claimed species to be static and to be created after some divine plan. Lamarck's ideas had no impact on the scientific community during his life and he died as a poor man. The next large step to a better theory was made by Darwin about half a century later (Darwin, 1859). His ideas emphasise the role that natural selection plays for biological evolution. Natural selection *is* the main force that drives evolution. Mayr (1991) summarised Darwin's five principal assumptions:

1. Evolution: as Lamarck already proposed, species change and evolve continuously. Their evolution is neither static nor cyclic.
2. Every group of organisms descends from a common ancestor. If we extrapolate this theory to all organisms, it states that all species, from animals to plants and microorganisms, share one common ancestor representing the origin of life.
3. Species proliferate: the great diversity of species on Earth can only be explained by branching. One species can bifurcate into two or more, for example by occupying different and geographically isolated environments.
4. Evolution is a gradual process and a population gradually changes.
5. For natural selection a higher reproductive success means higher fitness. The huge diversity among a single species is related to a relatively small number of perfectly adapted phenotypes at a given moment, which may change according to the environment.

Darwin's theory gave us a great insight of the way evolution works, although he had no idea how and which of the traits of an organism are transfered from generation to generation. Later, Mendel (1866) revealed the first mechanisms of genetic inheritance. However, his work began to be studied thoroughly only after the discovery of chromosomes with a microscope. Darwin's theories turned out to be basically right until now.

From now on, this section will focus on the framework of a general model of biological evolution, that is, we will present some important strategies used to simulate evolutionary phenomena. We will observe how individuals occupy the space of all possible living forms, or, to name it more concretely, the genetic space. How can the idea of genetic diversity be merged into a mathematical concept? Gregory Mendel, considered the founder of genetics, carried out experiments with sexually reproducing plants. He found out that inherited traits do not mix: a gene, corresponding to some particular trait of its carrier, is either inherited from at least one of the parents or not. An organism owns a gene or lacks it, genes cannot be subdivided into half genes or other even smaller fractions. The elements of Nature are discrete. We know that genes make part of the chromosomes and correspond there to a particular DNA strand. A trait can be the result of the action of more than one gene and can thus exhibit more variety depending on the number of genes and the way they contribute to that trait.

A genome is composed of DNA strands floating in the cell plasma (for procaryotes or, in other words, bacteria), or enormous strings of DNA which combine into chromosomes in the cell nucleus (eucaryotes, as, for example, animals and plants). The DNA is the code that determines the sequence of amino acids that make up the proteins, or that regulates their production. This means that it controls, for instance, how an organism develops or how it can defend itself against diseases. Each cell of an organism contains this information, encoded in an alphabet of four characters: the four nucleotides, adenine (A), thymine (T), cytosine (C), and guanine (G). Hence, each gene is identified by some particular sequence of nucleotides: AATTCACGATCGATCGTAG.... In order to construct an evolutionary model, the data of the DNA sequences can be translated simply from its quaternary code to the binary one of computers. A pair of bits 00, 01, 10 or 11, for instance, may correspond to nucleotides A, T, C or G, respectively. This data transformation assigns the genetic code into computer bits without loss of information and opens the way to perform computer simulations with real DNA data. The genome of an organism corresponds now to an enormous string of computer bits. Since the maximum length of the genome size in living beings is about 10^{10} nucleotides, the number of possible different configurations of the corresponding bit–string of maximally $2 \cdot 10^{10}$ bits is about $10^{6000000000}$, a number much larger than the roughly estimated number of atoms in our universe, 10^{79}. The space of possible genomes is enormous and spans over all possible configurations of bit–strings, having themselves variable lengths. Each organism, no matter

if it is a bacterium, a plant or an animal, corresponds to one particular bit–string and thus occupies one particular point in this genetic space.

Conveniently, each bit of the genome bit–string determines a new dimension. Hence, genetic space is finite and consists of two discrete states per dimension (bit set to one and bit set to zero). In general, the genome is not modelled by considering each nucleotide. Instead, each position of the bit–string corresponds to a particular gene, which has here only the two states zero and one. With this definition, the genetic distance between two individuals, or, in other words, the Hamming distance between two genetic configurations, can be calculated easily by comparing the bits on each position of the two strings. The Hamming distance is the number of all differing positions along the two compared bit–strings divided by the length of the bit–strings. Or in other terms: the number of bits which have to be flipped to convert one bit–string/configuration into the other, divided by the length. Of course, there are other, more sophisticated measurements of the genetic distance. However, the Hamming distance provides a simple and useful tool to characterise how distinct the genomes of two individuals are. Figure 1.2 illustrates the calculation of the Hamming distance. In the case of nucleotides, the Hamming distance is calculated by comparing pairs of bits.

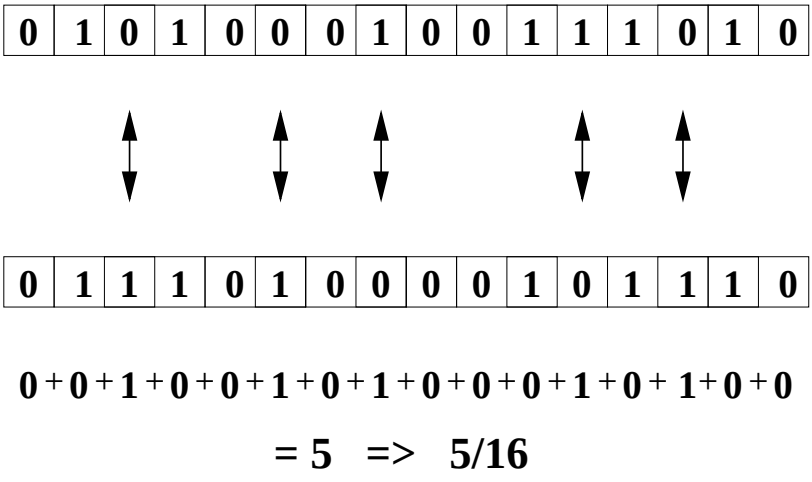


Figure 1.2: The genetic distance between two individuals is calculated by counting the bits by which the two strings differ. The result is divided by the genome length (here 16).

Almost all higher developed animals and plants reproduce sexually. In this case, each genome consists of a pair of homologous chromosomes and the species is called diploid (opposite to haploid for asexually reproducing species). For a more detailed

view on sexuality see Chapter 2. The corresponding genetic representation is made by assigning two bit–strings to each individual. These two strings are read in parallel. For instance, in Figure 1.3 the genome of the first individual consists of the strings A and B, and the one of the second individual consists of the strings a and b. The genetic distance between two genomes needs to be redefined due to the two possible comparisons: either A with a and B with b or A with b and B with a. Here we take the smallest value, corresponding to the smallest total number of switched bits, and divide it by the length of the entire genome, given by the length of both bit–strings of the diploid individual. Figure 1.3 illustrates the calculation of the Hamming distance.

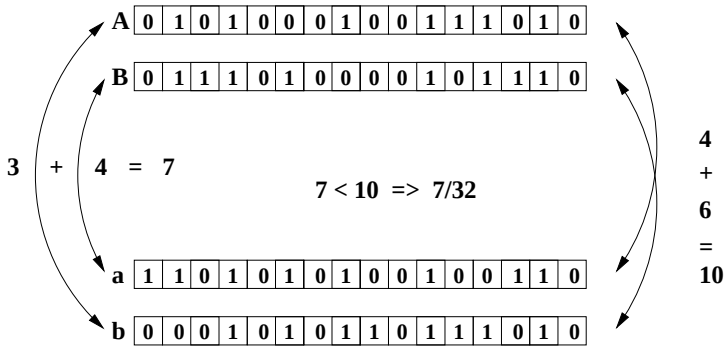


Figure 1.3: Hamming distance for two sexually reproducing individuals. By comparing A with a and B with b, a total of 10 bits differ. The alternative comparison of chromosomes A with b and B with a gives a difference of 7 bits. The smallest result is divided by the bit-string length and so the Hamming distance between individuals AB and ab is $7/32$.

After developing the concept of a discrete genetic space by defining the metric and the distances between different genomes, we focus now on the time evolution. Which kind of mechanism drives the system to genetic diversity? The genome of an organism does not change during its life but individuals of all species can reproduce. Chapter 2 presents the mechanisms of asexual and sexual reproduction. The present section discusses only how the genomes are modified by the process of reproduction. Most asexually reproducing organisms generate their offspring by simply making a copy of their genomes (cloning). Since the number of DNA compounds to be copied is huge, failures of this copying process occur frequently. These failures are called mutations and correspond to the situation where bits turns from zero to one or vice versa. Mutations occur randomly and can be simulated on the computer by the usage of random numbers. They are the first source of diversity in Nature. Chance and necessity control, by natural selection, the biological evolution (Monod, 1973).

The genome of an offspring of a sexually reproducing species is modelled by attributing a pair of bit–strings to each one, each of the pairs inherited from one of the parents. The two strings of the father are crossed at a random position and recombined into a new pair of bit–strings. One of the two new strings suffers mutations and will represent one of the offsprings' chromosomes. The same process is carried out with the genome of the mother. Hence, the genome of the offspring is a mix of the parents' ones, which provides a second source of genetic diversity.

A general model of biological evolution needs to consider all organisms living on Earth. Each one occupies a point in the genetic space which corresponds to its genome. The idea is to obtain a genetic distribution of all organisms that live at the time we focus on. On the smallest scale, isolated individuals are sparsely distributed in genetic space. The fact that even the small genomes of simple bacteria suffer several mutations, mostly without any effect on the phenotype, leads to the situation that the genome of the offspring always differs from the one of its parent. Although the genomes of sexually reproducing organisms are mixed more than the ones of asexually reproducing organisms, as explained above, the genome of the offspring in general is still placed near the parents' ones in genetic space.

After some time, many previously occupied points of the genetic space become free because the individuals with the corresponding genome die. Others become occupied due to the birth of new individuals. Even so, most of the points remain unoccupied. A single individual produces offspring according to a branching process distributing its children around its location. Then the offspring generates grandchildren and so on. Thus each individual can be the origin of a large branching process. Nevertheless, most lineages become extinct because the individuals of that branch present a low reproductive success. That is, the extinction of a lineage or equivalently, the death of its individuals, occurs due to natural selection, which is a very complicated process, distinct for each individual of each species at each time. The strategies described here do not consider any particular implementation of the selective process but consider only the fact that some individuals are able to reproduce more than the others. The important point is that, as a consequence, only a few, or if we wait sufficiently long, only one in the past living individual originated an entire population.

A negative zoom (a look on a larger part of genetic space) shows that a population of individuals, which distributes in genetic space as a sparsely filled cloud, is surrounded by a vast, empty area. Thus this population defines a species. The genetic difference between the individuals of different species is so large that they are not able to carry out any kind of reproduction, or they do not produce viable offspring. Reproductive isolation defines different species according to the *biological species concept* (Mayr, 1942). What about asexually reproducing populations like bacteria? Genetic separation of different populations also is valid for these organisms due to the fact that asexually reproducing organisms adapt to niches. Adaptation means that there is some set of genomes that contributes with a high fitness to the individuals

populating this niche. Slightly different genomes which lead to slightly less fit phenotypes are selected against. High reproduction rates and small time scales, common for procaryotes, lead to the situation that all lineages with a less fit phenotype vanish fast. But another mechanism crucially complicates the definition of bacterial species: even completely different bacteria can exchange DNA strands during their life, a process called lateral or horizontal transfer (Ochman et al., 2000). Hence, the genomes of lineages which have been separated a long time ago can make part of a new genome. This process can be very helpful for bacteria to survive an abruptly changing environment. For instance, a bacterium of tuberculosis gathers the gene of another bacteria that encodes for the resistance to antibiotics. The now resistant Mycobacterium tuberculosis has a much higher fitness and thus will generate, unfortunately, a long lineage.

A further negative zoom exhibits that several clouds of populations, each one defining a different species, are separated from each other by large empty spaces. According to deaths and births within a species some points in each cloud become occupied and others free, but the centre of mass seems to stay at rest during the time interval of several generations. The mean genetic composition of a species remains static. Larger observation times reveal that the species clouds move slowly through the genetic space, some bifurcate in order to give birth to new species, and others become extinct. Nevertheless, the clouds generally never collide due to the inhomogeneous and extremely sparse occupation of space. Contrarily, tracing back in time, different clouds begin to move towards each other and merge to form the originating species. This process repeats while we go on returning to the past until only one cloud remains in the entire genetic space. This cloud shrinks until only one organism, the ancestor of all living species, remains. Each pair of nowadays living individuals has a common ancestor, as well as each pair of species has its common ancestor species. On the other side, every species (if it does not become extinct) has the power to generate many new species in the future, a process similar to the case of branching of the descendants of an individual.

What leads to the movement of the "centre of mass" of a species? If we set natural selection to be static, the cloud would fluctuate only slightly around its centre of mass without any concrete direction. But natural selection is a dynamic process driven by changing environmental conditions and the interactions with other species, and so controls the direction of the movement of a species in the genetic space in search for genetic configurations that encode for phenotypes with higher fitness. Mutants are not, in general, fitter than their parents. Only a very small number of individuals has a higher fitness due to rarely occurring beneficial mutations. These offspring reproduce more successfully and thus the centre of mass moves towards the coordinates of the genomes with higher fitness. Every time, when a beneficial mutation is tossed, the genetic structure of a species slightly transforms. Hence, natural selection controls the direction of movement whereas mutations control its speed by chance.

The amazing similarity between the behaviour of individuals and the one of entire species leads to the question of what we will observe if we zoom to an even larger region in genetic space. A further negative zoom shrinks the species to small, sparsely distributed points. Nevertheless, again a cloud of points is surrounded by a vast region of empty space separating this group from other groups of species, or, to be more exactly, of other genera. The negative zooms can be continued to some higher levels where each time a new, larger group of species (genus, family, ...) is merged together, separated from others by a vast, empty space.

According to this model of biological evolution, it seems that mathematical methods, successfully applied for critical physical systems of equilibrium, could be suitable to analyse evolutionary systems as well. We only have to find out how to renormalise natural selection and mutation to higher levels. Unfortunately, the system evolves in time, and not only length scales need to be controlled but also different time scales. Physicists were able to construct a complete theory of equilibrium critical phenomena which assigns each system to make part of a universality class. Recently, especially computer simulations indicated that there should exist a similar concept of universality for systems out of equilibrium (Odor, 2000; Hinrichsen, 2004). In this thesis, several critical phenomena are studied by means of computational models: In Chapter 3 critical phenomena in the biological speciation process, similar to physical systems are analysed, whereas Chapter 4 presents systems of language competition, which exhibit evolutionary dynamics.

Section 1.3 demonstrated that length and time scales are directly linked to each other in critical systems. Providing two individuals of the same species, the average time one needs to go back in time to find their first common ancestor depends on the size of the species. More individuals correspond to more branching processes and thus the average time is longer. Correspondingly, the average time to trace back the first common ancestor species of two species of the same genus is much longer. And the first common ancestor genus of two genera lived even a much longer time ago. Hence, if we look at a larger spatial scale, a larger time scale should be considered. The here described model of biological evolution exhibits linked time and length scales, long–term memory and self–similarity, and thus should be declared a critical system.

Natural selection acts on the phenotypic features of an individual. The phenotype is partly determined by its genome, and defines an individual's response to its environment and its genetic design, accounting for its resistance to infectious diseases, its capacity to escape predators, ... Additionally, it expresses genetic defects that generate for instance life–threatening genetic diseases. In order to be able to perform computer simulations of biological evolution, the phenotype needs to be calculated from an individual's genome. Thus a function needs to be used, which translates the genome into phenotypic features. The probability to die and the reproductive success are determined for each individual by its phenotype and/or the external influences

exerted on it, like interactions with other individuals or the environment. Death and reproduction can depend on ageing, competition for resources, assortative mating, and many other features. In Chapter 3 phenotypic traits are encoded by particular bit–strings of the genome. Cultural traits, also implemented as bit–strings, provide another extension for a computational model. These traits are not inherited by the offspring from their parents. Children learn, for instance new languages, by interacting with the environment, like going to school, or living in a neighbourhood where another language is dominant (see Chapter 4).

Individual–based computer simulations on critical phenomena need to treat large populations and need to consider runs over many time steps. Power laws are measured over decades of time and size and so extensive data is required. The implementation of genomes, phenotypes and cultural traits by bit–strings makes it possible to calculate the death probability and the reproduction rate of an individual by the use of fast bit–operators. Simulations become fast with such an approach and do not require too much memory. As already shown in Section 1.3, the results for a system with critical behaviour depend strongly on the past and thus as well on the initial conditions. Therefore, one must run the same program with the same parameter set several times with different initialisations in order to obtain appropriate statistics for mean values and fluctuations by averaging over different runs. de Oliveira (2002) discusses some advantages of computer simulations over analytical approaches considering evolutionary systems. Two major reasons capture the importance of the computer approach: first, the absence of a general "Darwin equation" implicates difficulties to describe evolutionary systems appropriately. Second, differential equations rely on mean–field approximations and therefore neglect fluctuations.

Mean–field approximations are very common among scientists who treat evolutionary phenomena. In a very popular approach the genetic information of a population is described by the frequencies of genes/alleles carried by the individuals (see, for instance, Redfield (1994) and Kondrashov and Kondrashov (1999)). This is a comfortable way to avoid storing every individual's genome in the computer's memory. The frequency of a gene denotes the number of individuals which currently share this gene and describes how often the gene is expressed in a population. By replacing the many individuals by their gene frequency, one is taking only an "average individual" as representative of the whole population, thus neglecting fluctuations which occur within the reproduction of different individuals of the same generation. Hence, this mean–field approach should be treated very carefully because it can provide wrong results. The existence of phase transitions, bifurcations and other critical phenomena could be predicted not only quantitatively, but also qualitatively wrong.

Chapter 2

The Penna model

Figure 2.1: Benjamin Gompertz (1779-1865).

The Penna model (Penna, 1995) for biological ageing is a simple individual–based computational model capable to reproduce the Gompertz–law of mortality for animals as well as for humans, and has been applied successfully to study many evolutionary phenomena (Moss de Oliveira et al., 1999b; Stauffer et al., 2006). In this chapter the main features of this model will be described. Extensions of the model like the sexual version or the addition of a phenotypic trait are crucial ingredients to treat speciation and language competition. The chapter is organised as follows: first the concept of biological ageing is provided by presenting experiments and theories on this topic. The next section introduces the asexual version of the Penna model and

its application. After that, the sexual version and some of its extensions are presented. Section 2.4 discusses the present author's simulations to obtain a mortality plateau, also known as the oldest old effect.

2.1 Biological Ageing

One of mankind's oldest dream, the discovery of a "fountain of eternal youth", unfortunately seems will never be reached, at least according to the experimental results and empirical theories we will show in this chapter. When we become older, parts of our body begin to ache and our efficiency, for instance in sports, decreases. The higher the ages we reach are, the higher is the probability that we die. Obviously, humans share this property with all sexual species. Given the probability $\mu(a)da$ to die in the time interval da after reaching the age a, it is possible to define *ageing* as an increase of this probability. In the following μ will be called the mortality function.

Death can occur due to intrinsic factors, when one or more of the bodily functions fail to work properly or due to external reasons like predation, starvation and many other adverse environmental conditions. Humans have already succeeded in decreasing enormously their death rates due to external reasons, while to observe ageing in animals it is necessary to keep them in zoos or laboratories, free from all adverse conditions. In this case it is amazing how long some animals can survive without being threatened by other ones. Fig. 2.2 depicts the mortality function of German men and women for the year 2004. We emphasise that the mortality is shown on a logarithmic scale.

2.1.1 Experimental data

Vast experimental data has been collected in the last century, including life expectancy, mortality and survival probabilities (Fig. 2.3). Fig. 2.2 shows that the mor-

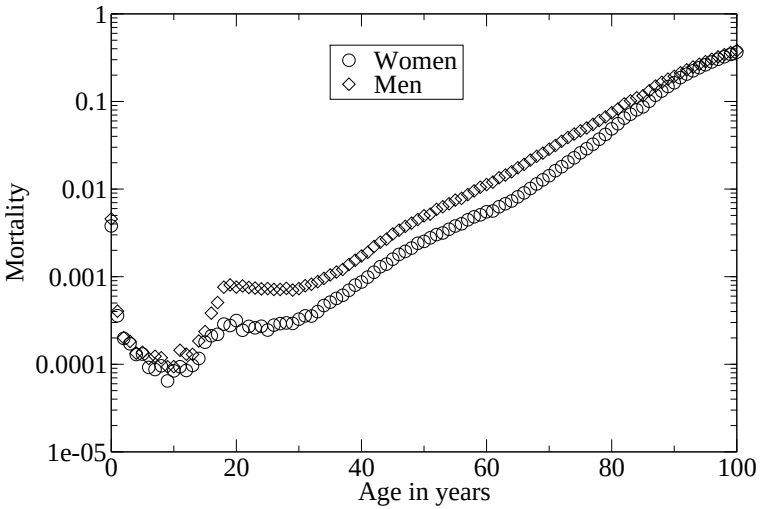


Figure 2.2: Mortality function of German men and women in 2004 (www.destasis.de).

tality function for German men and women increases exponentially with age a:

$$\mu(a) \propto e^{ba+c}, \tag{2.1}$$

where b is the Gompertz slope. This law, observed by Gompertz (1825), was proved to be valid for the populations of most industrialised countries. The original law without the small constant c was modified, still in the 19th century, by adding c to the Gompertz exponential increase. This is called the Makeham modification (Makeham, 1860).

If $S(a)$ is the probability of surviving from birth to age a, then the mortality function is:

$$\mu(a) = -\frac{d(lnS(a))}{da}. \tag{2.2}$$

Due to the fact that data normally is given in yearly intervals, Equation (2.2) needs to be approximated to:

$$\mu(a) = \ln\left(\frac{S(a)}{S(a+1)}\right). \tag{2.3}$$

The fit of the measured mortality function (Figure 2.2) to the Gompertz law shows that it looses its validity for ages smaller than 25 years. Child mortality, even after recent medical achievements which were able to lower it crucially, still plays an important role in human mortality. The mortality function also differs from the Gompertz law for very old ages for many animals. This "effect of the oldest old" will be described in detail in Section 2.4.

Medical progress not only decreased child mortality but additionally increased our life expectancy from 35 to 80 in the last 250 years, by lowering the death probability at intermediate ages. The slope of the mortality function increased (not shown) and survival probabilities now approach a step function (see Fig. 2.3).

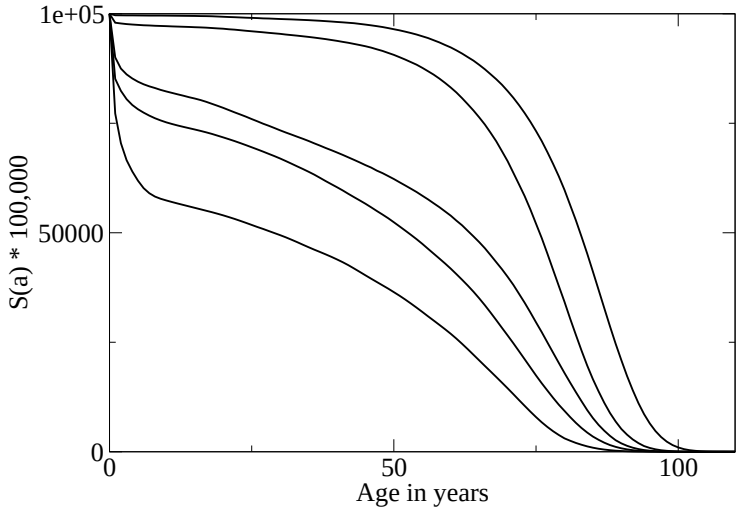


Figure 2.3: Survival probabilities for Swedish people for the years 1800, 1850, 1900, 1950, 2000 (from left to right).

The data presented above were obtained for humans, but the same general behaviour can be frequently observed for other species as well. In order to avoid deaths by external factors (as starvation, predation and so on), experiments have been performed and data have been collected in laboratories and zoos. A Gompertz law is equally reported for almost all animals and even for some bacteria, as well as for objects like cars (for an overview on mortality see Vaupel et al. (1998)). Exceptions are mayflies (ephemerals, Carey (2002)) whose mortality function increases linearly with age.

2.1.2 Theories

For a review of most of the theories of ageing from a biological point of view see Kirkwood (2005). Reviews of computational models can be found in Moss de Oliveira et al. (1999b) and Cebrat and Laszkiewicz (2005). Although there are many different theories to explain ageing, it is still not clear which are the main mechanisms involved. Some of the most accepted ones are summarised below.

- Weissmann at the end of the 19th century argued that we die in order to make place to our children. Thus the age at which an individual becomes able to reproduce, the minimum reproduction age, and its age of death should self–organise at certain ages. Stauffer and Radomski (2001) captured this self-organisation through a very simple model that, however, does not give a mortality increasing exponentially with age.

- The theory of mutation accumulation, which provides the base of the work presented in this thesis, was discussed at first by Medawar about 50 years ago. Inherited diseases, leading to death, are the main cause of ageing according to this theory. Each of these diseases becomes active at a particular age of an individual. Thus, as we become older, more and more diseases, inherited genetically from our parents, threaten our lives decreasing our survival probabilities. Diseases killing an individual before it reaches the reproduction age are not transmitted to the offspring and so should vanish from the population. On the other side, diseases acting late in life allow the individual to reproduce and so are not selected against. Additionally, they help a species to survive by killing old individuals, if their presence does not have any beneficial effect to the population but to compete for the same resources with the youngsters.

- Similar to the theory of mutation accumulation is antagonistic pleiotropy: Some inherited mutations have beneficial effects in youth and bad ones at old ages. For instance, a mutation increasing the amount of Calcium in the body helps to build the bones at young ages but can lead to arteriosclerosis at old ones.

- Another theory of ageing is the soma theory of Kirkwood: the production of offspring and ageing are controlled by the finite limit of resources. Individuals are forced to balance between spending theses resources at young ages, or keeping them up to older ages.

- Damage can accumulate in our bodies during life by abrasion or improper usage. Additionally, it is known that oxygen radicals can damage the DNA–strings of our genome by causing harmful somatic mutations. Hence, certain damages may endanger the entire organism and lead to death.

- At the end of each DNA strand there are sections called telomeres. These sections are partly lost during cell division. In experiments made *in vitro*, that is, on isolated cells outside a living body, this deficit limits the ability of a cell to divide infinite times. After some dozens of divisions the process stops. The limit is called Hayflick limit (Hayflick, 2003). The enzyme Telomerase operates against this loss of telomeres in living organisms by repairing the ends of the DNA. Hence, the concentration of telomerase in our body regulates the replacement of dead cells. Too high concentrations enhance the probability to generate cancer cells due to bad mutations. On the other side, too low concentrations can lead to a malfunction of the organism due to the small number of living cells. Thus there should be a balance between these two extremes. Simulations considering telomerase and cancer gave reasonable results (Masa et al., 2005), with an exponential growth of the mortality function.

- The last theory of ageing described here is based on the reliability theory designed for machines: the reliability of our immune system to remove dangerous cells like cancer cells worsens when we get older. Gavrilov and Gavrilova (2001) suggested that the reliability stops to worsen for very old ages. With this modification the mortality function shows a plateau for the oldest old. Shklovskii (2005) proposes a linear decay of the ability of an organism to get rid of that cells. It can be shown with a simple calculation that the mortality function follows the Gompertz law in such a scenario. Pletcher and Neuhauser (2000) combined the reliability theory with the one of mutation accumulation and obtained reasonable results. The mortality function was calculated for some non–living objects like cars and a Gompertz law similar to the one for humans and animals has been found (Vaupel et al., 1998). Every important function of these objects is fulfilled by several similar units, similarly to the function of cells in an organ of our body. Reliability theory was found to be able to explain ageing of these non–living objects (Gavrilov and Gavrilova, 1991).

Many of the theories presented above do not exclude each other, and thus can be combined. In fact, the Penna model, based on the mutation accumulation theory, has been successfully applied considering also telomerase (Masa et al., 2005), antagonistic pleiotropy (Sousa and Moss de Oliveira, 2001) or Weissmann's proposal (Stauffer and Radomski, 2001). Due to its versatility, robustness and a particularly simple and fast computational strategy, the Penna model is now the most widespread one to simulate ageing and many other features of biological evolution. It was the first computational model to reproduce the Gompertz law of mortality successfully.

2.2 The asexual version of the Penna model

The informations that are transmitted from parents to offspring are encoded in the DNA of each of our cell nucleus. The nucleotides adenine (A), thymine (T), cytosine (C), and guanine (G) provide an alphabet of four letters to describe our genes. Hence, the fact that the information in our computers is packed into a binary system, invites us to represent genes and DNA by computer bits (Eigen, 1971).

The Penna model (Penna, 1995) implements the mutation accumulation process using an individual–based strategy. Each individual of the population is considered as an independent unit and is represented by its "chronological genome". This genome is a bit-string, usually of length $L = 32$ bits, and contains the information of when the individual will start to suffer from the effects of a given genetic disease. In computational terms it is suitable to implement it as an *unsigned long integer* ($C/C++$ nomenclature) which has 32 bits on a normal personal computer.

Diseases: The two possible states of each bit of the bit-string account for activity (1) and non–activity (0) of a life–threatening inherited disease. The idea is to link each bit-position to a time interval of the individual's life; at each interval a different disease may become active or not, depending on the value (0) or (1) of the bit at that position. In this way, for $L = 32$, each individual can live at most for 32 periods. Each iteration corresponds to read a new bit-position of all "chronological genomes" or, equivalently, to investigate among the whole population which individuals carry a disease that will start to affect its health from that period until death. Each period may correspond to days or years, depending on the species. Here we will refer to it as "one year", which means that no individual can live beyond 32 years. At the end of every iteration the age of all alive individuals is increased by one. As soon as the current number of accumulated diseases of any individual reaches a threshold T, the individual dies. For instance, suppose a 10 years old individual, that is, an individual's genome whose 10 first bits have already been read. Suppose also that it has accumulated two active diseases during this period (has two bits 1 up to that age, like in 0000010001....). If the threshold is $T = 3$, this individual may survive to the next iteration, when its 11-th bit will then be read. However, if $T = 2$, this individual dies at the current iteration, that is, at the age $a = 10$.

Reproduction: When an individual reaches the minimum reproduction age R, it generates b offspring on that and on all following years, until death. Each offspring genome is a copy of the parent's one, except for a deleterious mutation that may occur with probability m, at a randomly chosen position of the offspring genome. Only deleterious mutations are carried out, that is, if a zero bit is randomly chosen to mutate, it is set to one in the offspring genome; however, if an already set bit is

selected, it remains one in the offspring genome and no mutation occurs. Beneficial or back mutations ($0 \rightarrow 1$) are neglected because they occur about 100 times less frequently than the deleterious ones (Pamilo et al., 1987).

If every offspring had its genome worse than that of its parent the whole population would die out fast due to the accumulation of deleterious mutations (Lynch and Gabriel, 1990). This effect is called mutational meltdown and is avoided in the Penna model by those cases where the bit randomly chosen to mutate in the offspring genome is already set, and in fact no mutation occurs. The offspring is then genetically as good as the parent and will have the same chances to reach the minimum reproduction age and to produce its own offspring, contributing to the preservation of the species.

Figure 2.4: Pierre Verhulst (1804-1849).

Verhulst factor: With the strategy described above, depending on the value of the birth rate b, the population either dies out or grows exponentially. In order to prevent the unlimited growth, the population size must be controlled either by reducing the number of offspring, or by adopting some additional death rule. A constant population size can be obtained, for instance, by randomly choosing individuals with age $a \geq R$ to generate offspring, just until the total number of individuals equals the one of the previous iteration (de Oliveira et al., 2004a). Within this approach the birth rate is adjusted automatically to compensate the number of genetic deaths. Obviously, simulations with a constant population size never lead to extinction and so do not allow the detection of mutational meltdown and make it difficult to check the robustness of the population in the presence of external influences. Nevertheless, this

strategy avoids the noise induced by fluctuations in the total number of individuals and can be useful if applied with these draw-backs in mind.

A more realistic approach to avoid an exponential growth is to introduce competition, for instance for food or space, among the individuals of the population. The simplest way to implement such environmental effects is the so–called Verhulst factor,

$$V = \frac{N(t)}{K}, \tag{2.4}$$

where $N(t)$ is the current population size and K is the *carrying capacity*, denoting the maximum population size the environment can sustain. The Verhulst factor accounts for the probability of an individual to die in an iteration due solely to competition, independently of its age or genome. In the original Penna model, at each iteration a random number between zero and one is tossed for each individual. If this number is smaller than V the individual dies. Deaths by this additional factor result in a rather stable population size, generally fluctuating around a value ten times smaller than K.

Another, biologically more realistic, but computationally more unstable approach, applies the Verhulst factor only to the newborns (Sá Martins and Cebrat, 2000) instead of to all alive individuals. A further method to avoid random deaths at all is to implement the Penna model on a spatial lattice (Sousa and Moss de Oliveira, 1999; Makowiec, 2001). This approach takes into account the position of the individuals on a square lattice and simulates a more realistic scenario where the species is distributed over large areas and the individuals do not present a high mobility. Then, a newborn survives only if it can occupy an empty lattice site besides the one of its mother. In this case random deaths can be completely avoided and the population size is controlled by the number of lattice sites. The spatial distribution of the population does not interfere with its genetic and age structures, but a larger computational time is needed due to additional parameters, like the movement rate. In this way non–spatial versions are preferred if there is no interest in a particular spatial effect.

A detailed analysis and comparison between a constant population model and the traditional Penna model with its random killing Verhulst factor will be given in Section 2.4.

Summarising, the parameters of the original asexual version of the Penna model are:

1. $N(0)$ - initial population
2. K - carrying capacity
3. T - threshold for inherited diseases
4. R - reproduction age
5. b - birth rate

6. m - mutation rate

These parameters are kept constant during the whole simulations and typical values correspond, for instance, to $T = 4$, $R = 8$, $b = 1$, $K = 100,000$ and $m = 1$. In order to obtain good statistical results, quantities like the mortality or the survival rate are averaged over at least the last 10,000 iterations.

Simulations with larger bit–string lengths do not change significantly the results (Penna and Stauffer, 1996). Also the substitution of each bit-string by an integer number, in such a way that a life–threatening disease only becomes active at its associated age if this integer number exceeds some value varying between zero and nine (Bernardes and Stauffer, 1995), gives qualitatively the same results.

Figure 2.5 shows the frequency of bits 1 as a function of age, for a single run of the traditional Penna model. It can be observed that $T - 1$ inherited diseases become fixed before the minimum reproduction age, for the entire population, which means that the asexual population lives at the edge of death. For ages higher than R, the number of bits 1 that become fixed increases strongly. As mentioned before, old individuals have already produced offspring and so are not anymore needed to maintain a stable population. If they were to stay alive, they would continue to compete with the youngsters for the limited resources. This is the explanation for ageing according to the mutation accumulation theory: selection pressure decreases with increasing age and old individuals die due to the accumulation of harmful mutations. This effect leads also to a maximum age of life (de Almeida et al., 1998), similar to reality.

The exact positions where the bits 1 become fixed depend on the random seed with which the simulation is initialised. For other simulations with different random seeds, but the same parameters, the fixed diseases can appear at different positions. For simulations over long times, that is, for a number of iterations higher than the population size N, it can be shown that the entire population descends from *one* common ancestor. This so–called Eve effect is observed in real populations as well and provides an essential characteristic of evolutionary dynamics presenting critical behaviour (de Oliveira et al., 1997). The common ancestor of the entire population can be a different one if we perturb the system only slightly by, for instance, choosing a different initial random seed.

Figure 2.6 depicts the mortality function versus age in a semi–logarithmic plot (see Equation (2.10) in Section 2.4). It has to be emphasised that deaths by the Verhulst factor are not counted, since the fraction of individuals that die due to Verhulst is the same at all ages. Thus the curve would only be shifted to higher values of the mortality function. Mortality increases exponentially at nearly all ages and the Gompertz law is well reproduced. Young individuals with age smaller than T do not die due to genetic diseases because they cannot have already accumulated T active inherited mutations.

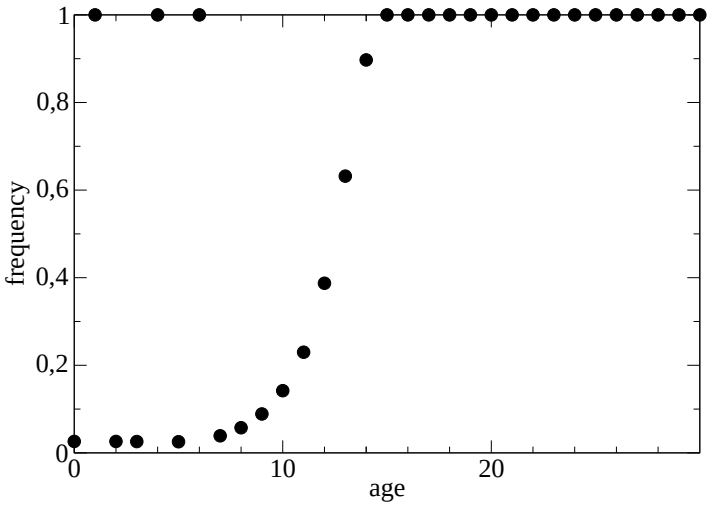


Figure 2.5: Frequency of set bits in a simulation of the asexual Penna model. The parameters are $N(0) = 5000$, $K = 750,000$, $T = 4$, $R = 8$, $b = 1$ and $m = 1$. From the figure it can be noticed that $T - 1$ mutations become fixed before the minimum reproduction age R.

The Pacific Salmon does not only taste wonderful but apparently does not age during its life. It lives until it generates offspring and dies. Salmons enter their spawning streams after living in the oceans (for instance after two years for the Pink Salmon), usually returning to the place where they originated, in order to reproduce and to die. The mayfly provides another example of this so called catastrophic senescence. It is very easy to simulate this effect using the Penna model: it is enough to replace the instruction *if age* $\geq R$*, reproduce* by *if age=*R*, reproduce*. This simple modification leads to a scenario where the survival probability of the whole population jumps abruptly to zero at the age $R + 1$ (Penna et al., 1995), as a consequence of limiting the reproduction period. Now the unavoidable mutations accumulate randomly from age $R + 1$ to 32, since there is no selection pressure to prevent it: with or without mutations the individuals stop reproducing at age R.

For an already stable population, the survival rate is given by the ratio between the

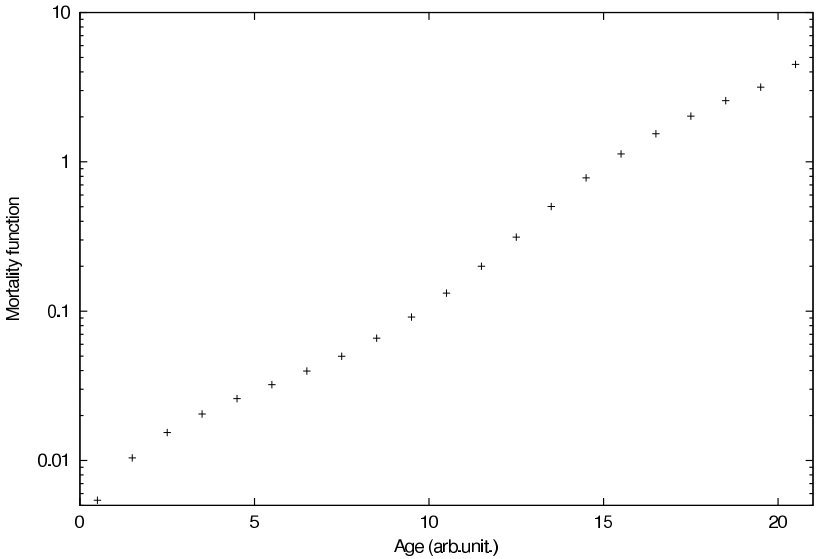


Figure 2.6: Semi–logarithmic plot of the mortality function for the asexual Penna model. It increases exponentially as predicted by the Gompertz law. The parameters are the same as in the previous figure, except for $T = 1$. Simulations with different parameters lead to similar results (Figure from Moss de Oliveira et al. (1999b)).

number of individuals with age $a + 1$ and the number of individuals with age a:

$$S_r = \frac{N(a+1)}{N(a)}. \tag{2.5}$$

It corresponds to the probability of surviving from age a to age $a+1$. The normalised survival rate is obtained by dividing all the survival rates by the first one, that is, by the survival rate from age 0 to age 1. The advantage of this normalisation is to eliminate the Verhulst factor, since from age 0 to 1 deaths can only occur due to this factor. In this way the results do not depend on the population size but only on its genetic charge. Figure 2.7 shows the difference between the survival rate for the usual case, which declines smoothly at ages higher than R, and the survival rate for the catastrophic senescence case, where reproduction occurs only once. (Observe also the similarity between the shape of the survival rate corresponding to the usual case (circles) and the shape of the survival probabilities obtained with experimental data presented in Fig. 2.3.) Attempts were made to obtain the catastrophic senescence effect using models which do not consider the theory of mutation accumulation, but only with large difficulties or even without success (Jan, 1994; Meyer-Ortmanns, 2001).

As stated above, child mortality is not observed with the Penna model. Instead, young

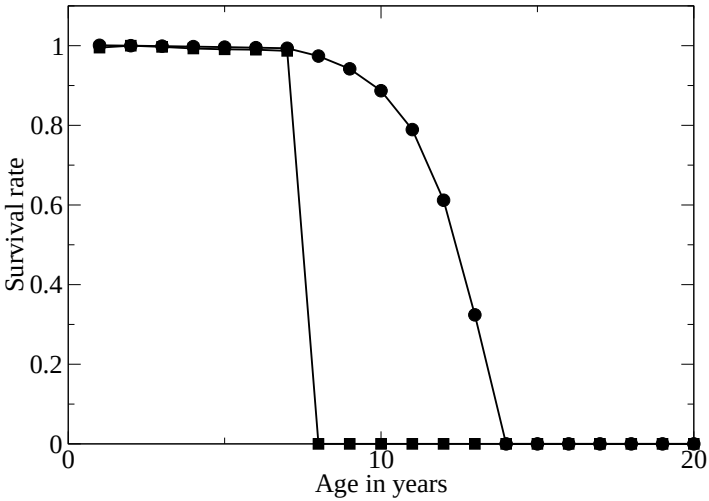


Figure 2.7: Normalised survival rates given by the asexual Penna model for the catastrophic senescence case where reproduction occurs only once at $R = 8$ (squares) and the usual case where it occurs from $R = 8$ until death (circles). The other parameters are: $N(0) = 50,000$, $K = 500,000$, $T = 1$, $b = 2$ and $m = 1$.

individuals have lower mortality since there is no genetic death for ages smaller than T. Also in reality, there is a strong deviation of collected statistical data from a Gompertz law as can be seen by in Figure 2.2. Simulations of the Penna model which consider the effects of housekeeping genes succeeded to explain such high child mortality (Laszkiewicz et al., 2001). These genes are responsible for cell maintenance and cell activity and encode proteins required for essential functional and structural purposes in most cell types, independent of their particular histology (i.e., tissue-specific cell characteristics). Moreover, their expression does not depend on the current state of a cell and thus the comparison of the expression of housekeeping genes with the expression of other genes offers a valuable method to measure the relative frequency of the latter ones. Defects of housekeeper genes cause early death during the development of an organism, occurring from times shortly after the formation of a zygote (first cell of an individual) up to later childhood.

Dealing with the other extreme of ages, deviations from the Gompertz law may emerge for the oldest old. The mortality function no longer increases exponentially

with age leading to a mortality plateau at first observed for flies (Carey et al., 1992). Vaupel et al. (1998) provides an overview of the species which present such a deviation. Until now, there is no conclusive evidence for the appearance of a mortality plateau for humans. Section 2.4 presents a more detailed description of this phenomena and deals with simulations which account for the effect of the oldest old and its impact on the population structure.

2.3 The sexual version of the Penna model

This section presents the extension of the Penna model to species which reproduce sexually and will face questions like “Why does sex exist?”, “Why do women have menopause?” and “Why do men have a higher mortality?”. Sexual reproduction is observed for most eukaryotes: mammals, plants, birds, reptiles,.... Procaryotes have no cell nucleus and their genetic material is distributed along the plasma. Their mayor group, the bacterias, reproduce asexually simply by making a copy of their genomes (mitosis) at each cell division.

First we focus on the biological background of sexual reproduction. Each cell of sexually reproducing organisms contains two sets of homologous chromosomes in its nucleus. Individuals with these two sets are called diploids whereas asexually reproducing ones, like bacteria, are called haploids.

Sexually reproducing species also develop haploid cells containing only one set of chromosomes. These cells, the gametes - or, in other terms, sperms and egg cells - are the components needed to generate offspring. Both types of sexual organs produce gametes by meiosis. First the same process as in asexual organisms, mitosis, is carried out: The pair of homologous chromosomes is duplicated generating a new pair almost identical to the original one (the copying machinery is not perfect and so the mutations appear). Furthermore, the DNA strings of each chromosome break at identical positions (but different for the two pairs of chromosomes) in order to recombine by exchanging the complementary pieces. As a consequence, a piece of an homologous set now makes part of the other one and vice versa. These processes of crossover and recombination mix the genome so that the two new pairs of homologous chromosomes differ from the original ones. After that, the cell divides into two diploid ones containing each one of the two new sets of chromosomes. Finally, each cell divides again into two haploid ones by distributing one set of the already crossed strings (one set of chromosomes) to each. The outcome of meiosis corresponds to four gametes, each one with a different set of chromosomes, differing also from the original ones. Figure 2.8 illustrates the processes of mitosis and meiosis. Under circumstances which can be very enjoyable, a gamete of the father joins the one of the mother and produces a zygote, a cell which finally contains a pair of homologous

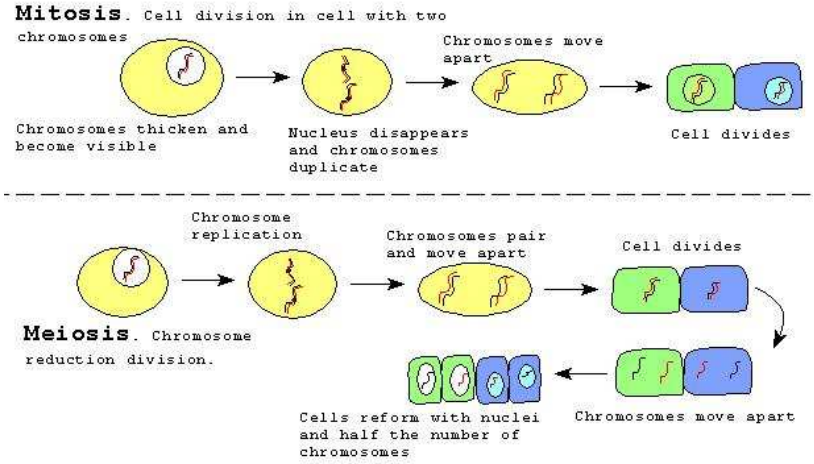


Figure 2.8: Illustration of the process of asexual (mitosis) and sexual reproduction (meiosis). In mitosis the chromosomes duplicate, finally producing a new cell. Diploid organisms duplicate their genome by mitosis as well. Afterwards the chromosomes are crossed and recombined in order to build four gamete cells. This process leads to genetically distinct gametes. The cross–over of the chromosomes is not shown in this figure.

chromosomes, one set from the father and the other from the mother. To summarise the whole process of reproduction: a pair of chromosomes is copied (leading to mutations) and mixed (crossover and recombination) in order to produce four gametes. Two gametes, one from each parent, combine to produce the zygote, which contains the genetic information coming from the mother in one chromosome and that of the father in the homologous one.

How do we include this rather complicated process into the Penna model? Fortunately, it seems more complicated than it is. Figure 2.9 shows that each individual now is characterised by two bit–strings (of four bits each in the case of the figure), which are the components of the diploid genome. At each iteration/year a new position is read on *both* strings. A female which already reached the minimum reproduction age searches for a mating partner in order to produce offspring. A father with an age equal or larger than the minimum reproduction age is selected randomly. Directed selection, the so called assortative mating, will be discussed in Chapter 3. Offspring will be male or female with equal probability. The two bit–strings of each parent are cut at a random position and recombined as illustrated in Figure 2.9. The genome of the offspring contains one bit–string coming from each parent. Each one

of the new bit–strings suffers a bad mutation (from zero to one), with probability m, at a randomly chosen position.

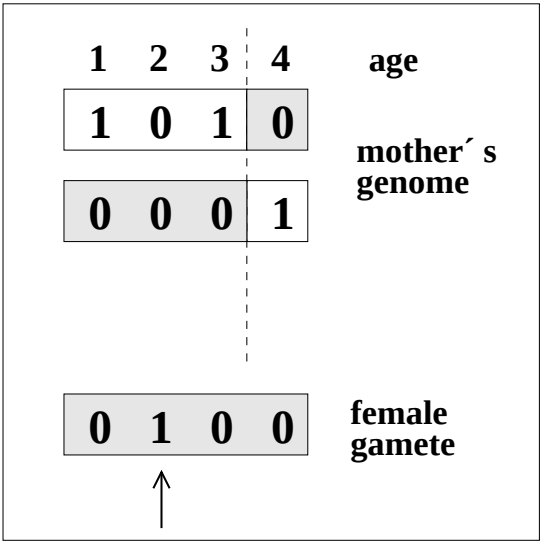


Figure 2.9: Schematic representation of gamete formation. The pair of bit-strings of the mother is cut at a random position and recombined to form a gamete. Mutations are then introduced at random positions (arrow). Only bad mutations are allowed. The same process occurs with the father's genome. The union of the two gametes forms the offspring genome.

Genes can have different states called alleles, coding for instance, for the colour of a flower. The number of possible alleles is limited (at least by the number of possible configurations of the DNA of that gene). When the offspring inherits equal alleles from both parents it is called homozygous for that gene, and the expression of the gene in the offsprings' phenotype is assured. If it inherits different alleles from each parent, the now heterozygous offspring expresses one of the two alleles in its phenotype. The trait associated to a given allele can be dominant or recessive (or none of both, sharing a more complicated outcome). A dominant trait is expressed if its corresponding allele is present at least in one of the chromosomes. For the expression of a recessive trait both chromosomes need to contain the corresponding allele.

The sexual version of the Penna model uses a simplified concept of dominance for certain alleles (Stauffer et al., 1996). The positions (encoding for the activity of a certain life–threatening disease at the age this position will be read) are distinguished between recessive and dominant positions. At each iteration one bit of each of the

two diploid strings is read in parallel. If the nature of a given position is dominant, already one gene set to one (heterozygote) in one of the two bit–strings suffices to turn on the activity of its corresponding disease. At recessive positions both bits need to be set to one (homozygote) to activate a disease. Usually, the number of dominant positions is chosen to be smaller than that of recessive ones.

Using this concept of dominance we are able to count the number of active deleterious mutations in the same way as in the asexual model, in order to find out at which age an individual will die. At the beginning of the simulation we define how many positions are going to be considered the dominant ones. Then we randomly select which are going to be these positions. They remain fixed during the whole simulation and are the same for all individuals.

The mortality functions obtained using the Penna model for asexual (2.6) and sexual populations can be compared. The conclusion is that the introduction of sexuality does not crucially change the shape of the mortality curve. In both cases an exponential increase of the mortality with increasing ages is obtained, in agreement with the Gompertz law.

Now we will treat the questions of why there is menopause and how it originated. Menopause is understood as the cessation of females reproduction at intermediate ages, as observed not only for humans but also for many animals and even for a fly species. Menopause is simulated through the Penna model by introducing a maximum reproduction age, M. Individuals can generate offspring only at ages between the minimum reproduction age R and M.

The results are similar to those obtained for the Pacific Salmon using the asexual version of the Penna model, if both R and M are the same for females and males. In a population where all individuals stop reproduction at a given maximum reproductive age M, individuals with a 0-bit at age a have selective advantage over others only if $a \leq M$. There is no selective advantage at all for $a > M$. Thus, genetic drift will sooner or later populate all individual genomes with 1-bits at ages beyond M. The consequence is the catastrophic senescence: The survival probability of the whole population vanishes for $a \geq M + 1$. Observe that for a given individual, to have a 0-bit at a given age a is a selective advantage, compared to other individuals, only if this age falls into the reproductive period of its descendents. This advantage is the more pronounced the larger is the number of descendents which will be still reproductive at that same age a.

More interesting is the realistic situation where females have a maximum reproduction age M, while males can reproduce until death. Now, many individuals of both sexes survive beyond ages larger than M. It is the presence of the males which helps females to survive menopause. Men can reproduce until old age; on the other hand the sex of the child is determined randomly. If all the bit positions above the menopause age would be equal to one, they would not only kill all the post-menopausal females

but also the males above that age, and thus would reduce the number of births. Moreover, now an individual with a 0-bit at some age $a > M$ have a selective advantage, since this age a may fall into the reproductive period of its male descendants. Both males and females have the quoted “half-advantage", because in both cases the descendents are half-to-half divided into male and female offspring. There is absolutely no advantage at all of males over females.

But why did menopause evolve? The arguments given above explain the stability of menopause in a population where menopause already exists, but not why it appeared. Observe that if there is no menopause (males and females can reproduce until death) the whole population survives longer than if females menopause is considered, as shown in Figure 2.10.

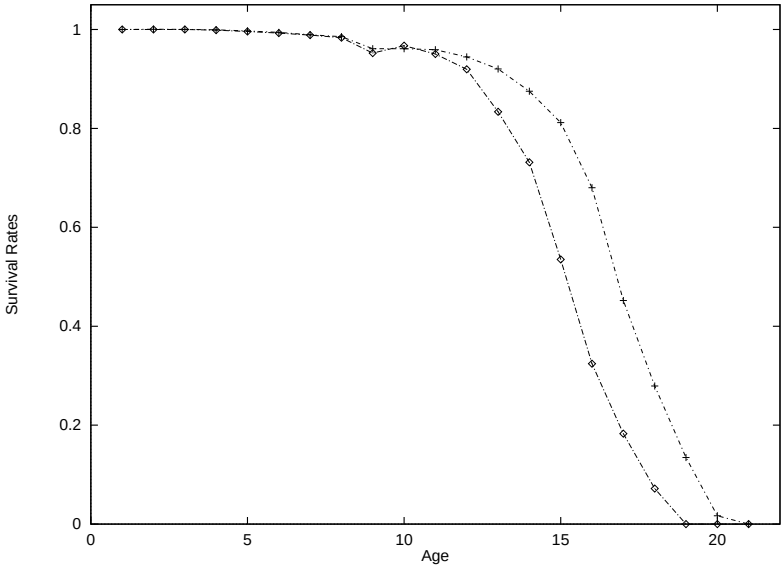


Figure 2.10: Normalised survival rates obtained with the sexual Penna model. Males reproduce from $R = 8$ until death (age=32). For females, diamonds correspond to reproduction from 10 to 12 and stars from 10 to 32 (no menopause). It can be seen that without menopause the whole population survives longer. The other parameters are: $T = b = 4$ and $m = 2$ (one mutation from each parent) (Data obtained from S. Moss de Oliveira).

The self–organisation of menopause using the Penna model was obtained in 1999 (Moss de Oliveira et al., 1999a), in the following way: Initially all individuals can reproduce from $a = R$ until $a = 32$ (or until death, since no individual can live

beyond 32 years). So at the beginning all females have a menopause age equal to 32. Each female offspring inherits the mother's menopause age with probability p, or the mother's menopause age +1 or -1 with probabilities $1 - p/2$. Then, two new ingredients are considered, common for humans and many animals: Parental care and a risk of giving birth that increases with the mother's age. Now a newborn needs the mother to stay alive for pc iterations; if the mother dies before it, the offspring dies as well. Figure 2.11 depicts the results of these simulations showing that menopause self–organises below the maximum lifespan for $pc \geq 4$. This outcome shows that

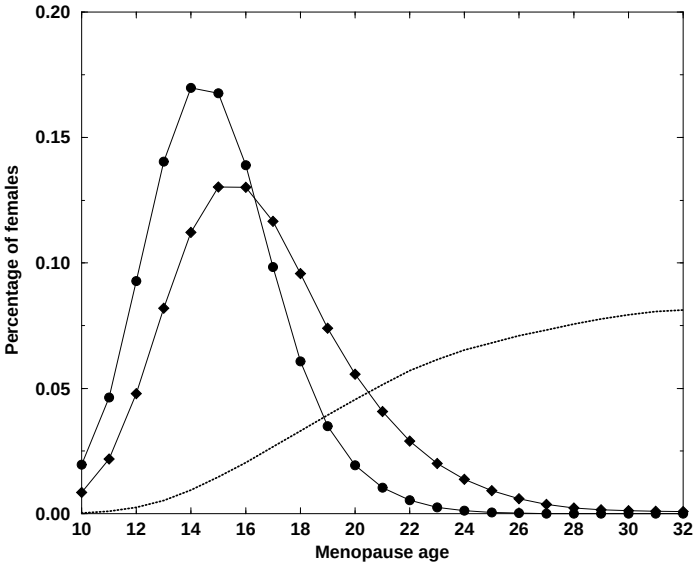


Figure 2.11: Histogram of the percentage of females with a given menopause age, for $R = 10$. The line corresponds to no parental care ($pc = 0$); diamonds correspond to $pc = 4$ and circles to $pc = 5$. For $pc < 4$ iterations the self-organisation of the menopause age was not obtained.

the evolution of menopause results directly from properties shared by many animals and that are not particular for humans. Some people like to give cultural reasons to explain menopause like that grandmothers help to take care and educate the children. Indeed, some correlations between the survival of grandmothers and grandchildren were found for humans Lahdenperä et al. (2004) but not for lions. However, a strong correlations between the survival of parents and descendants was already found using

the traditional sexual version of the Penna model (de Oliveira et al., 1998), due to the simple fact that good (and also bad) genes are transmitted from parents to offspring. So, it is not surprising that some correlation appears also between the survival of grandmothers and grandchildren.

Now we will focus on the question: "Why women live longer than men?". The results presented above indicate that there is no evolutionary advantage, neither for women nor for men, related to their different maximum reproduction age. However, while women's genomes have two X–chromosomes, only one X–chromosome is built into the men's genome. The single X–chromosome is accompanied by the smaller Y–chromosome. During meiosis the X– and the Y–chromosome distribute equally to build two types of male gametes, containing either only the Y–chromosome or only the X–chromosome. The type of male gamete which wins the final competition to gather the female gamete (X-chromosome) and form a zygote defines the sex of the offspring. Any bad mutation in an X-chromosome means higher risk for men than for women, since the female genome can counteract its harmful effect by activating the same gene in its other X–chromosome. Simulations with the Penna model (Schneider et al., 1998) using a larger diploid genome distinguishing between X and Y chromosome could reproduce typical human reality: Mortalities for men are twice as high as for women up to very old age where the ratio of the two gets close to unity.

A crucial test for this XX-XY chromosome explanation are the life expectancies of birds. In bird species the situation is the opposite to that of mammals: in the genome of female birds a second X–chromosome is substituted by an Y–chromosome whereas the genome of male birds contains a pair of X–chromosomes. Thus the mortalities of female birds should be higher than the ones of male birds. Unfortunately, no agreement exists if there is a difference of life expectancy for distinct sexes for birds.

At a first glance, one could think that sexual reproduction gives only disadvantages to the vast number of species showing this type of reproductive regime: since only females get pregnant the number of offspring is reduced by a factor two, besides the fact that males also compete with females for food and space. Why did Nature switch from asexual cloning to sexual reproduction for almost all species? Simulational results obtained with the Penna model showed that the reason is the higher genetic diversity generated by sex through the mixing of the genomes of the parents to produce the offspring. This higher diversity leads, for instance, to difficulties for parasites to adapt to their hosts (Howard and Livelyk, 1994; Sá Martins and Cebrat, 2000). For the same reason sexual populations can adapt faster to a rapidly changing environment (Sá Martins and Moss de Oliveira, 1998; He et al., 2005). This scenario was tested through experiments with yeast (Goddard et al., 2005) and agrees with the simulational results.

But is the influence of parasites and catastrophic changes of the environment sufficiently strong to prevail sexual reproduction or is the cost of sex for females too

high (Redfield, 1994)? The fitness of a sexual population needs to be compared with the one of an asexual population. In Sá Martins and Stauffer (2001) a new ingredient was introduced into the Penna model: Each deleterious mutation diminishes the survival probability of an individual by a small percentage in every time interval, in addition to the usual lethal effect if the threshold T is reached. This new ingredient indeed leads to a higher fitness of sexual populations.

In another approach (Scharf, 2004), pre-selection was simulated: a high number of bad mutations reduces the ability of a male gamete to reach and enter the egg cell. Under these circumstances individuals with higher fitness generate more offspring. In this way sexual reproduction involves a crucial evolutionary advantage, since for asexual cloning neither selection of a partner nor this pre-selection exists to improve the offspring fitness.

Selection of a partner is related to assortative mating, in opposition to random mating. The female chooses the appropriate mating partner in order to provide a better survival probability to its offspring. Assortative mating not only helps a population to adapt to different ecological niches but also can be an essential ingredient for a species to bifurcate. Biological speciation and its mechanisms will be treated in Chapter 3.

2.4 Simulations of a mortality plateau

In the original version of the Penna model, an individual dies due to inherited diseases when its current number of accumulated mutations, n, reaches a threshold value, T. Since the mean number of diseases increases with age, the probability to die is zero for very young ages ($n < T$) and equals 1 for the old ones ($n \geq T$). Here, instead of using a step function to determine the genetic death age, we test several other functions that may or may not slightly increase the death probability at young ages ($n < T$), but that decreases this probability at old ones (Schwämmle and Moss de Oliveira, 2005). Our purpose is to study the oldest old effect, that is, a plateau in the mortality curves at advanced ages. Imposing certain conditions, it has been possible to obtain a clear plateau using the Penna model. However, a more realistic one appears when a modified version, that keeps the population size fixed without fluctuations, is used. We also find a relation between the birth rate, the age-structure of the population and the death probability.

2.4.1 Introduction

As already mentioned, still in the 19th century Gompertz found that the mortality function increases exponentially with age. Less or more pronounced decreases of this mortality from exponential growth at old ages, also known as the oldest old effect, have been observed in humans and mainly, in flies (Vaupel et al., 1998).

The asexual version of the Penna model was solved analytically by Coe et al (Coe et al., 2002), who show that the replacement of a sudden death rule after the accumulation of T deleterious mutations (step function) by a probability to survive given by a Fermi-function, leads to a plateau in the mortality curve. Numerically, only a very short plateau was observed before (Thoms et al., 1995; Huang and Stauffer, 2001).

In our simulations of the original sexual Penna model, we obtain that when the genetic death probability at advanced ages is given by a smooth function instead of the usual step function, but is greater than zero at very young ages, the birth rate has to be extremely increased to avoid population meltdown. In this case it is very difficult to measure the oldest old effect, since very few individuals survive until old ages. A small plateau has been observed by imposing a death probability equal to zero for very young ages ($n < T$), as in the original Penna strategy.

Using a model where the population size is constant without fluctuations, we obtain a very clear plateau in the mortality curves, even considering non-zero values for the probability to die at young ages. We also obtain that the age distribution of the population changes dramatically according to the smoothness of the death probability functions at old ages (Schwämmle and Moss de Oliveira, 2005).

In Section 2.4.2 the main features of the original Penna model are briefly reviewed, the genetic death probability functions that are used in order to study the oldest old plateau are introduced and the corresponding results are presented. In Section 2.4.3 we describe a model in which the population size is kept constant and show the results obtained for the same death probability functions of the previous section. In Section 2.4.4 we present the conclusions.

2.4.2 The Penna model for sexual populations

In the original version of the model two strings of 32 bits that are read in parallel represent the diploid genome of an individual. A deleterious mutation is defined by two set bits at the same position of both strings or by a single set bit at a dominant position. At the beginning of the simulation a fixed number of dominant positions are picked and positioned without bias along the genome and remain fixed during the whole process. At every iteration or "year" one more bit position becomes active and the corresponding individual becomes one year older. It dies for genetic reasons if its current number of deleterious mutations reaches the threshold T, which corresponds to the following genetic death probability, $f(n)$:

$$f(n) = \Theta(n - T), \tag{2.6}$$

where n is the current number of deleterious mutations and $\Theta(x)$ is the step or Heavyside function. In order to limit the population size, an additional death probability, the already mentioned Verhulst factor (Equation 2.4), is applied to each individual independently of its age or genome.

At every iteration, any female with age equal or above the minimum reproduction age, R, randomly chooses a male, also with age $\geq R$, to breed and generate b offspring. To construct one offspring genome first the two bit-strings of the mother are cut in a random position (crossing), producing four bit-string pieces. Two complementary pieces are chosen to form the female gamete (recombination). Finally, one deleterious mutation is randomly introduced. The same process occurs with the male's genome, producing the male gamete. These two resulting bit-strings form the offspring genome. The sex of the baby is randomly chosen, with a probability of 50% for each one. This whole strategy is repeated b times to produce the b offspring.

Approximations to the step function:

We use the following approximations of the step function, in order to smooth the original genetic death rule, of killing the individual after the accumulation of exactly

T deleterious mutations:

$$\text{Fermi} - \text{like function} = f_1(n) = \frac{1}{1 + e^{-2p(n-T)/32}}, \tag{2.7}$$

$$\text{Arctangent function} = f_2(n) = \frac{\arctan(2p(n-T)/32)}{\pi} + \frac{1}{2}, \tag{2.8}$$

$$\text{Error function} = f_3(n) = \frac{1}{2} \cdot [\text{erf}(p(n-T)/32) + 1], \tag{2.9}$$

where n is the number of active deleterious mutations and p is a parameter that controls the smoothness of the approximations. When the value of p increases, the death probabilities given by Equations (2.7) to (2.9) converge to the one given by the step function (Equation (2.6)). Observe that what we call a Fermi-like death function is in fact one minus a Fermi-function. Figure 2.12 compares the three approximations with $p = 1$ and also the Fermi-like function with $p = 10$, with the step function death probability.

Results

In simulations of N time steps, the mortality function, $\mu(a)$, is measured over the last N_m time steps, in the following way:

$$\mu(a) = -\ln(1 - \frac{\sum_{t=N_m}^{N} D_{gen}(t, a+1)}{\sum_{t=N_m}^{N} P(t,a)}), \tag{2.10}$$

where $D_{gen}(t,a)$ is the number of genetic deaths (not produced by Verhulst) at age a and time step t, and $P(t,a)$ is the number of individuals with age a at time step t. This way of calculating the mortality is equivalent to the one given by Equation (2.2).

In all simulations that follow, the values of the parameters are: $T = 3$, $R = 10$, $b = 1$, $P_{max} = 200,000$, $N = 100,000$, $N_m = 50,000$ and the number of randomly chosen positions where the bits 1 are dominant is 5.

The mortalities obtained using any of the death probabilities given by Equations (2.7) to (2.9) with $p \geq 10$, are equivalent to those obtained with the traditional step function, that is, no plateau appears. Smaller values of the smoothness p lead to population meltdown. This can be avoided by increasing the birth rate b to very high values ($b > 100$ for $p < 1$), which produces strong fluctuations in the population size making it very difficult to observe a plateau. In fact, to observe a plateau in such conditions it was necessary to decrease the minimum reproduction age from $R = 10$ to $R = 8$ and also to work with very large populations (about one million individuals) to avoid the fluctuations just mentioned and to have a good statistics for the oldest

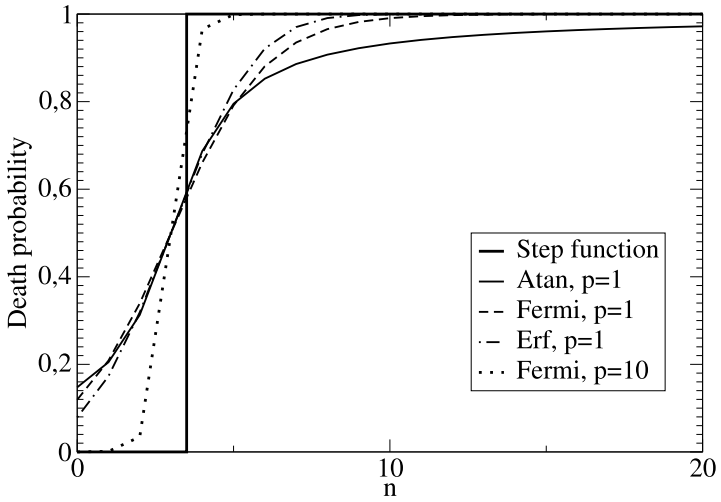


Figure 2.12: Death probabilities according to the different approximations of the step function given by Equations (2.7) to (2.9), versus the number of accumulated diseases. The parameter p controls the smoothness of the functions. The step function (Equation (2.6)) is presented for comparison. Notice that for $p = 1$ there is a finite probability for the very young (small n) to die, but the probability for the older to die is smaller than that given by the step function. For $p = 10$, the behaviour of the Fermi-like death probability becomes almost equivalent to the step function one.

old. We emphasise that in the original Penna model, considering only bad mutations, there is a minimum birth rate to avoid population meltdown, but no upper limit for it. However, a chaotic behaviour like in the logistic map was found for high birth rates with a minimum reproduction age lower than the threshold T (Bernardes et al., 1998). Nevertheless, the stronger selection is, the larger is the minimum birth rate. Another strategy to avoid population meltdown ($p < 1$) is to set $R = 1$, which was used in Coe et al. (2002) to obtain the plateau.

In order to obtain the mortality plateau without restricting the minimum reproduction age R, we set all values of the death probability $f(n)$ to zero for $n < T$. In this way the birth rate $b = 1$ does not need to be increased, and the mortality for different values of p is shown in Figure 2.13, where the death probability is the one of Equation (2.7). For young ages the mortality function follows the Gompertz law. Now a

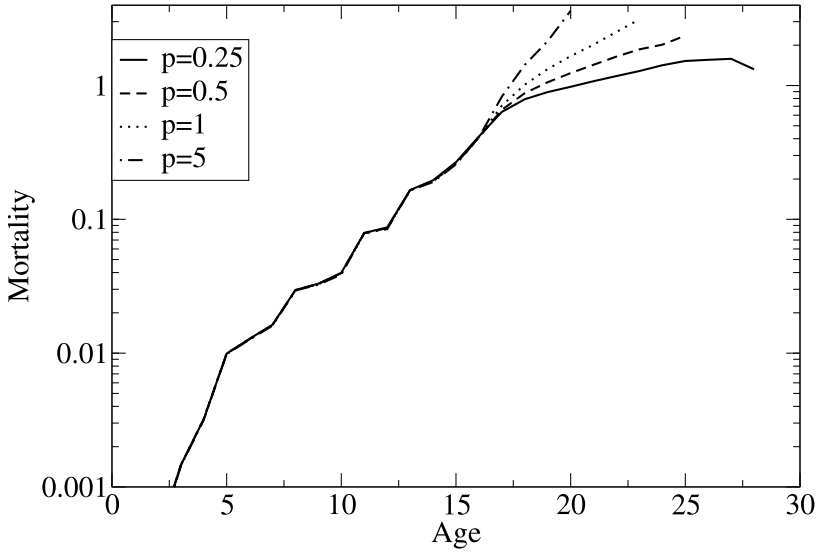


Figure 2.13: Comparing the mortality functions for different values of p, using a Fermi-like death probability function. The results for small values of p are similar to those of the analytically solved asexual model. The fluctuations are due to fixed deleterious mutations of almost all individuals at some ages below the minimum reproduction age.

nice plateau can be observed, similar to the results in Coe et al. (2002). Its length depends on the smoothness p. The different death probabilities of Equations (2.8) and (2.9), also setting to zero the genetic deaths for $n < T$, yield similar mortality functions, as shown in Figure 2.14.

2.4.3 Model with constant population

In order to study the population age structure using the death probabilities of Equations (2.7) to (2.9), but without neglecting deaths for $n < T$, we have implemented the sexual version of a model with constant population, introduced in de Oliveira et al. (2004a). This model has the advantages of avoiding the Verhulst factor already

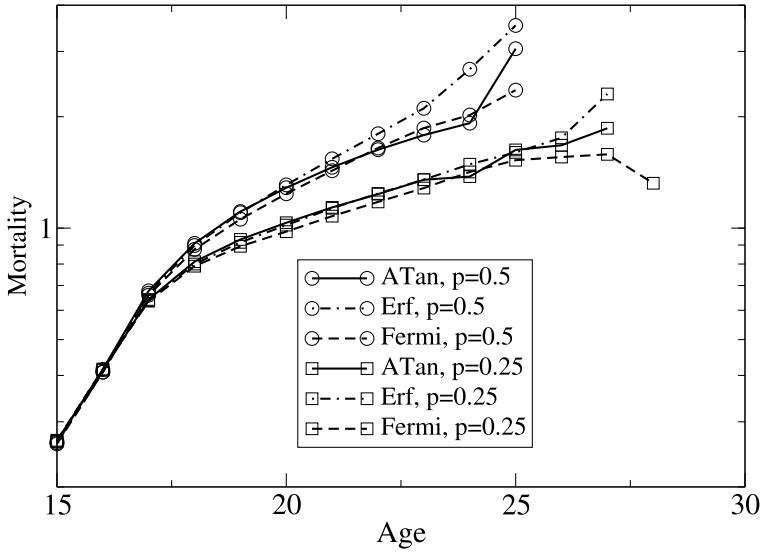


Figure 2.14: Comparing the tails of the mortality for different death probability functions. They differ only slightly for different functions. Individuals with $n < T$ do not suffer genetic death.

criticised by some biologists (Sá Martins and Cebrat, 2000) and preventing chaotic fluctuations of the population size. The only difference between this model and the Penna one is that whenever an individual dies for genetic reasons, a male and a female are randomly chosen to mate and produce an offspring. So, the population size does not fluctuate, since there is no Verhulst factor, and the measured data are much cleaner. Additionally, the birth rate is controlled automatically and population meltdown or unlimited growth are prevented. Nevertheless, the simulation can break down if there are no individuals older than the minimum reproduction age, which occurs for $p < 1$ as well as for too small populations. The population size (200,000 individuals) and simulation time (1,000,000 time steps) have to be large, to produce a mortality function which ranges up to old ages. The genetic deaths and the age distribution are measured over the last 500,000 time steps.

Figure 2.15 shows that the mortality functions do not differ very much from the ones measured with the modified Penna model of Section 2.4.2, Figure 2.13, for old ages.

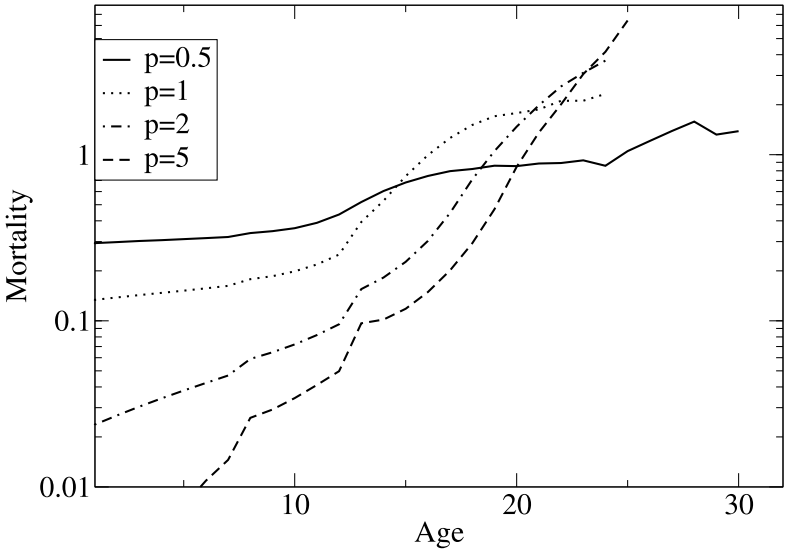


Figure 2.15: Mortality functions using the constant population model with the Fermi-like death probability function, for different values of p. At young ages there is no Gompertz law for small p, due to the non-negligible genetic deaths for $n < T$.

But with decreasing values of p the mortality increases considerably at young ages. The exponential growth is replaced by an almost constant behaviour until the minimum reproduction age. The mortality functions do not vary qualitatively for the different approximations of the step function (Figure 2.16), as already observed in the simulations of the Penna model.

Interestingly, we observe a change in the curvature of the population age distribution, depending on the value of p - Figure 2.17. The smoother the death probability is, the smaller is the mean age of the population. Most of the individuals die at young ages before reaching the age of reproduction. The birth rate increases crucially in order to maintain the population constant. The very few individuals who reach advanced ages can live very long. The really small number of these individuals explains why the mortality plateau is not observed for small populations or short simulation times. Thus, the fluctuations of the values of the mortality function at very old ages, shown in Figure 2.16, are due to poor statistics.

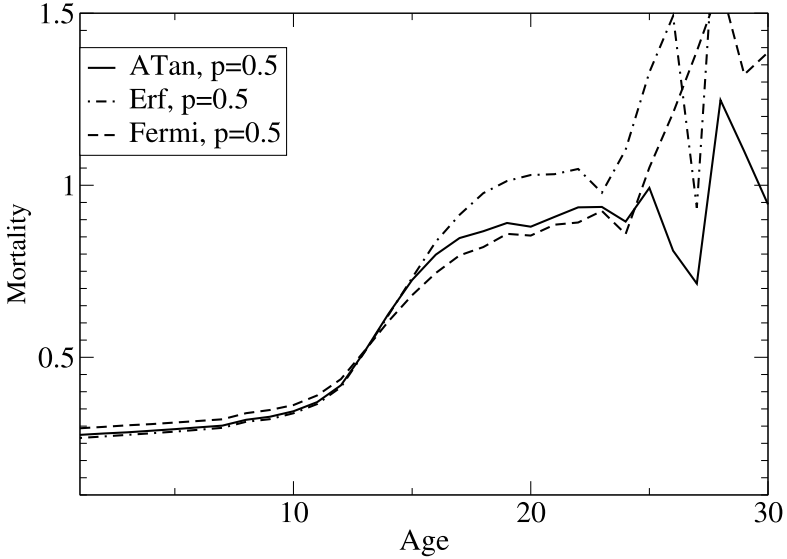


Figure 2.16: Comparison between the mortality functions of different smooth death probabilities using the constant population model, in linear scale. The plateau appears for all of them. The fluctuations of the mortality functions for ages above 24 result from a weak statistics.

2.4.4 Conclusion

With our sexual simulations we reproduce the asexual results of Coe et al. (2002), by implementing a Fermi-like death probability function in the Penna model. The main differences between this model and the asexual model of Coe et al. (2002) are that there, reproduction begins at birth, i.e. $R = 1$ and its Fermi survival probability function depends on the age, while in our case $R = 10$ and the death probability depends on the current number of deleterious mutations.

Our results reveal that the observation of a mortality plateau, using the traditional Penna model with a Fermi-like or any other death probability function smoother than a step function is a rather complicated task. For a reproduction age $R > 1$ most of the individuals die before reaching the minimum reproduction age R. The only way to avoid population meltdown is to increase the birth rate. Simulations with

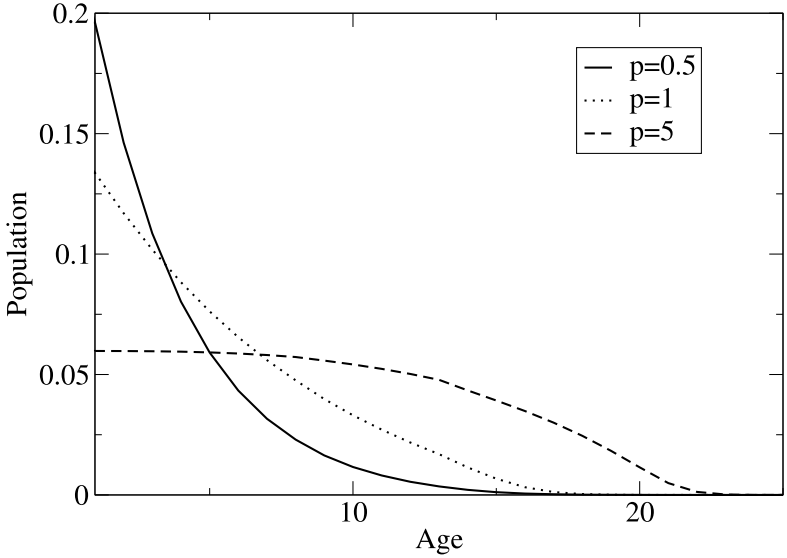


Figure 2.17: The population density changes its curvature for small p. Only few individuals reach old ages.

large population size and simulation time show a small plateau. Nevertheless, the high chaotic fluctuations of the population size due to the large birth rate makes the simulations difficult. However, neglecting genetic deaths before the accumulation of T deleterious mutations, the model reproduces the Gompertz law up to old ages where the mortality function shows a plateau. Additionally, the birth rate does not need to be increased.

In order to avoid neglecting genetic deaths before the accumulation of T mutations, we have used a constant population model. Large populations and simulation times also lead to a clear plateau in the mortality function, which may not follow the Gompertz law, depending on the value of p. For small p, many individuals die before reaching the reproduction age, which may change completely the population age structure.

The different approximations of the step function that have been tested in both the modified Penna model and the constant population model, have lead to similar results

for old ages. Thus, we conclude that the effect of the oldest old results from the smoothness of the genetic death probability at old ages, within the theory of mutation accumulation.

The existence of plateaus in the mortality curves of Drosophilae and other organisms is a matter of fact, as has been reported for instance in Vaupel (1997) and Vaupel et al. (1998). However, the number of Drosophilae surviving up to ages where the plateau appears is extremely small. This same effect has been observed with simulations using the constant population model, but not with the modified Penna model where a reasonable number of individuals survive until advanced ages. The reason is that to obtain the plateau with the Penna model, it is necessary to neglect deaths before the accumulation of T mutations, which allows many individuals to survive up to the minimum reproduction age. The very small number of individuals reaching an age to observe a plateau explains the difficulty to measure the oldest old effect in Nature. Only experiments with more than a million of Drosophilae yield clear mortality plateaus, and even so, their statistics still remain quite poor.

Comparing the very small mortality plateau of humans with the large ones of Drosophilae, medflies, wasps and Nematodes (Vaupel et al., 1998) we propose that there is a relation between the presence of a large mortality plateau and high birth rates, smooth death probabilities and the curvature of the population age distribution. Organisms with a high death probability at young ages need a high birth rate in order to have sufficient individuals reaching the reproductive age. This leads to a mortality plateau and a population distribution with a positive curvature. We suppose that this relation is valuable for simple organisms. A similar relation between the mortality plateau and the population age distribution has already been observed in Gotthard et al. (2000) for butterflies, as well as in Gerhard (2002) for zebrafish. Unfortunately, more data concerning higher developed animals are still missing.

Chapter 3

Biological speciation

3.1 Introduction

The diversity of today's species does not result from an independent development of each of them. Darwin had already formulated the theory of a *Common Universal Descendant* (Darwin, 1859), in which all organisms on Earth are descendant from one common ancestor or one ancestral gene pool. This theory is now generally accepted by biologists. Hence, life is the consequence of an incredible diversification of one type of species into many others, with sometimes very different functions. The process of speciation, that is, the bifurcation of one ancestor species into at least two new ones accounts for Nature's diversity and will be envisioned in detail in this chapter.

Speciation does not occur frequently, and its time and geographic scales generally are immense. Its occurrence depends mainly on three crucial factors: mutations, natural selection and geographic distribution. Only particular combinations of these ingredients are able to increment the number of species in Nature. There exist distinct types of speciation, correspondingly. This chapter focuses on the types of speciation which are not due to chromosomal rearrangements, as for instance speciation by polyploidy (the accidental duplication of the set of chromosomes, a process which has occurred for many plant species) (White, 1978).

First, in order to follow a consistent description, the term species and its exact meaning must be defined. The most accurate definition, the so called *Biological Species Concept*, was first given by Mayr (1942). With this concept, individuals of actually or potentially interbreeding populations of a species are reproductively isolated from the ones of all other species. The absence of reproducing hybrid offspring, generated by parents coming from different species, inhibits that the genes of the individuals of

different species become mixed, and thus the gene flow between distinct species is zero.

The isolating mechanisms which prevent the production of viable and reproducing hybrids can be summarised to the following two types: premating (prezygotic) and postmating (postzygotic) isolation. Prezygotic isolation prevents the union of gametes. The mating partners do not interbreed due to reasons like different mating seasons, habitat isolation, behavioural isolation, or the incompatibility of their sexual organs (mechanical isolation). Postzygotic isolation can occur after the union of the gametes. The hybrid offspring dies after fertilisation, has reduced viability, or becomes partially or completely sterile.

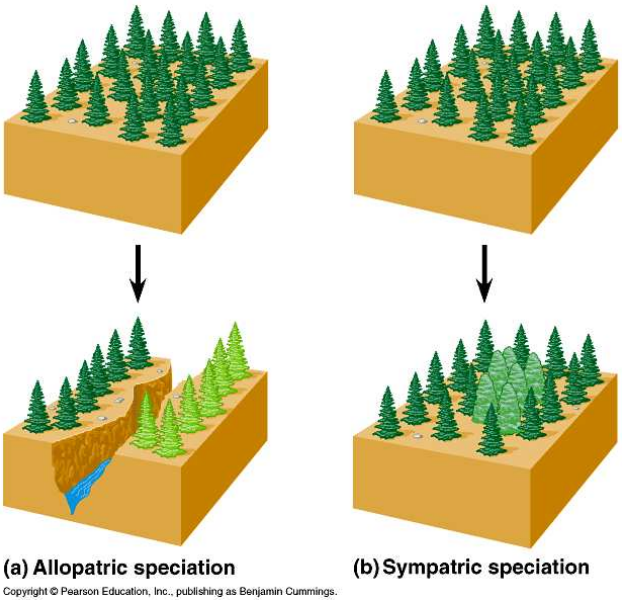


Figure 3.1: The difference between speciation in allopatry and speciation in sympatry.

Speciation occurs due to barriers that prevent the gene flow between sub-populations of the ancestor species. The barriers can be geographic or non-geographic (Figure 3.1). A physical barrier, like a mountain or a river, which divides the habitat in such a way that the original population becomes completely separated into two (or more) isolated groups, can direct the species to the process of allopatric speciation. In this case the sub-populations evolve independently (Mayr, 1963). The individuals of these non-mating populations become genetically different by adapting to distinct environmental conditions or due to uncorrelated genetic drift. Genetic drift means

the fixation of arbitrary mutations which favour neutral traits, as a consequence of the sampling error on allele frequencies in a population. It is enhanced in small populations and is illustrated in Figure 3.2. Allopatric speciation is known to be a gradual and slow process, where the sub-populations become distinct because of their geographic separation, and reproductive isolation develops slowly.

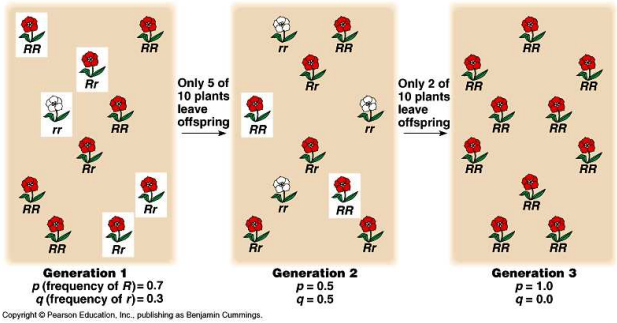


Figure 3.2: Genetic drift in a small population. A particular allele predominates due to stochastic processes.

The absence of geographic barriers does not prevent the bifurcation of a species into two genetically different ones, but is intuitively more difficult (Tauber and Tauber, 1989). Mechanisms which decrease the gene flow between the sub-populations now play a crucial role. In spite of the difficulty to prevent gene flow when there exists contact between all individuals, the theory of sympatric speciation became more and more accepted in the last decades. The combination of laboratory experiments (Rice and Hostert, 1993), measurements (Schliewen et al., 1994; Schluter, 1994; Seehausen and van Alphen, 1999; Via, 2001) and numerical models (Gavrilets, 2000; Lande, 1982; Turner and Burrows, 1995; van Doorn et al., 1998; Higashi et al., 1999; Kondrashov and Kondrashov, 1999; Dieckmann and Doebeli, 1999; Luz-Burgoa et al., 2003; Porter and Johnson, 2002; Sá Martins et al., 2001) gave enormous insights into the processes driving it. Experimental evidences include, for instance, various species of cichlids which live in the volcanic lakes of West Africa. The absence of physical barriers and the environmental homogeneity of these lakes indicate that other mechanisms than geographic separation lead to the formation of such speciational diversity (Seehausen and van Alphen, 1999). Sympatric speciation is supposed to be a fast process (Gavrilets, 2000)

Which mechanisms are able to substitute the effect of a geographic barrier and lead to speciation even at the presence of an initially large gene flow between the individuals? Two mechanisms are thought to be strong enough to give rise to speciation in sympatry (Lande, 1982; Turner and Burrows, 1995; van Doorn et al., 1998; Higashi et al.,

1999; Kondrashov and Kondrashov, 1999; Dieckmann and Doebeli, 1999; Gavrilets, 2000; Luz-Burgoa et al., 2003; Arnegard and Kondrashov, 2004):

(i) The force that drives the adaptation of two sub-populations to two different niches, whereas hybrids are evolutionarily discriminated, is called disruptive selection. Under such circumstances, the gene flow between the two sub-populations of the ancestor species becomes small, and thus this process plays a strong role (Maynard Smith, 1966; Endler, 1977; Felsenstein, 1981). Disruptive selection is related to the competition among the individuals of a species for different resources. For instance, some species of the Darwin finches, living on the Galapagos Islands, need to feed temporarily on large seeds or small seeds. Seeds of intermediate sizes are not available in years with small amounts of precipitation. As a consequence, individuals of the same species develop either large beaks or small beaks, whereas finches with intermediate beak sizes are selected against and their number decreases in the population (Grant and Grant, 2002). Nevertheless, disruptive selection should not be sufficient to obtain a complete reproductive isolation. Whenever a hybrid offspring survives up to the reproduction age and generates offspring, the gene flow between the differently adapted populations is maintained. However, the model of Porter and Johnson (2002) suggests that a small gene flux between different populations does not prevent them from speciation if the hybrids present a low viability.

(ii) Another important ingredient for sympatric speciation is assortative mating. Females choose their mating partners according to some preferences, related to particular traits of the males. Assortative mating, also known as sexual selection, is favoured by evolution in case of disruptive selection, which gives a lower fitness to the hybrids. In this case, prezygotic reproductive isolation develops as soon as females start to mate assortatively instead of randomly (Arnegard and Kondrashov, 2004). Let us assume that the choice of a mate depends on two traits: male display (e.g. nuptial hue, varying from red to blue through purple) and female preference for variants of display. If some females prefer red males and others prefer blue males, this can tear the population apart and create a pair of species consisting of red-preferring females and red males and of blue-preferring females and blue males (Higashi et al., 1999; Gavrilets, 2000). Some authors claim that assortative mating alone is enough to produce reproductive isolation (Almeida and de Abreu, 2003).

Until recently, it was believed that the traits of assortative mating and disruptive selection should be associated in a non-random way (linkage equilibrium) (Felsenstein, 1981). In this case the two traits are connected to the same genes. This type of sympatric speciation and its consequences for population dynamics are presented in the following two sections. However, models have shown that a species divides into two new ones even if these two traits are not linked (Kondrashov and Kondrashov, 1999; Dieckmann and Doebeli, 1999; Luz-Burgoa et al., 2003). Nevertheless, sympatric speciation seems to occur only under the conditions of strong selection and assortative mating. These mechanisms must be evolutionarily advantageous in order to

establish prezygotic isolation, and thus speciation is a rare process in Nature.

In most of the models, the influence of criticality on the macroscopic behaviour of a population during the process of sympatric speciation has been neglected, by the use of mean–field models or by making simulations with small population sizes (around $1,000$ individuals). The role of stochasticity in such a scenario should be taken into account as done in Sections 3.2 and 3.3 (published in Luz-Burgoa et al. (2006) and Schwämmle et al. (2005a), respectively).

Speciation obviously does not only occur in the previously presented extreme cases of complete geographic isolation (allopatry) or arbitrarily interacting populations (sympatry), but also in intermediate situations, where the population distributes over a geographic range and remains in contact (for a review see Gavrilets (2004) and Coyne and Orr (2004)). The area of contact between two genetically different populations of the same species is called hybrid zone. Different environments like a continuous gradient of changing food, altitude, or other external conditions, can lead to the adaptation of a species to particular and distinct values of these resources (Slatkin, 1973; Endler, 1973; Kirkpatrick and Barton, 1997). This may or not result in speciation (Lande, 1982; Sanderson, 1989; Day, 2000). If intermediate values are selected against, as in the case of disruptive selection, the hybrid offspring of two differently adapted individuals has lower fitness, and parapatric speciation is known to be possible. The conditions which lead to this type of speciation are still not well understood. Parapatric speciation seems to be possible as the outcome of additional assortative mating, as in the case of sympatry (Gavrilets, 2004), or even when random mating is preferred (Section 3.4 and Schwämmle et al. (2005b)).

Taking into account the here presented types of speciation, a simple dynamical model can be constructed in order to analyse the macro-evolution of a large number of species, which originate and become extinct according to the competition for resources among them. Such a model is presented in Section 3.5 (published in Schwämmle and Brigatti (2005)), and the resulting statistics are compared to the fossil record.

3.2 Phase transition in a computational model for sympatric speciation

As already explained, the branching of a single population into two or more species without prevention of gene flow through geographic segregation is known as sympatric speciation (Tregenza and Butlin, 1999; Mayr, 1963; Bush, 1969; Futuyma, 1979), the classical example in progress of which is the apple maggot fly *Rhagoletis pomonella* (Bush, 1969; Filchak et al., 2000; Feder et al., 2003). The theoretical models about the conditions which would facilitate sympatric speciation can be grouped according to two main ingredients (Via, 2001; van Doorn and Weissing, 2001): disruptive natural selection on characters associated with competition for resources (Kondrashov and Kondrashov, 1999; Dieckmann and Doebeli, 1999) and disruptive sexual selection (Higashi et al., 1999; Gavrilets, 2000).

In this section we investigate the sympatric speciation process with computer simulations. We present an individual-based model which assumes that competition for different resources and sexual selection are the dominant forces, acting on the population through the phenotypic characteristics of the individuals(Luz-Burgoa et al., 2006).

Sympatric speciation is obtained as we tune up the strength of competition X between individuals with different phenotypes. As a function of this control parameter, we can characterise, through the behaviour of the order parameter - the density of selective females $\overline{\rho_s}$ - a phase transition from a non-speciation to a speciation state of the system. The first derivative of the order parameter with respect to the control parameter is consistent with a phase transition behaviour by exhibiting a sharp peak at the transition point. For different resources distribution, σ_k, the transition point is shifted and it is then possible to map out the phase diagram of the system in the $\sigma_k \overline{\rho_s} X$ space. The inverse of the parameter related to sexual selection strength, the latter being in our case the number of mating choices per female, behaves like an external field in the system and, as thus, is also a control parameter. To sum up, we found out that the macroscopic effects of the biological parameters used in our model reveal fingerprints typical of thermodynamic quantities in a phase transition of an equilibrium physical system.

3.2.1 Introduction

Herbivorous insects have long been considered prime candidates for sympatric speciation because of an intimate and frequently highly specialised relationship with their host plants (which serve as habitat, food resource, and, often, mating location) (Drès and Mallet, 2002). The apple maggot fly *Rhagoletis pomonella* has been consid-

ered, since 1966, as the classical example of sympatric speciation in progress (Bush, 1969). *R. pomonella* shifted from utilising the unabscised fruit of its native host hawthorn (*Crataegus spp.*) to utilising the introduced, domesticated apple (*Malus pumila*) sometime in the mid-1800s in the Hudson River Valley region of the state of New York. Evidence suggests that the species is in the process of shifting and adapting to this new host plant (Filchak et al., 2000; Feder et al., 2003).

To simulate and study the sympatric speciation process we use the Penna model (Penna, 1995). The first simulations (Sá Martins et al., 2001) performed with this same purpose considered a population where a single phenotypic characteristic was related to both assortative mating and competition for resources. Inspired by effects of the El Niño on global climate, an abrupt ecological change was used, as in Kondrashov and Kondrashov (1999), to provoke disruptive selection, which led to speciation through the development of assortative mating. This ecological change was numerically introduced by suddenly modifying the carrying capacity of the environment, that appears in the Verhulst factor, from a constant value to one that depends on the individual's phenotype. Using the same computational strategy of Sá Martins et al. (2001), simulations were also performed considering individuals with two independent phenotypic traits (one for competition and the other for assortative mating) (Luz-Burgoa et al., 2003; Moss de Oliveira et al., 2004), obtaining speciation as well.

A different strategy was used to simulate sympatric speciation of predators in a food web (Luz-Burgoa et al., 2005) of three species: a basal resource, herbivores and their predators. In this case, three types of intra-specific competition were adopted, depending on the phenotypic group of the predators, and their strength was kept constant during the whole simulation, while the carrying capacity still changed abruptly at some point in time, simulating an abrupt change of the ecology. In particular, a constant parameter X was introduced, establishing the fraction of the populations of extreme phenotypic predators which the intermediate phenotypic individuals would compete with, besides competing among themselves.

In the present model we adopt the same kind of intra-specific competition depending on the individuals phenotypes, but varying the value of X. Also no abrupt change in the ecology is considered. We show that the competition strength X plays the role of a control parameter in a phase transition, and that the fraction of sexual selective females in the population shows a behaviour similar to an order parameter.

3.2.2 Model

The sympatric speciation model

In this model competition for food and assortative mating are related to the same phenotypic trait, as for instance, the individual's size. This trait is represented by a new pair of non age-structured bit-strings, added to the original structured one, which suffers the same process of crossing and recombination described in the previous chapter (see Figure 3.3). The phenotypic characteristic is measured by counting, in this new pair of bit-strings, the number of bit positions where both bits are set to 1, plus the number of dominant positions (chosen as $D_p = 16$) with at least one of the two bits set. It will therefore be a number k between 0 and 32, which we will refer to as the individual's phenotype. We call M_p the mutation probability per locus of this phenotypic trait. Differently from the age-structured part of the genome, mutations in this new portion can occur in both directions: $0 \rightarrow 1$ and $1 \rightarrow 0$.

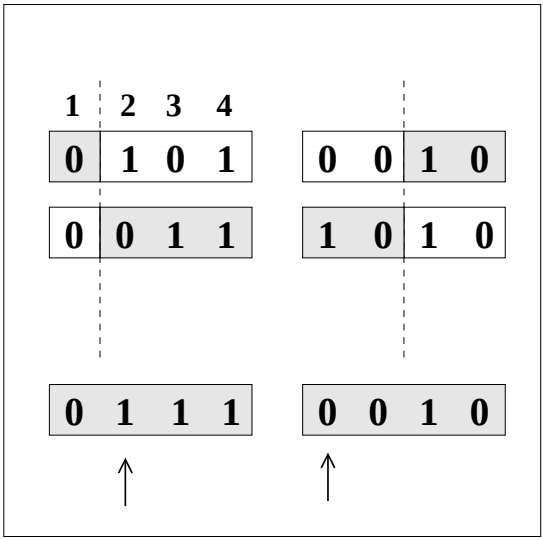


Figure 3.3: Schematic representation of gamete formation in the case where each genome is represented by two pairs of bit-strings. The first pair is age-structured as in the original Penna model while the second one is non-structured and codes for a phenotypic trait like the individual's size. For the non-structured pair mutations can occur in both directions.

In order to consider intra-specific competition depending on the individual's pheno-

type k, we modified the logistic Verhulst factor, originally introduced to avoid unlimited population growth, introducing three intra-specific competition terms, each one related to a given phenotypic group:

$$V(k,t) = \begin{cases} V_1(k,t), & 0 \leq k < n_1; \quad \text{extreme phenotypes.} \\ V_m(k,t), & n_1 \leq k \leq n_2; \quad \text{intermediate phenotypes.} \\ V_2(k,t), & n_2 < k \leq 32; \quad \text{extreme phenotypes.} \end{cases} \tag{3.1}$$

As in the original Penna model, at every time step, and for each individual, a random real number uniformly distributed between 0 and 1 is generated; if this number is smaller than $V(k,t)$, the individual dies. For the extreme phenotype groups the competition is given by:

$$V_{1(2)}(k,t) = \frac{P_{1(2)}(k,t) + P_m(k,t)}{F}, \tag{3.2}$$

where $P_{1(2)}(k,t)$ accounts for the population with phenotype $k < n_1$ ($k > n_2$) at time t, respectively, and $P_m(k,t)$ accounts for the population with phenotype $k \in [n_1, n_2]$. F is a constant proportional to the carrying capacity. The Verhulst factor for intermediate (m) phenotypes is:

$$V_m(k,t) = \frac{P_m(k,t) + [P_1(k,t) + P_2(k,t)] * X}{F}, \tag{3.3}$$

where X is the strength of competition between intermediate and extreme phenotypic populations. Eq. (3.2) means that individuals with extreme phenotypes (P_1, P_2) compete with those belonging to the same phenotypic group and also with the whole intermediate population, but there is no competition between extreme phenotypes of different groups because we are assuming they are specialised to some extent ($[0, n_1]$,$[n_2, 32]$) on particular resources F. Individuals with intermediate phenotypes (P_m) compete among themselves and also with a fraction X of each population presenting an extreme phenotype (eq. 3.3).

In order to consider assortative mating, we introduce into each female genome a single locus (bit) that codes for this selectiveness, also obeying the general rules of the Penna model for genetic heritage and mutation. If it is set to 0, the female is not selective in mating (random mating). It is selective (assortative mating) if this locus is set to 1. We call M_S the mutation probability for this locus, which can be in both directions ($0 \rightleftarrows 1$). We start the simulations with all females set non-selective. Mutated females that are born selective choose mating partners according to the following mating strategy: If a female has phenotype $k < 16$ ($k > 16$), it chooses, among N_m males, the one with the smallest (largest) phenotype value k; If a selective female has $k = 16$ then it chooses randomly to act as one of the above. Notice that with this strategy all females, that reach the minimum reproduction age R, reproduce every time step from R until death.

Threshold for bad mutations	$T = 3$
Mutation rate	$M = 1$
Dominant positions	$D = 3$
Minimum reproduction age	$R = 10$
Number of males available for mating choice	$N_m = 50$
Birth rate	$b = 2$
Mutation probability in phenotypes	$M_P = 0.01$
Dominant positions	$D_P = 16$
Selectiveness mutation probability	$M_S = 0.001$
Range of intermediate phenotypes	$k \in [n_1 = 13, n_2 = 19]$
Carrying capacity	$F = 2 \times 10^5$

Table 3.1: Parameters for the age-structured pair of bit-strings (upper part, traditional Penna model) and for the non structured one (lower part). For all figures the total number of time steps in the simulation runs was $t = 4 \times 10^4$.

3.2.3 Results

Sympatric speciation in a constant carrying capacity

At the beginning of the simulations females are non-selective and all the 6×10^3 individuals (half males and half females) have a random phenotype. At every time step of the simulation we measure the density of selective females, that is, the ratio between selective females and the total number of females. We calculate the phenotype distributions of the population at three different moments, one at the very beginning, another in the middle and the last one at the end of the simulation. The values of the used parameters are shown in table 3.1.

For the parameter $X = 0$, the population with intermediate phenotypes does not compete with the extreme phenotypic ones (eq. 3.3) and, in fact, suffers less competition than the other two (eq. 3.2). In this case, nearly all females remain non-selective (see Fig. 3.4d, lower line) and the phenotype distribution corresponds to a stationary Gaussian function centred at $k = 16$ (Fig. 3.4a, full squares). That is, there is no sympatric speciation even if the carrying capacity for extreme phenotypes is not zero (Dieckmann and Doebeli, 1999). As opposed to the situation for $X = 0$, when we introduce a strong competition for the population with intermediate phenotypes, by setting, say, $X = 1.0$, only the individuals with extreme phenotypes $k = 0$ and $k = 32$ survive, as shown in Fig. 3.4b, full squares. In this case the density of selective females goes to $\rho_s \approx 1$ very fast, that is, the females with extreme phenotypes mate only with males of its same phenotypic group (see Fig. 3.4d, upper line). This means that there are two new sympatric species, reproductively isolated, even if the

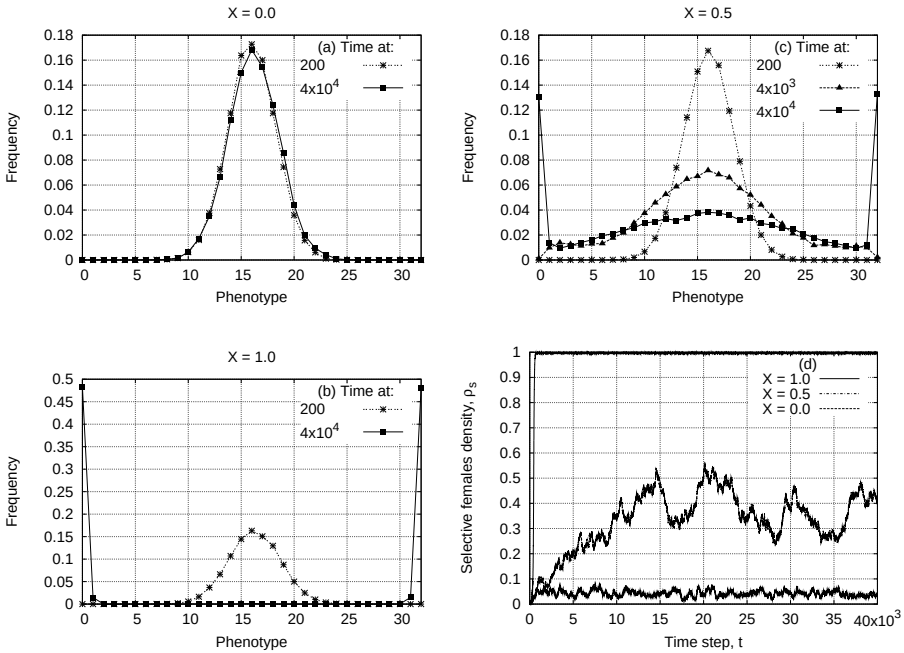


Figure 3.4: In a, b and c we show the phenotype distributions of the whole population for different strengths X of competition. During the initial steps of the simulation, $t = 200$, the distribution is, in all cases, a Gaussian centred at intermediate phenotypes. For (a) and (b), the distributions at $t = 4 \times 10^3$ are equal to those at $t = 4 \times 10^4$ and are stationary. For (c) the distribution is not unique neither at $t = 4 \times 10^3$ nor at $t = 4 \times 10^4$ (see text). In (d) we show the time behaviour of the density of selective females, ρ_s.

carrying capacity is not favouring the extremes more than the intermediate phenotypes (Kondrashov and Kondrashov, 1999). For the competition strength $X = 0.5$, the phenotype distribution is not unique: in runs that differ by the choice of the seed of the random number generator, the final distribution sometimes has one maximum at $k = 16$ (Fig. 3.4c, full triangles) and some other times it has two maxima at $k = 0$ and $k = 32$ (Fig. 3.4c, full squares). The time behaviour of the density of selective females presents large fluctuations (see Fig. 3.4d, central line).

Figure 3.4d shows an important change in the population organisation, from a non-speciation state with $\rho_s \approx 0$, to a sympatric speciation state with $\rho_s \approx 1$, as we increase the strength of competition, X, for the intermediate phenotypes. To deter-

mine the range of values of X for which sympatric speciation may be obtained, we will analyse the behaviour of the mean density of selective females, for many different strengths of competition.

Phase transition behaviour

For each value of X we carry out 10 simulations with the same parameters, but using different initial seeds for the random number generator. In each simulation we calculate the mean value of the density of selective females during the last 10^4 time steps, and then average the results of the ten runs.

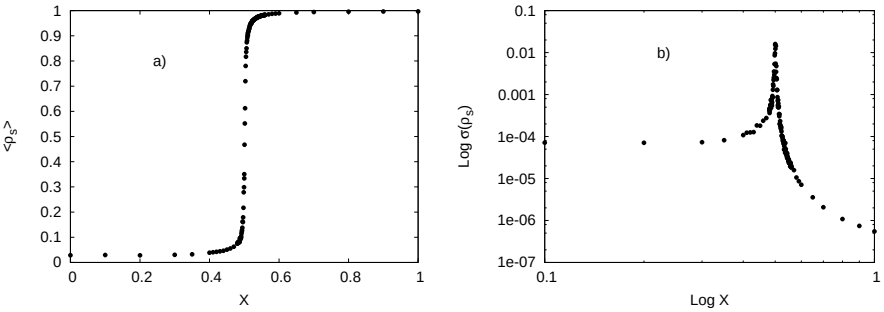


Figure 3.5: a) Mean values of the selective females density, $< \rho_s >$, the order parameter of the speciation transition, as function of the control parameter X, the strength of competition. b) Standard deviation of $< \rho_s >$ versus X in a logarithmic scale; the standard deviation of $< \rho_s >$ is proportional to the first order derivative of $< \rho_s >$.

The behaviour of the mean density $< \rho_s >$ as function of X is shown in Fig. 3.5(a). The population changes rather abruptly from a non-speciation to a speciation state when we change slightly the strength of competition, close to $X_c = 0.5$. Another fingerprint of the macroscopic effect, X on $< \rho_s >$, is the peak shown by the logarithm of the first derivative of the order parameter at X_c, Fig. 3.5(b). These behaviours are very similar to what happens to an order parameter as a function of the control parameter in an equilibrium phase transition of a physical system. This transition separates a single-species phase from one in which two species coexist in sympatry. In the single-species phase, Fig. 3.5(a) $X < X_c$, the population presents a high diversity with many different phenotypes in the population, see the squares in Fig. 3.4(a), and has a mean size of $\approx 25 \times 10^3$. In the two-species phase, Fig. 3.5(a) $X > X_c$, the mean size of the whole population is $\approx 50 \times 10^3$, or twice the value of the former phase, and the phenotypes in the population cluster around only two distinctively

separated values, Fig. 3.4(b) squares. In the Fig.3.5(b), the large values attained by $\sigma(\rho_s)$ just above X_c arise from large fluctuations in the number of individuals.

Fingerprint of disruptive selection in the phase transition

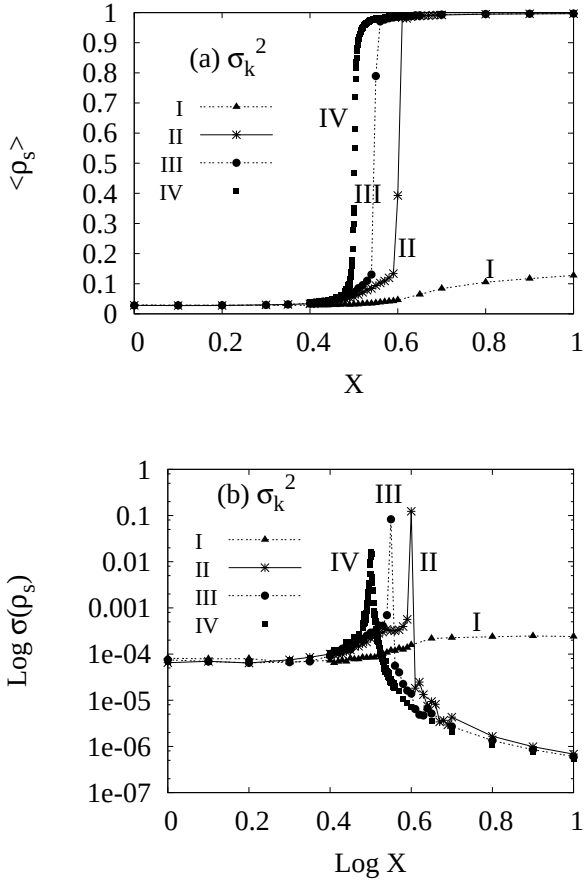


Figure 3.6: The figures show the effect of disruptive selection on the speciation transition. The resource distribution for I to III corresponds to F_{σ_k} with $\sigma_k^2 = 10^3, 5 \times 10^3, 10^4$, respectively. $F_{\sigma_k} = F$ corresponds to IV.

Ecological conditions have been considered an essential ingredient for divergence and speciation in sympatry (Orr and Smith, 1998). To evaluate its importance in a

phase transition context, we simulated different ecological conditions by modifying the width, σ_k, of the resource distribution of the environment, which has so far been considered as a constant F in the Eqs. (3.2) and (3.3). It will now be phenotype-dependent and will drive the population to experience a disruptive selection between the specialist and intermediate phenotypes. Its general functional form is: $F_{\sigma_k} = 2 \times 10^5 e^{-(k-16)^2/\sigma_k^2}$, where each individual, with phenotype k, will feed on a different resource niche: For small values of σ_k^2, the specialists will have fewer resources than individuals with intermediate phenotype.

In Fig. 3.6(a) the macroscopic effect of the width of the resource distribution is the shift suffered by the transition point, an effect similar to the shift of the transition point driven by changes in pressure for PVT systems. For small value of σ_k^2 and for $X > X_c$, case I in Figs. 3.6(a) and (b), the population prefers a non-speciation state, even in the presence of a high competition for intermediate phenotypes. This happens because there are not enough resources for two groups of specialists. It is nevertheless important to notice that the population has a large diversity in this case. That is, the phenotype distribution looks like Fig. 3.4(c) triangles, but it is a stable distribution, see Fig. 3.6(b) I.

Fingerprint of assortative mating in the phase transition

Sexual selection in the population is associated to the number of mating choices each female performs before reproduction, the parameter N_m in table 3.1. The probability of a selective female with phenotype $k < 16$ to mate with a male of opposite phenotype is $P_{<16} \approx (0.5)^{N_m}$. In the previous section, $N_m = 50$ and this probability is almost zero, meaning that the selective females are highly discriminatory against the opposite phenotype. With $N_m = 3$ the probability becomes $P_{<16} = 0.125$ and it is then possible for a selective female with $k < 16$ to mate with a male of phenotype $k > 16$.

When we reduce the number of mating choices per female, we can see that the phase transition is destroyed, Fig. 3.7. For an equilibrium physical system the phase transition disappears when there is an applied external field, as, for an example, happens to the paramagnetic transition of magnetic materials at the Curie point. In this example, the application of an external magnetic field drives a slight alignment of the localised atomic magnetic moments in its direction, which avoids the establishment of a true paramagnetic phase (Reichl, 1998).

A small value of the sexual selection strength, equivalent to the application of a magnetic field, produces an increase of the selective female density for $X < 0.5$ - see Fig. 3.7a (circles). This density approaches 0.5 since the difference between selective mating and random mating is small. For $X < 0.5$ and large values of sexual selection strength, $N_m = 50$ for example, the selective females density decreases because the

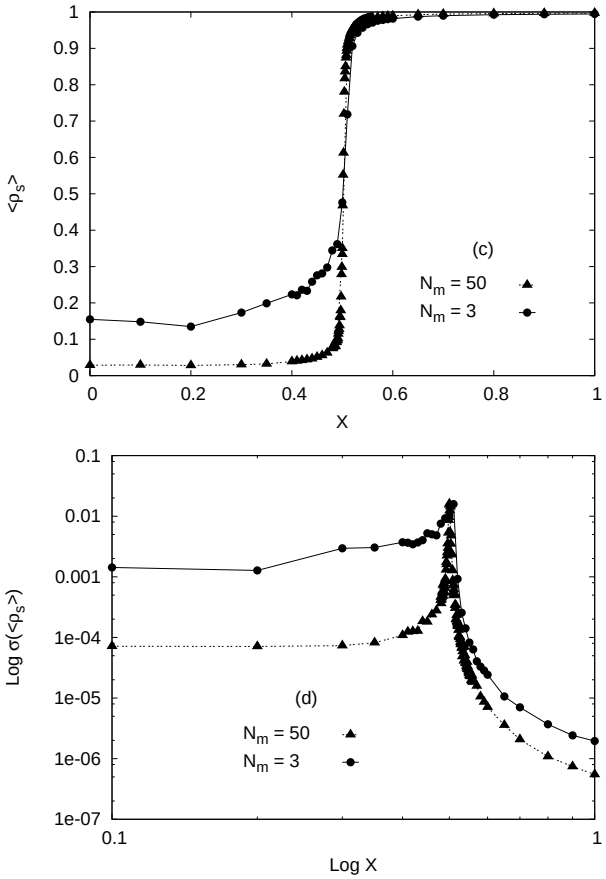


Figure 3.7: Determination of the effect of the sexual selection strength, N_m, in the phase transition context versus the strength of competition, X. It is important to point out that the simulation time for $N_m = 3$ was 8×10^5, 20 times bigger than in the other cases.

individuals with extreme phenotypes are in great disadvantage and selective females would have offspring with this kind of phenotype.

3.2.4 Conclusions

In conclusion, we report here an investigation of the macroscopic effects of the parameters (X, F_{σ_k} and N_m) on the origin of species by sympatric speciation, characterised by the behaviour of $< \rho_s >$, in a simple model. The behaviours of these biological parameters are similar to the thermodynamical parameters in a phase transition of a physical system. Furthermore, the analogies for X and F_{σ_k} are in qualitative agreement with other individual-based models that studied the necessary ecological conditions for sympatric speciation (Dieckmann and Doebeli, 1999; Kondrashov and Kondrashov, 1999). The analogy between N_m and an external field shows that this parameter acts as another control parameter, in addition to X and F_{σ_k}, since it plays a role analogous to an external field in a physical system. The meaning of this result is that sympatric speciation may be driven by changes in sexual selection strength alone, see Fig. 3.7a for $X \lessapprox X_c$ and $N_m = 3$. Again, this result is in qualitative agreement with sympatric speciation by sexual selection found e.g. in Refs. (Higashi et al., 1999; Gavrilets, 2000).

We emphasise that in our model the population was able to self-organise and eventually speciate without the need of two different resource distributions, for extreme and intermediate phenotypes, in contrast with Refs. (Kondrashov and Kondrashov, 1999; Sá Martins et al., 2001; Luz-Burgoa et al., 2005).

Phase transitions in physics have a large history and we believe these analogies between biological and physical parameters will help to understand the sympatric speciation universal behaviours and universal exponents mentioned in Section 1.4.2

3.3 Phase transition in a mean-field model for sympatric speciation

Here we present an analytical model for population dynamics with intra-specific competition, mutation and assortative mating as basic ingredients (Schwämmle et al., 2005a). The set of equations that describes the time evolution of the population size in a mean-field approximation may be decoupled. We find a phase transition in the sympatric speciation process when the parameter that quantifies the competition strength is varied. This transition, previously found in the computational model of Section 3.2, happens to be of first order.

3.3.1 Introduction

Until recently, the possibility of sympatric speciation was still under debate, but observations of micro-evolution (Friesen et al., 2004) and the development of theoretical frameworks (Lande, 1981; Kondrashov and Kondrashov, 1999; Chow et al., 2004) have established it as a valid conjecture in the last years, turning sympatric speciation into one of the favourite themes of research in modern evolutionary theory (Turelli et al., 2001; Gavrilets, 2004; Coyne and Orr, 2004).

A variety of theoretical models have been proposed to explain sympatric speciation, from analytical mean-field type ones to more realistic individual-based models. Computational representations based on variations of the Penna model for biological ageing (Penna, 1995), popular amongst physicists working on the statistical mechanical aspects of evolutionary theory, belong to this latter class. Previous work on such representations have shown that sympatric speciation appears when driven by a change in the character of the distribution of ecological resources, as suggested by some biologists (Kondrashov and Kondrashov, 1999). From this perspective, sympatric speciation appears as a transition between two different organisations of some population. In the present work, we develop a variation that allows a mean-field approximation with analytical solution, in which the nature of this transition may be further discussed.

The computational model (Section 3.2) has intra-specific competition, mutations and assortative mating as its sole ingredients. It's mean-field approximation leads to a set of simple equations that reproduces some of the features of individual-based models, and whose solutions show a clear signature of the above mentioned phase transition.

3.3.2 The computational model - a brief review

We take as starting point the sexual version of the Penna model, as described for instance in refs. (Moss de Oliveira et al., 2000, 1999b) and in Section 2.3. In addition to the age-structured pair of bit-strings that represents the genome for purposes of ageing analysis, each individual carries an extra pair of non-structured bit-strings of 32 bits each, that encodes a genetically acquired phenotype trait, as already published in ref. (Sá Martins et al., 2001). This extra pair of genetic material is inherited with the same dynamics of the age-structured pair, involving a meiotic cycle with crossing and recombination of each parent's bit-strings. The trait for a particular individual is obtained by counting the number of loci in the non-structured pair where the allele 1 is either homozygous or dominant, and is an integer in the interval $[0, 32]$ which determines the individual's survival probability and its mating preferences. The positions where the allele 1 is dominant are chosen randomly at the beginning of the simulation and are the same for all individuals. According to this number, the population is divided into three groups (subpopulations). We will follow the dynamical evolution of the size of the three subpopulations independently: P_1 for the one with small values of the phenotypic trait, P_2 for the one with large values, and P_i for the intermediate one (called P_m in 3.2). The survival probability is $1 - V$, where V is the so-called (modified) Verhulst factor. This factor has a resource-size parameter, the carrying capacity C (called F in Section 3.2), and represents a mean-field competition for the ecological resources of the environment. It has a different value for each one of the three subpopulations, representing different levels of competition for those resources:

$$V_{P_{1,2}} = \frac{P_{1,2} + P_i}{C}, \;\; V_{P_i} = \frac{X(P_1 + P_2) + P_i}{C} \tag{3.4}$$

where we set $C = 100,000$. The intermediate subpopulation P_i competes with a fraction X of the sum of the subpopulations with extreme values of the phenotypic trait, and this fraction will drive the speciation phase transition. Each of the subpopulations 1 and 2 competes only with itself and with the intermediate one. This variation of the Verhulst factor has previously been used in a study of sympatric speciation in food webs (Luz-Burgoa et al., 2005). A genetic trait, encoded by a single bit and subject to mutation, determines female selectivity in mating. This trait is initially set to zero: every female selects a mating partner randomly. Observe that due to mutations the offspring of a selective female may be non-selective and vice-versa. Mating preference also depends on the value of the phenotype trait. A selective female of population P_1 or P_2 chooses to mate, among a set of A (called N_m in Section 3.2) males from its own subpopulation, the one with the most extreme value of the phenotypic trait. A selective female of population P_i chooses randomly to act as one of the above. Any non-selective female mates randomly. The number A of available

males is a measure of the female's selectivity degree: the larger A, more selective is the female.

3.3.3 Results of the computational model

We focus on the identification of the phase transition already mentioned. Fig. 3.8 compares the final states of simulations carried out with extreme values of X with the one at the transition point $X = 0.5$. In all cases, $A = 50$, the mutation probability at birth of a locus of the phenotypic trait is 0.01, and the total time equals $100,000$ MC steps. Speciation is well observed for $X = 1$, where complete reproductive isolation leads to the absence of gene flux between the extreme populations and consequently to the extinction of intermediate phenotypes. Genetic variety and gene flux increases crucially at the transition point $X = 0.5$. The signature of the transition is the abrupt change of the fraction of selective females in the population, N_s (called ρ_s in Section 3.2), which acts as an order parameter. In a single species environment, this fraction is close to 0, and it increases to 1 with the event of speciation (in fact, $1 - N_s$ would provide a better order parameter with $1 - N_s = 0$ for speciation). As the control parameter X is increased from 0, the order parameter N_s undergoes a clear transition, as seen in fig. 3.5 of Section 3.2. A similar transition also occurs if a selective female chooses the mating partner that most closely matches her own phenotype trait, instead of the most extreme one (Luz-Burgoa et al., 2003), showing that the extreme mating strategy is not crucial.

At the transition point the fluctuation of N_s, measured as the mean deviation of multiple realizations of the simulations, presents a peak. As the selectiveness parameter A is increased, so does the steepness of the transition. The transition can also be seen in the behaviour of P_1, P_i, $P_1 + P_2 + P_i = P_{total}$, and $P_1 - P_i$. In fact, we will use this last quantity to signal the occurrence of speciation, as commented below.

3.3.4 Mean-field approximation

A mean-field approximation to the microscopic computational model can be cast in the form of a system of coupled differential equations. Dynamics of all three subpopulations are frequency-dependent; for the intermediate subpopulation, we add a competition for ecological resources with a fraction x of each of the extreme-sized ones, as done in the microscopic model. The system of equations is thus:

$$\frac{dP_1}{dt} = (a - b)P_1 + bP_i - \frac{1}{C}(P_1 + P_i)P_1 \tag{3.5}$$

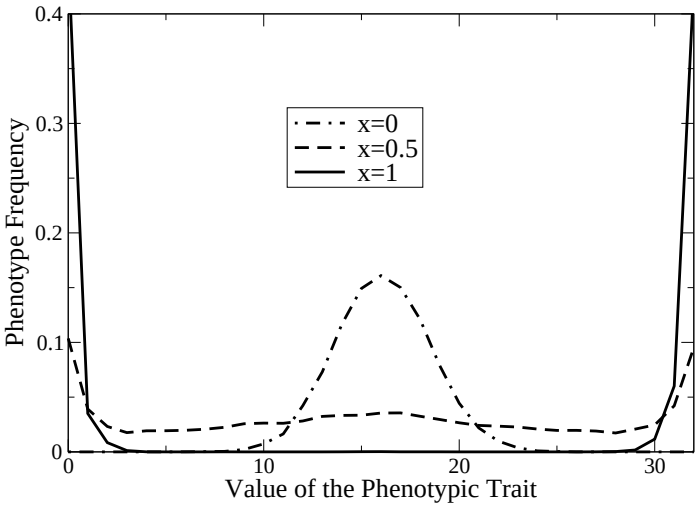


Figure 3.8: The frequency of individuals as a function of the value of the phenotypic trait in the final state of the simulations, for some values of the competition degree X.

$$\frac{dP_2}{dt} = (a-b)P_2 + bP_i - \frac{1}{C}(P_2 + P_i)P_2 \tag{3.6}$$

$$\frac{dP_i}{dt} = (a-2b)P_i + bP_1 + bP_2 - \frac{1}{C}(xP_1 + xP_2 + P_i)P_i \tag{3.7}$$

The parameter a describes the birth rate, and is the same for all subpopulations. In order to characterise the exchange parameter b, we have to envision the two processes crossing–over and mutation. In the first, the bits of the phenotypic trait are reshuffled by crossing-over of the gametes of both parents, a process that can lead to drastic changes in the value of this trait. The amount of this change is controlled by the number of males (A) each female will choose from a mating partner, as well as by the number of selective females in the population. Additionally, mutations may change the number that characterises an individual's trait. This last process is independent from the reshuffling and thus can be described by a constant value. These two processes are rather difficult to include in a mean-field model. For simplicity, we chose to model them by postulating that each subpopulation with extreme phenotype

generates a fraction b of its offspring with a phenotype of the intermediate subpopulation. The latter looses a fraction of $2b$ offspring to the extreme subpopulations. The parameter b synthesises the combined effect of the mutation rate and of the degree of assortativity in the mating process, which should be proportional to the reciprocal value of A. Thus, even if assortative mating is maximal, the parameter b cannot be set to zero, since we still have to model the effect of mutations. We will refer to b as a parameter of female selectivity in the following, keeping in mind that it also contains the effect of mutations. Competition is introduced through the density-dependent Verhulst factor, to which the parameter for the carrying capacity C is related.

Because of the intrinsic symmetry of the model, the subpopulation with a small value for the phenotype trait (P_1) has a dynamical evolution equivalent to the one with a high value for the trait (P_2). In each of these, the time evolution of its size depends on its current value as well as on the size of the intermediate subpopulation. We assume, as an approximation, that they are equal at all times, and set $P_1 = P_2$ in eq. (3.5):

$$\frac{dP_i}{dt} = (a - 2b)P_i + 2bP_1 - \frac{1}{C}(2xP_1 + P_i)P_i. \tag{3.8}$$

The system of eqs. (3.5) and (3.8) can be simplified by the following transformations:

$$f = \frac{1}{4C}(P_1 + P_i);\ g = \frac{1}{4C}(P_1 - P_i);\ \epsilon = 2x - 1, \tag{3.9}$$

ϵ being a control parameter in terms of which the transition is set at $\epsilon = 0$. As a result of this transformation, the system of equations now reads:

$$\frac{df}{dt} = af + bg - (\epsilon + 4)f^2 + \epsilon g^2, \tag{3.10}$$

$$\frac{dg}{dt} = (a - 3b)g - \epsilon g^2 - 4fg + \epsilon f^2. \tag{3.11}$$

To look for characteristics of the stationary solutions, which are the fixed points of the differential system, we set the time derivatives to 0 and obtain a relation between the functions g and f:

$$g = -f\frac{4f - a}{4f - a + 2b} \tag{3.12}$$

The existence of the phase transition in the mean-field approximation is clear in fig. 3.9, in which the exact and stable solutions for the size of the subpopulations are

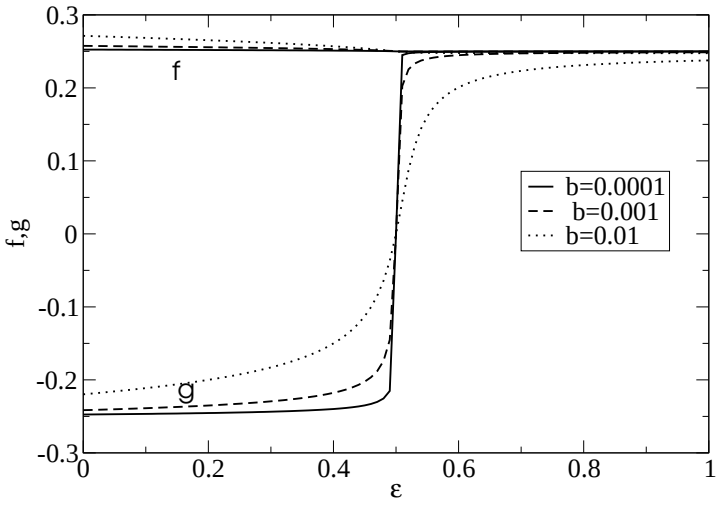


Figure 3.9: The stable fixed points of the mean-field model for different values of b, characterising female selectivity. When b increases, the transition becomes smoother.

shown as a function of the new control parameter ϵ. The transition point $\epsilon = 0$ separates a region in phase space where the subpopulations still interbreed ($\epsilon < 0$) and the intermediate population is large, from another one, in which the subpopulation that results from interbreeding all but vanishes. The latter characterises assortative mating within each of the subpopulations. These can now be said to constitute two different non-mating species.

Exploration of the parameter space shows that this transition becomes smoother as the selectivity degree parameter b increases, as shown in fig. 3.9. In all cases, the function f is nearly constant (compared in fig. 3.10 to the computational model), as can be seen directly from the results of the simulations of the computational model. The latter results also show fluctuations in the values of all subpopulations, as well as in g, that diverge as the transition point is approached. This is not true for f though, for which the fluctuations are small and do not show any change of behaviour at the transition. The role played by the female selectivity A is equivalent to the corresponding parameter in the mean-field approximation, b: an increase in selectivity A, corresponding to a decrease in b, sharpens the transition.

In order to obtain a full characterisation of the transition, and supported by the results

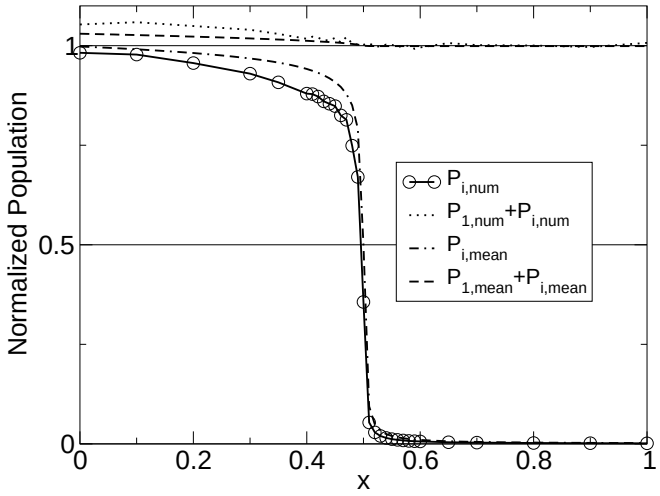


Figure 3.10: Comparison of the result for $P_{i,num}$ of the computational model ($A = 50$) with the mean-field approximation $P_{i,mean}$, fitted with $b = 0.001$. The quantity $f \propto P_1 + P_i$ is nearly constant. The horizontal full straight lines are guides to the eyes.

of the simulations, we impose f to be some constant from the start, and independent of ϵ. Additionally, we neglect the term bg in eq. (3.10). The relations $f > 0$, $f > g$ as well as $b \ll a$, valid in all cases we have studied, justify this simplification. It is easy to compute the value of f from the differential system at the transition point $\epsilon = 0$, $f = \frac{a}{4}$. f can be set constant also because of the following reason: If we add a small perturbation δ to f in eq. 3.12 we obtain $g = -\delta\frac{a}{2b}$ with $b \ll a$. We are also left with a single differential equation for g,

$$\frac{dg}{dt} = -\epsilon g^2 - 3bg + \epsilon\frac{a^2}{16} \tag{3.13}$$

with a stationary solution that exhibits symmetry with respect to the transition point:

$$g_* = -\frac{3b}{2\epsilon} + \sqrt{(\frac{3b}{2\epsilon})^2 + (\frac{a}{4})^2}. \tag{3.14}$$

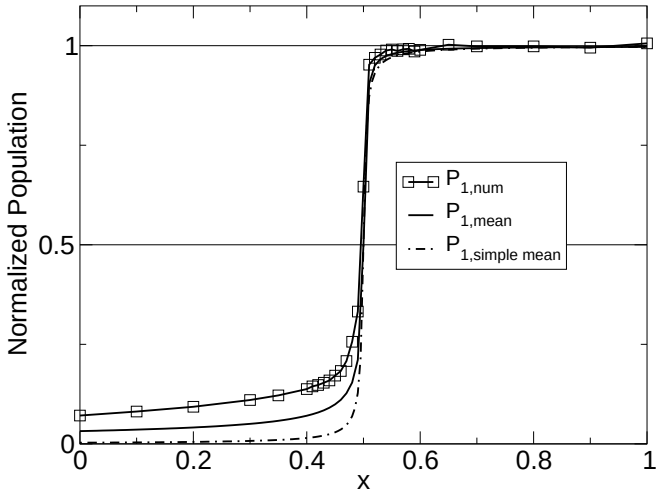


Figure 3.11: Comparison of the result for $P_{1,num}$ of the computational model ($A = 50$) with the mean-field approximations $P_{1,mean}$, fitted with $b = 0.001$. The simplified mean-field model $P_{1,simple\,mean}$ also reproduces well the simulations.

To establish the validity of these last approximations, we may compare the above result with the stable solution of the full mean-field solution. Fig. 3.11 shows both results, and compares them to the numerical results. Deviations do exist, but they do not change the qualitative nature of the solutions.

We now proceed to obtain the time dependence of the solution. This behaviour could be interpreted as if, after reaching the stationary state, the value of the control parameter was changed; the solution then approaches a new equilibrium. This can be done more easily if we apply the transformation $z = \frac{1}{g-g_*}$ to get the equation for $z(t)$,

$$\frac{dz}{dt} = \epsilon + \sqrt{9b^2 + \epsilon^2 \frac{a^2}{4}} \cdot z = \epsilon + rz \tag{3.15}$$

with a time-dependent solution for $g(t)$ given by

$$g(t) = -\frac{r}{\epsilon} \cdot \frac{1}{1 - \phi e^{rt}} + g_* \tag{3.16}$$

The parameter ϕ depends on the initial condition. If it has a value in the interval $0 < \phi < 1$, then $g(0) < g - 2f = -P_1 - 3P_i$, which lies beyond the range of g. The analytical solution is a very good approximation to the result of the simulations, yielding the same exponential behaviour.

If we impose the condition that f has a constant value, still at the transition point $\epsilon = 0$, then the solution for $g(t)$ is $g(t) = g_0 e^{-3bt}$, whereas the full solution, neglecting only the term bg is given by

$$f(t) = \frac{a}{\beta e^{-at} + 4}, \; g(t) = g_0 e^{-3bt} \frac{1}{\beta e^{-at} + 4} \tag{3.17}$$

where g_0 and β depend on the initial conditions. Unfortunately, the exponential decay we obtain in the analytical solution does not allow us to determine a precise relation between the selective strength parameters of the microscopic and mean-field models, due to too high a level of fluctuations.

3.3.5 Conclusions

The microscopic models for the study of sympatric speciation, and in particular those based on variations of the Penna model, have proven their value yielding a number of interesting results and providing some background for a testing ground of evolutionary theories. We have here reported that one of those versions admits a mean-field approximation with an analytical solution that closely matches the results of the simulations. This new tool can be helpful in the characterisation of sympatric speciation as an out-of-equilibrium phase transition, and help in the study of the statistical properties of the system on both sides of that transition.

3.4 Monte Carlo simulations of parapatric speciation

Parapatric speciation is studied using an individual–based model with sexual reproduction (Schwämmle et al., 2005b). We combine the theory of mutation accumulation for biological ageing with an environmental selection pressure that varies according to the individuals' geographical positions and phenotypic traits. Fluctuations and genetic diversity of large populations are crucial ingredients to model the features of evolutionary branching and are intrinsic properties of the model. Its implementation on a spatial lattice gives interesting insights into the population dynamics of speciation on a geographical landscape and the disruptive selection that leads to the divergence of phenotypes. Our results suggest that assortative mating is not an obligatory ingredient to obtain speciation in large populations at low gene flow.

3.4.1 Introduction

Here we modify the Penna model discussed in Chapter 2, Penna (1995), Moss de Oliveira et al. (1999b) and Stauffer et al. (2001) and include an environmental selection pressure that, besides acting according to individuals phenotypes, also varies according to their positions on a spatial lattice. Using this strategy we study under which conditions parapatric speciation happens and observe that it depends strongly on the fluctuations of the system, as already obtained in previous simulations of sympatric speciation (Luz-Burgoa et al., 2003; Sá Martins et al., 2001). The connection of the individual deaths with their phenotypic traits and lattice positions through a simple function is shown to produce a complex behaviour of the whole population, that may or may not yield speciation.

Our implementation of the sexual Penna model with a phenotypic trait on a spatial lattice is based on Sousa (2004) and Luz-Burgoa et al. (2003). We succeed in reproducing qualitatively the results of Gavrilets (1997), although the effect of sexual selection in our model is shown to be so weak that it can be neglected.

In the next section we explain our model, and in Section 3.4.3 we present the results. In Section 3.4.4 we discuss some relevant aspects of the model and Section 3.4.5 contains the conclusions.

3.4.2 Model

The age–structured part of the genomes is modelled by the sexual version of the Penna model described in Section 2.3.

Phenotypic Trait, Spatial Lattice and Ecology.

Similar to the model of Section 3.2, a second pair of bit-strings of each genome is translated into some phenotypic characteristic of the individual. We call the effective number of bits 1, taking into account the dominance, the phenotype number n, which is an integer between zero and 32. For example, we may consider that small values of n correspond to small sized individuals, while large values of n denote big ones.

The individuals are distributed on a two dimensional square lattice. They move at every iteration, with a rate m_m, to a randomly chosen less or equally populated nearest neighbouring site. If all nearest neighbours sites are more populated than the current individual's site, the movement is not carried out. This strategy guarantees a fast and balanced distribution of individuals over the whole landscape. The reproductive females select their mating partners randomly from the reproductive males localised at the same or at a nearest neighbour site. Reproduction between different phenotypes is not forbidden. Offspring are distributed into empty nearest neighbouring sites. If there is no empty site, offspring is not produced. In this way the population size is controlled by the size of the lattice (Makowiec, 2001), and there is no need to use the random killing Verhulst factor, present in the traditional version of the Penna model to avoid unlimited population growth.

The interaction between phenotypic trait and geographical position on a square lattice of linear size L is given by:

$$E(x,n) = S \cdot \left(1 - \left| g(x) - \frac{n}{32} \right| \right), \tag{3.18}$$

which we call the ecological function. It gives the probability of an individual dying, at an iteration, depending on its x-position and phenotype number. The parameter S is the strength of the interaction and varies between zero and one. The larger the value of S, the stronger is the selection pressure acting on the individuals. The coordinate function is given by $g(x) = \frac{x}{L-1}$, where the coordinate x is an integer between zero and $L-1$. For extreme phenotypes with $n = 0$, the ideal region in which to live corresponds to $x = L-1$ where $E(L-1, 0) = 0$, while for extreme phenotypes with $n = 32$ the ideal region corresponds to $x = 0$. Individuals with intermediate phenotypes also live better at the extremes of the lattice, but are less fit than those with extreme phenotypes living in the ideal extreme of the lattice. Figure 3.12 illustrates the ecological function behaviour for three different values of n.

3.4.3 Results

In this section we describe the relevant features of speciation found with our simulations, that is, we focus on the interaction between phenotypic trait and the lattice. For

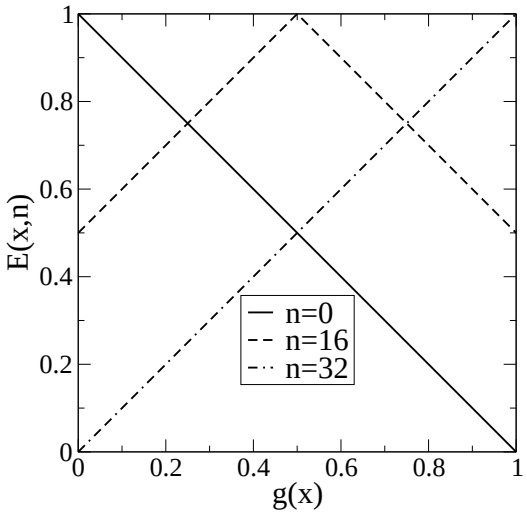


Figure 3.12: Behaviour of the ecological function (or probability to die according to x-position and phenotype) $E(x, n)$. Individuals with high or low n survive better on opposite sides of the lattice whereas the ones with intermediate phenotype numbers have a higher death probability everywhere.

results of the traditional sexual Penna model with Verhulst factor or on a lattice we refer to Moss de Oliveira et al. (1999b) and Makowiec (2001), respectively.

The fixed parameters that we adopt in the simulations are:

i) Threshold number of genetic diseases $T = 3$;

ii) Minimum reproductive age $R = 8$;

iii) Birth rate $b = 4$;

iv) Rate of bad mutations in the chronological genome $m = 1$;

v) Number of dominant positions in the chronological genome $D = 5$.

vi) Mutation rate of the phenotypic trait $m_p = 0.15$ or $m_p = 0.2$.

vii) Number of dominant positions in the phenotypic trait $D_p = 16$.

The relevant parameters for speciation are the mobility m_m, the lattice size L and the strength S of the environmental pressure.

We start the simulations with all the genomes randomly filled with zeroes and ones, and all individuals randomly distributed on the lattice. In order to reach a genetically stable initial population, we run the simulations without any ecological function for 1,000 iterations. During this period the dynamics of the population is neither affected by the phenotype numbers nor by the lattice positions of the individuals. The initial distribution of the phenotype numbers is regulated solely by the mutations, and shows a Gaussian behaviour (central curve of Fig. 3.13).

After these transient steps, the ecology is abruptly changed by setting the ecological function as an additional death probability. Disruptive selection driven by the ecology leads to a better survival of individuals with high and low phenotype numbers, depending on their current positions on the lattice. Three different situations, described below, can be observed, where the environmental pressure and the mobility are the crucial parameters.

i) At low selection pressures (S small), and independently of the mobility, the distribution of the phenotype numbers remains unaltered (Gaussian). The population decreases slightly at intermediate positions on the x–direction, but during the entire simulation individuals stay in contact over the whole lattice. Gene flow prevents disruptive selection from dividing the system into two sub-populations.

ii) For intermediate selection pressures and mobilities ($m_m \sim 1.0$), shortly after turning disruptive selection on, the system reaches an extremely dynamical state where fluctuations may or may not drive the system to divergence. In the cases where speciation does not occur, the adaptation of the phenotypes on one of the lattice sides is faster and gene flow forces the individuals on the other side to adapt themselves to the opposite phenotype (dashed-line with stars in Fig. 3.13).

When phenotypic adaption is balanced, the distribution of phenotype numbers bifurcates. Figure 3.13 shows that even in the case of speciation, the phenotypic distribution usually drifts away from symmetry before bifurcating, but the final and stable state corresponds to two populations with different phenotypes. We emphasise that during the speciation process the whole population stays in contact and gene flow can not be neglected as in allopatric speciation.

Figure 3.14 shows the typical spatial distributions of the phenotypes at four different moments of the simulations. Initially, the population is homogeneously distributed over the whole lattice. As soon as the new ecology is turned on, almost all individuals occupying the central x-positions of the lattice die, and the population becomes temporarily divided into two similar groups, with weak contact between them. When

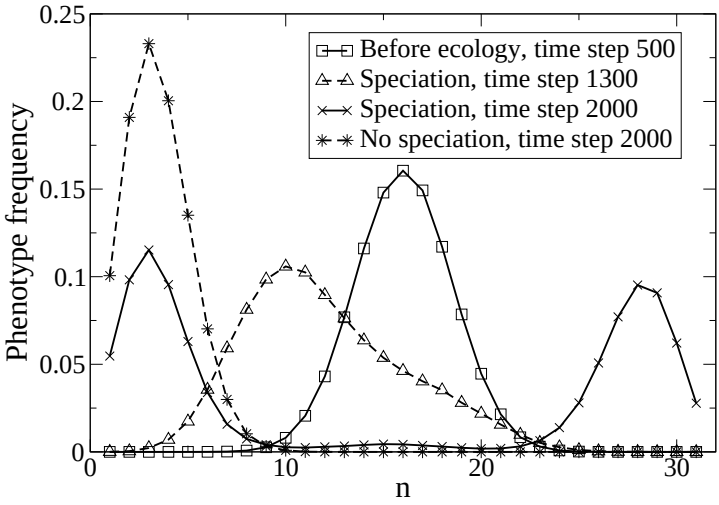


Figure 3.13: The evolution of phenotypic frequency (given by the second pair of bit-strings) versus time. The same set of parameters may or not yield speciation for different random seeds. The central curve (solid line, squares) corresponds to the distribution of phenotype numbers before switching on the ecological function (t = 1,000 time-steps). The final distribution when speciation occurs is given by the double-peaked distribution (solid line, crosses). The dashed line with stars corresponds to the final distribution for a case where speciation has not occurred; the dashed line with triangles shows the intermediary distribution in the course of speciation. The parameters: $S = 0.24$, $m_m = 0.99$ and $m_p = 0.2$.

the adaptation process of the extreme phenotypes starts, offspring with intermediate phenotype numbers continue to be produced. As the adaptation proceeds, competition with the more fit extreme phenotypes makes the intermediate ones disappear. Finally, when speciation occurs, each half of the lattice becomes mostly occupied by one of the two extreme phenotypes, respectively. The number of iterations needed to reach the final distribution is about 5000, which corresponds to 625 generations. However, we would like to emphasise that we have run our simulations for up to 100.000 time-steps, to be sure we were obtaining stable distributions.

The final result of a simulation where no speciation occurred, using the same parameters as in Figure 3.14 but with another initial random seed, is illustrated in Figure 3.15.

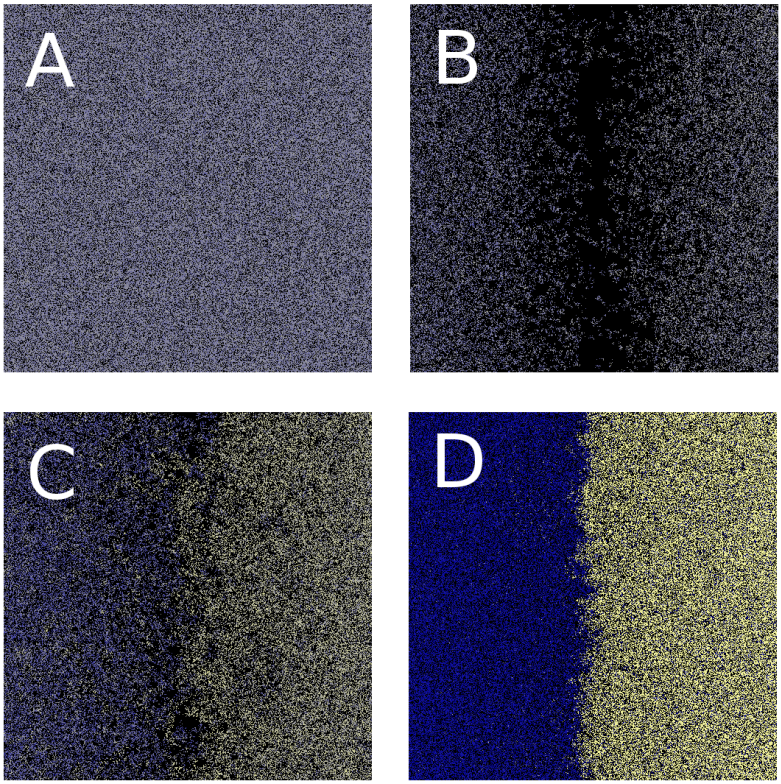


Figure 3.14: Illustration of the phenotype distribution on a 500×500 lattice. The parameters are $m_m = 0.9$, $S = 0.25$ and $m_p = 0.1$. Black sites are empty. The colours indicate the average value of the phenotype numbers (between yellow and blue). Without disruptive selection, the initial population is homogeneously distributed over the whole lattice (A). When the ecological function is turned on, the population is divided into two regions with weak contact (B). Selection prefers different phenotypes with respect to the horizontal location of the individuals and adaptation proceeds (C). Fluctuations decide if the final result is speciation or if it is a single population with phenotypically similar individuals. In case of speciation, two phenotypically different populations can easily be distinguished, each one occupying one side of the lattice (D).

In this case only one of the extreme phenotypes remains.

iii) Low mobilities or very high selection pressures prevent speciation events. In both cases a great part of the population dies out at the time when the ecological function is set. Fluctuations dominate divergent adaptation and the initial Gaussian distribution of phenotypes moves to one of the extremes.

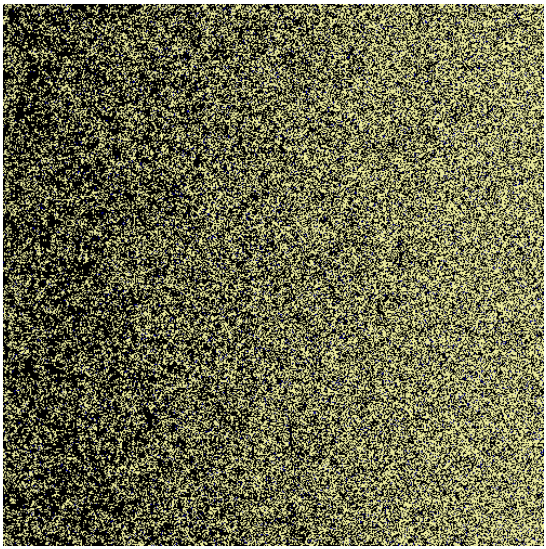


Figure 3.15: Another random seed has driven the system to the case of no speciation. One of the two sub-populations randomly dominates and finally occupies the whole lattice.

It is important to say that for small population sizes fluctuations always seem to prevent speciation, independently of the mobility: no speciation events have been obtained for lattice sizes smaller than $L = 150$.

Concerning the selection pressure, it was also found by Doebeli (Doebeli and Dieckmann, 2003) that very high values of S prevent speciation.

In order to study the effect of sexual selection in our simulations, we introduce the assortative mating strategy used in Gavrilets (1997) to prevent the mating of extreme phenotypes (prezygotic isolation). We measure the absolute difference of the phenotype numbers of both male and female, before mating. If the difference is larger than d, they can not reproduce. If there is no appropriate male among the nearest neighbours, no offspring is produced. Figure 3.16 shows the final phenotype distributions for different strengths S of the ecological function, in cases where speciation occurred. We compare different results using random mating to one where assortative mating is used, with $d = 10$. It can be seen that assortative mating completely

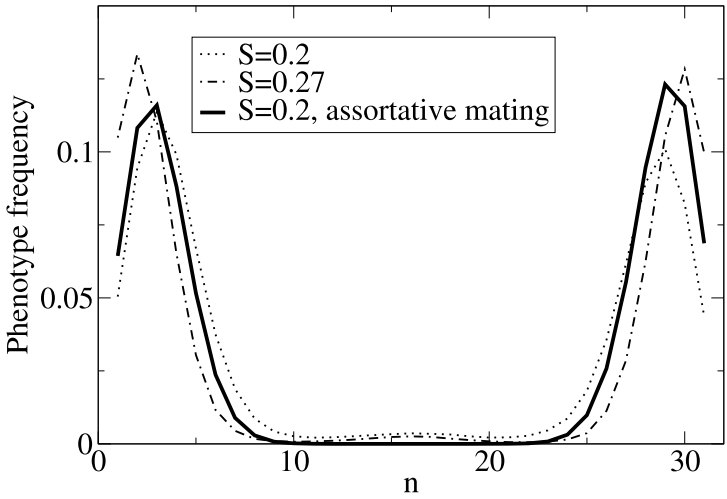


Figure 3.16: Comparison of the final states of the phenotype distributions for different values of the parameter S. The stronger the ecology, the less frequent are the hybrids. Assortative mating leads to the non-existence of hybrids, depicted by the thick curve where $d = 10$. The mobility $m_m = 0.9$ and the mutation rate related to the phenotype is $m_p = 0.15$.

prevents the production of hybrids with phenotype numbers around 16. Additionally, the occurrence of speciation is controlled by the parameter d, as in Gavrilets (1997). Very small values of d ($d < 8$) prevent speciation due to the lack of genetic diversity, which is an important ingredient for the distribution of phenotype numbers to bifurcate.

In Figure 3.17 we show the histogram of the fraction of the population that dies at a given age, for different phenotype numbers. The majority of the hybrids die at low ages and do not generate offspring. These hybrids present low viability and thus characterise a speciation process (Porter and Johnson, 2002).

A cline is defined as a gradient in a measurable character, for instance a phenotypic trait. Relative to the dispersal rate of a species, the slope of a cline between regions is indicative of the extent to which the inhabitants have differentiated. A steep cline means sharp differentiation while a gentle cline means indistinct divergence between

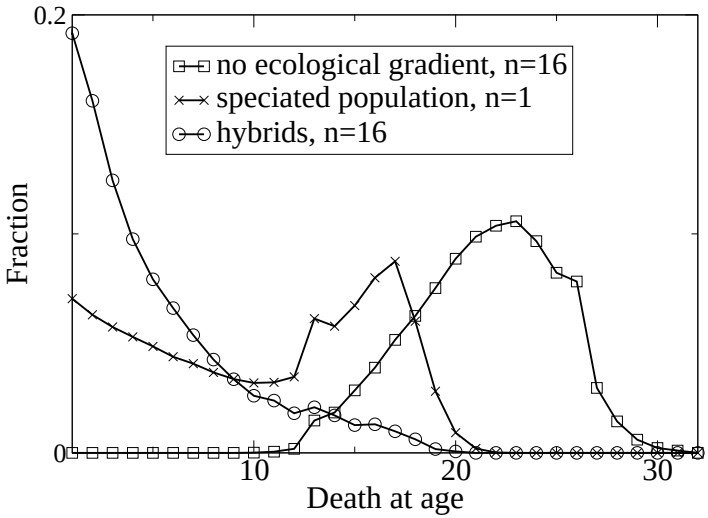


Figure 3.17: Life span for different phenotype numbers. Most of the hybrids die at low ages.

areas (Endler, 1973). In our case we choose the phenotype number, n, as the measurable character. Figure 3.18 shows the fraction of individuals with $n = 0$, $n = 16$ and $n = 32$ at each position x of the lattice. A steep cline can be observed for the $n = 0$ and $n = 32$ populations, as well as the almost disappearance of the hybrids with $n = 16$.

A common outcome in Nature consists of phenotypically distinguishable forms at geographic extreme regions and inter-grading hybrid forms in between. In our case disruptive selection due to the ecological function in eq.(1) prevents such a scenario. However, using the following ecological function:

$$E(x, n) = S \cdot \left| g(x) - \frac{n}{32} \right|, \tag{3.19}$$

hybrids are now favoured and so do not disappear, that is, there is no speciation as shown in figures 3.19 and 3.20. From fig. 3.19 we can observe that the mean value of the phenotypes changes continuously with the geographic position x and there is no sharp separation between the two extreme regions. It is important to say that in using eq.(2) speciation is not obtained even if assortative mating is included (fig. 3.20).

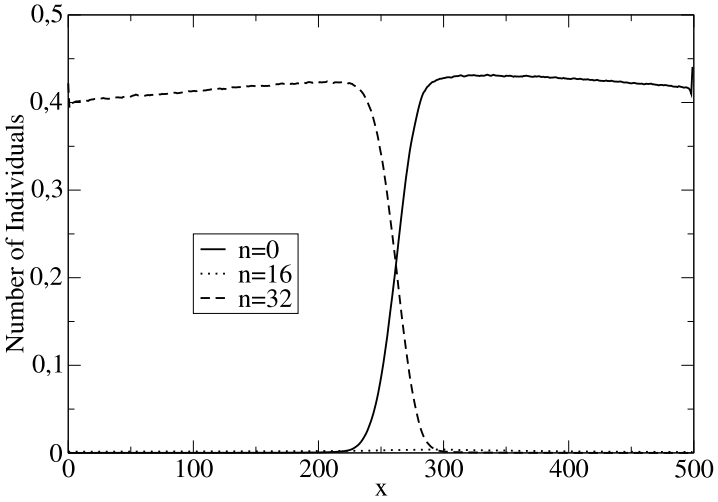


Figure 3.18: Frequency of individuals with a given phenotype number for each position x of the lattice, averaged over the last 10,000 time steps.

3.4.4 Discussion

As reported by Gavrilets (Gavrilets et al., 1998) the dynamics of parapatric speciation is very fast (less than 1,000 generations) and is independent on the mutations rate m_p of the phenotypic trait. We have made some simulations with smaller mutation rates and the time needed to reach a steady state did not increase for $m_p > 0.0001$.

In our model the ecological function must be disruptive, i.e. individuals with intermediate phenotypes have to be discriminated. In a non–disruptive ecology individuals of all phenotypes can adapt to their local environments and hybrids evolve easily at intermediate x-positions. At the end of the simulations individuals of all phenotypes populate the lattice. If the selection against individuals with intermediate phenotype numbers is not strong enough, only an unstable polymorphism appears: The two subpopulations coexist with large gene flux between them until one of them completely dominates and uniformly occupies the whole lattice.

Different from other speciation models, ours allows fluctuations of all quantities, which hinders adaptation and the division of the system into two different pheno-

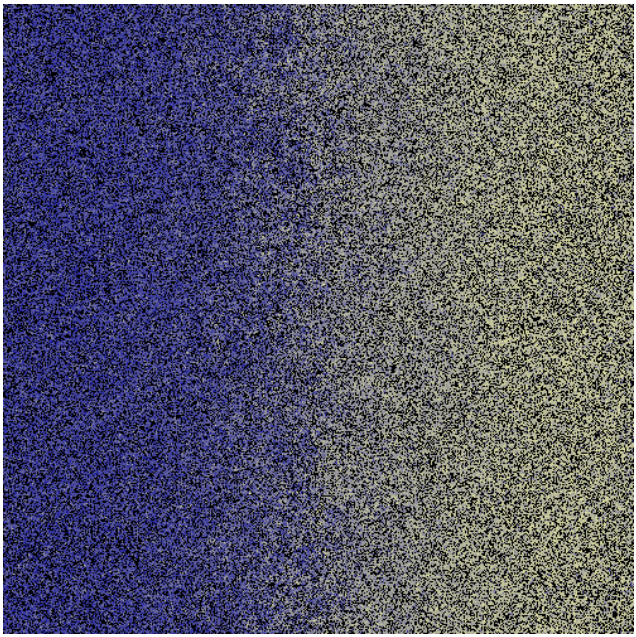


Figure 3.19: Final state of the simulation without disruptive selection. No speciation occurs.

typic populations, even for intermediate values of the selection pressure. This could explain the not so frequent occurrence of speciation in Nature, where many environmental factors act on the different population quantities, like the phenotypic distribution, and where fluctuations of these quantities are ubiquitous. Even if the conditions are optimal, speciation remains a statistical event (that is, for ten different initial random seeds, about five result in speciation and the other five result in an unimodal phenotypic distribution). Speciation is observed frequently for large lattices, where the phenotype distributions fluctuate less. Our results suggest that parapatric speciation occurs preferably in cases where a large population undergoes a sudden disruptive selection over large geographical distances compared to the range of individuals movements.

We have studied the effect of assortative mating in our model, but the final results obtained were nearly the same as those using random mating, although the rule of Gavrilets (1997) increases the probability of speciation occurring. Even without assortative mating, only a very small number of hybrids is born (less than 1% of the total population) due to the small range of the mating region (only between nearest neighbours individuals). Moreover, Figure 3.17 shows that these hybrids die

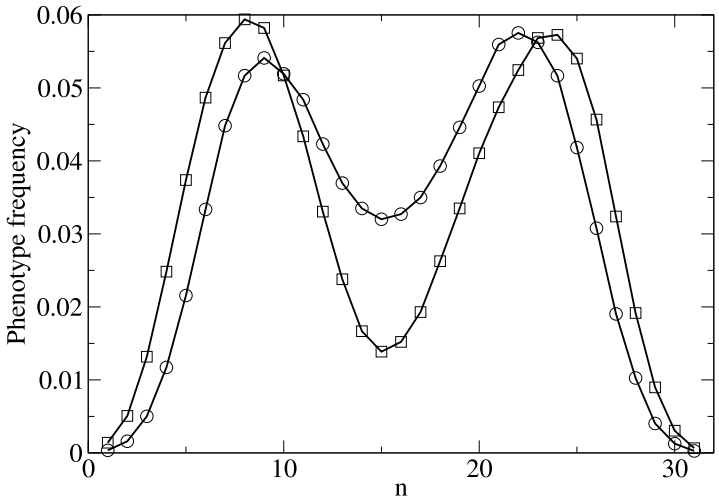


Figure 3.20: Final phenotype distribution for simulations with the ecology function eq.(2). Even for a simulation with assortative mating gene flux prevents speciation.

mainly at low ages and do not produce offspring, which can be interpreted as a form of postzygotic reproductive isolation. In this way a small gene flow does not prevent speciation in this parapatric scenery, even without assortative mating. Models with small population sizes or mating over large geographical distances need assortative mating in order to obtain speciation (Gavrilets, 1997; Doebeli and Dieckmann, 2003).

3.4.5 Conclusions

We present an individual-based model for parapatric speciation, where individuals with different phenotypes are distributed on a spatial lattice. Individuals may die due to genetic diseases or due to a competition for resources that depends on their phenotypes and on their geographical positions. Mating occurs only between next nearest neighbours. Surprisingly, even when considering random mating, fluctuations due to a disruptive selection may drive the system to speciation. On the other hand, under very strong disruptive selection, fluctuations prevent speciation to occur.

In fact, the importance of our approach is that it allows fluctuations in nearly all quan-

tities. Physicists are very conscious about the importance of fluctuations in physical systems, mainly when they present a phase transition, which can be regarded as a process of bifurcation from a single phase (for instance, gas) into a state where two different phases coexist (liquid and vapour). The simplest, naive strategy to deal with such a phenomenon is the mean-field approach, in which the influence of the many units of the system over a particular one is replaced by an average influence or an "average unity", disregarding completely all possible fluctuations. However, this kind of treatment sometimes signals the existence of a phase transition when it does not exist, since the fluctuations that would prevent the transition to occur are ignored (Baxter, 1982).

The speciation process is also a problem of bifurcation and mean-field approaches are widespread (see for instance Kondrashov and Kondrashov (1999)). Again, they certainly give the wrong speciation velocity and may predict a speciation event when it does not exist. An example of a mean-field approach as applied to genetic evolution follows. Instead of considering the genetic features of each individual separately, in a given generation, one considers the genetic frequency distribution of the whole population and imposes some rule for its time evolution from one generation to the other. Indeed, in this case, the genetic information of the whole population is collapsed into a single "average individual", characterising the above mentioned mean-field approach. On the other hand, our model considers each individual separately, and so does not ignore fluctuations. That is why speciation may or not appear depending on the initial random seed, for the same set of parameters.

3.5 Speciational view of macroevolution

We introduce a simple computational model that, with a microscopic dynamics driven by natural selection and mutation alone, allows the description of true speciation events (Schwämmle and Brigatti, 2005). A statistical analysis of the so generated evolutionary tree captures realistic features showing power laws for frequency distributions in time and size. Albeit these successful predictions, the difficulty in obtaining punctuated dynamics with mass extinctions suggests the necessity of decoupling micro and macro-evolutionary mechanisms in agreement with some ideas of Gould's and Eldredge's theory of punctuated equilibrium.

3.5.1 Introduction

The novel interpretation for old paleontological observation that Gould and Eldredge (Gould and Eldredge, 1977) presented in the 70's had a deep impact on evolutionary theory. The theory of punctuated equilibrium leads not only to a change in the paradigm with which some data were analysed, but also caused a definitive shift in the general way of thinking in theoretical biology. Actually, it has been used as a fundamental concept to develop the idea that it is necessary to decouple micro and macro-evolutionary mechanisms. Punctuated equilibrium shows how some evolutionary change may be produced by the success of certain species rather than by a directional and progressive transformation of a lineage.

Darwin's evolutionary theory understands the living world as the outcome of microscopic dynamics alone, driven by selection and mutation. That is, we suppose that there exists a causality in evolutionary changes driven by natural selection operating at an individual level. It is a natural tendency to extrapolate causality, such as defined at this individual level, at all magnitude and time, hoping that Darwinian natural selection alone could fully explain large-scale change in history of life (Gould and Eldredge, 1993).

In contrast, punctuated equilibrium suggests how the interposition of levels breaks this causal reduction and decouples micro- from macroevolution. Causality is suppressed by a hierarchical model with simultaneous action at genetic, organismal and species levels. From this perspective, the central problem of macroevolution is to understand, through a direct study of species, which ones prevail and do better than the others. On the other hand, the traditional gradualistic approach focuses on how natural selection is capable of causing adaptation during evolution, in a process occurring at the population level.

A famous model of long term analysis of evolutionary processes based on the idea of a decoupled macroevolution is the Bak-Sneppen model (Bak and Sneppen, 1993).

Here, interspecific interactions are taken into account as the predominant force capable of generating evolution, under the simplifying assumption that the number of species is fixed and origination is prohibited.

3.5.2 The model

In our approach, we will try to describe the whole mechanism of evolution by natural selection at a population level. Models with dynamics structured at the population level (de Oliveira et al., 2004b; Rikvold and Zia, 2003) or focusing on the micro-macro evolution relations (Chowdhury et al., 2003), also with a complex structure representing the hierarchical organisation at different trophic levels (Chowdhury and Stauffer, 2003), are present in the literature. However, they are all based on a predator-prey interaction type that can account only for the dynamics of extinctions and, for this reason, do not explore the problem outlined by the punctuated equilibrium theory. We will analyse instead a model where the interaction represents a natural selection responsible for speciation. Our purpose is to test if this driving force alone can account for all the phenomenology of macroevolution. For this reason, origination of new species is the crucial new phenomenon that our model must be able to account for. The aim is to define a mechanism that naturally generates disruptive selection and autonomously causes differentiation within a single species. Since we want a mechanism based on selection alone, we are not interested in simple stochastic models. We implement instead a self-modifying selective force based on frequency dependent selection (Dieckmann and Doebeli, 1999; Brigatti et al., 2005) that allows coexistence and branching of species. To sum up, we do not consider species-level fitness, but a mechanism that generates species autonomously. For this reason, we do not simply perform a refilling of extinct species (as for example in Ref.(Bak and Sneppen, 1993; de Oliveira et al., 2004b; Solé and Manrubia, 1996; Chowdhury et al., 2003)) and, as a consequence, their number is not fixed. With this strategy, we can test if it is possible to build up a simple model that unifies adaptation and differentiation into a single framework. This approach also unifies the three time scales (Stenseth and Maynard Smith, 1984) that characterise evolution: the fast population dynamical scale (controlled by natural selection), the slow evolutionary scale (controlled by the mutation process) and the ultra slow macroevolution (the timescale of the speciation/extinction dialectic). Although there is no doubt that single speciation and extinction events occur by the interaction between natural selection and mutation (slow and fast scales), as stated above, there is no general agreement whether macroevolution can be seen as a simple sum of discrete speciation events generated by population dynamics.

We will face this question by comparing the results of our model, born from this unifying view of evolution, with all the quantitative statistical properties observed in the fossil record: scale free behaviour for at least some range of the distribution in

time and size (Newman and Palmer, 1999), a time series of extinction events showing punctuated equilibrium (Gould and Eldredge, 1993), mass extinctions (Raup, 1986) and long term correlations (Solé et al., 1997). We will see that, although the scale free nature is reproduced, not all characteristics linked with punctuated equilibrium show up in the results.

The model worked out is simple and operates on the individual's level. For reasons of simplicity, the model is not developed in genotype space, as for instance in Ref.(de Oliveira et al., 2004b; Rikvold and Zia, 2003), but in the more easy-to-handle strategy space where an individual is represented by an integer number, the strategy parameter x ($0 \leq x \leq P$), that takes into account all the phenotypic characteristics that determine its biological success. Each particular value of the strategy parameter characterises the adaptation to a specific ecological niche. At each time step, an individual generates one offspring with the same strategy as its parent, eventually mutated by a random ± 1 factor with some probability μ, kept constant from the start of the simulation. We allow each agent to live until the occurrence of death caused by a selective pressure, which acts on the fast population dynamics scale, or when it reaches 32 Monte Carlo steps.

This selection is characterised by two different components. The first is density-dependent, responsible for limiting the size of the total population and controlled by the carrying capacity. The other, a frequency-dependent factor, takes into account how, in realistic situations, the tendency to occupy a more favoured region in strategy space balances with an increasing competition among individuals. The latter is the dynamic component of selection, which represents the feedback between individuals and ecosystem and takes into account the instantaneous distribution of the population. Thus natural selection is implemented through a death probability that, in the Monte Carlo simulation, takes the form:

$$S = \frac{1}{K} \cdot \sum_{y=1}^{P} N_y \cdot \exp(-\frac{(x-y)^2}{2b^2})$$

At each time step, a random number is tossed; the individual survives if this number is smaller than S. The strength of competition declines with distance in strategy space according to a Gaussian function with deviation b, and parameter K depicts the carrying capacity. x is the value of the strategy parameter of the individual that is feeling the selection pressure, and the sum runs over the y index that spans all of strategy space. We use periodic boundary conditions in order to avoid edge effects. By N_y we indicate the number of individuals with strategy y. This selective function, inspired by the ones in Ref.(Dieckmann and Doebeli, 1999; Brigatti et al., 2005), is directly modified by the evolutionary process and can lead to the self-organisation of a varying number of different strategy clusters, while allowing their branching and extinction. For this reason, it does not drive the system to an optimal ending point, but leaves it in a permanently changing dynamic state.

Since we deal with an asexual population, the biological characterisation of species, defined for sexual individuals as a reproductively isolated population, must be substituted by a more operational definition, based on a functional differentiation among phenotypical distinct groups. For this reason, we refer to species as a group of individuals that share most of their phenotypic features but which differ for a few traits. According to this definition, the algorithm used associates different species to different clusters of individuals that have a small strategy distance - one being already enough - between them. That is, the space between two clusters can not be occupied by individuals. Although spatial heterogeneity or predation may have a relevant influence on the dynamics of the population and, in particular, on the frequency of branching events, they are not taken into account in our model. Additionally, a static selection component that defines the general ecological condition and can be a cause of a directional selection, driving the population towards some fitness maximum in strategy space, does not change crucially the dynamics of the model.

3.5.3 Results

The dependence of the model's behaviour on the value of the parameters can be summarised by some simple rules. In general, the parameters of interest are only two, and are the ones that effectively control the branching probability. The carrying capacity (K) and the number of possible strategies (P), on the other hand, are not so crucial. In fact, the only role of the first is to regulate the population size, while P is correlated with the mean number of living species, which grows with P following a simple linear relation. In contrast, the mutation rate (μ) directly influences the branching probability. In the following we fix the value of μ so as to make possible a realistic evolutionary simulation, where mutations have to occur infrequently, which is in accordance with the fact that this parameter controls the dynamics on the slow evolutionary scale. For this reason, we set its value to 0.005. One parameter remains, b, whose value is responsible for controlling competition and, as a consequence, the force that splits up a cluster into two different ones. This drive increases as b is decreased, causing a larger number of occurrences of branching events. There is a simple relation between the mean number of species and b, taking the form: $N(b) \propto b^{-1}$. We adjust this last parameter by searching for an equilibrium between really slow branching dynamics, which happens for large b and is a case not suitable for a statistical analysis, and small b values, for which the population feels such a strong drive that it is impossible to define a stable evolutionary tree. In this last situation, where the branching events are so numerous that the distribution can not be well defined, with a large number of peaks connected by intermediate strategies, it is impossible to perform a cluster analysis. An example of a stable and living evolutionary tree generated for standard parameters values can be seen in fig. 3.21.

We start our analysis investigating the probability distribution of lifetimes $E(t)$ of the

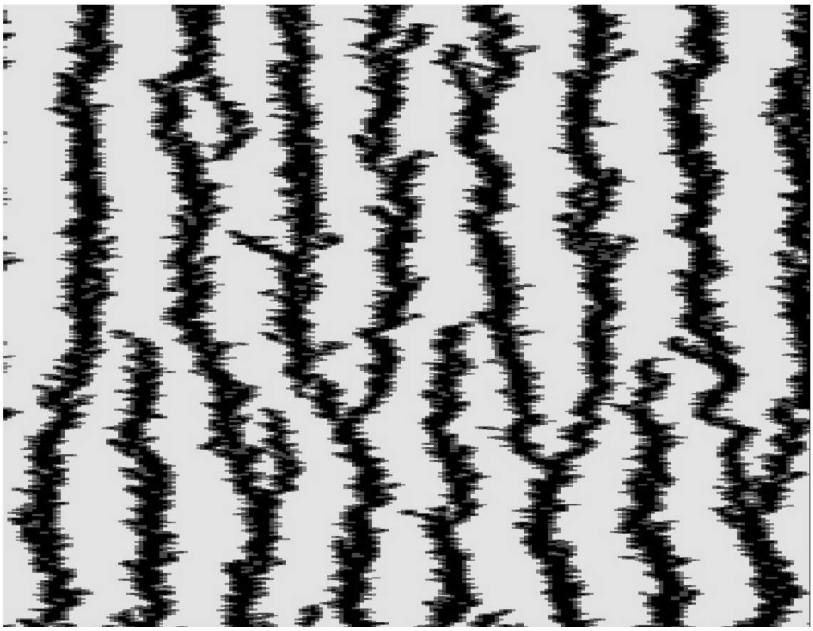


Figure 3.21: Time evolution of the population. The parameters used are: the Verhulst parameter (10000), the mutation rate (0.005), the number of strategies (200), the parameter b (15).

species, a central measure due to its comparability with observational results. From the data shown in fig. 3.22, we observe that a power law can be recovered

$$E(t) \propto t^{-\gamma} \;\; ; \;\; \gamma = 1.9 \pm 0.1$$

over about two decades, with an exponential tail for large times.

These values are comparable to data from extinction records. Even if their interpretation is still under debate, it seems that a power law fitting with an exponent close to -2 is more convincing than an exponential, for at least the shorter lifetimes (Newman and Palmer, 1999; Drossel, 2001). A similar behaviour was confirmed by other models as well: Ref.(de Oliveira et al., 2004b) agrees with our value for the exponent and Ref. (Chowdhury et al., 2003; Chowdhury and Stauffer, 2004) with the deviation from the power law for very long lifetimes.

The same figure shows also data related to the distribution of lifetimes for the origination processes. The life time of originations represents the time interval between one speciation event and the following one in the same lineage. These new data are

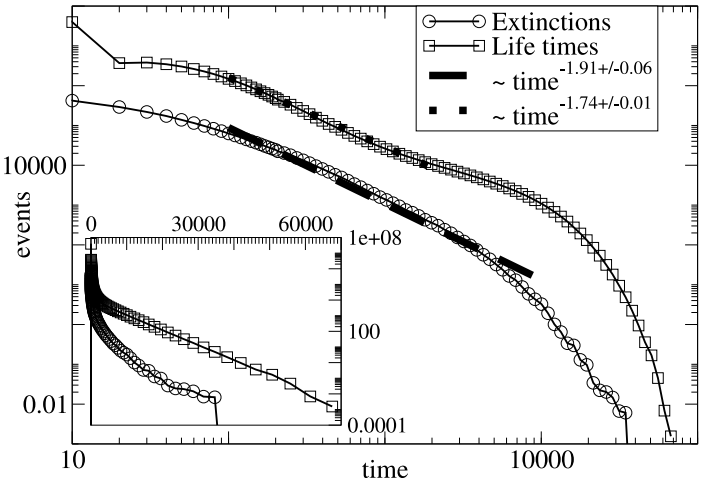


Figure 3.22: Frequency distribution for the lifetime of the species. The circles represent the lifetime of extinction events (from the branching of the new species until its extinction), the squares the lifetime between speciation events. The inset shows the exponential tails for large values of lifetimes. The parameters of this simulation are the same as in fig. 3.21, except for the number of strategies (500), and are used in all the following simulations. The simulation run has the duration of 10^7 time steps.

of more difficult interpretation because there are no observations in the fossil records. Moreover, only models where speciation events do not coincide with extinction events (such as they do in refilling models) but are defined by an internal dynamics can produce such results. The distribution of lifetimes of originations shows also a power law behaviour with $\gamma = 1.74 \pm 0.01$ with a rather extended exponential tail.

From our simulations we also obtain the distribution of extinction events as a function of their size s. By the term size we denote the number of individuals that make part of an extinct species from its origination until its disappearance (see fig. 3.23). It is possible to fit the data with a power-law: $E(s) \propto s^{-1.42 \pm 0.02}$. It is difficult to compare these results with the other ones present in the literature (de Oliveira et al., 2004b; Solé and Manrubia, 1996) because usually the size of the events is obtained by counting the number of species or families.

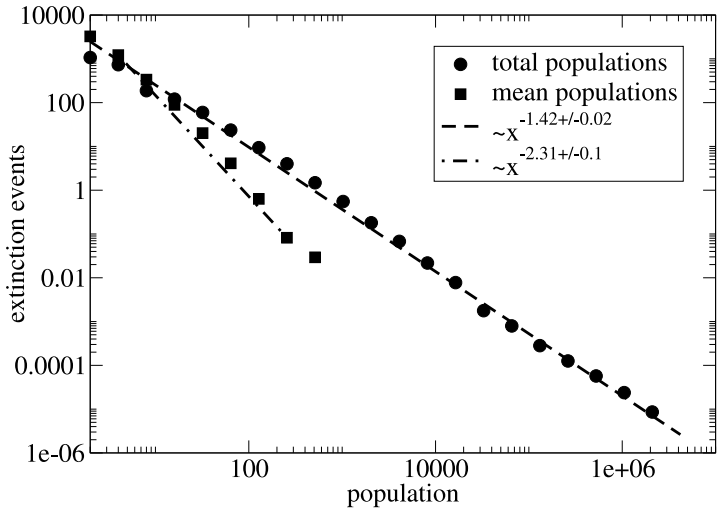


Figure 3.23: Frequency distribution for extinction size. The circles represent the number of all the individuals from the branching of the new species until its extinction. The squares make a temporal average in this interval.

From the results stated above it seems that our model is characterised by power law distributions However, by analysing the distribution of lengths of intervals without activity (period of stasis), we find a clear exponential behaviour (see fig. 3.24). If a critical process was involved, we should expect another power law. Moreover, the existence of a scale is easily perceived by analysing the time evolution of the number of extinctions (fig.3.24). No mass extinctions are present and in the time series we can not recover an intermittent behaviour characteristic of a punctuated equilibrium phenomenon.

Finally, we tried to detect long-range correlations in the time series of extinction events (inset of fig. 3.24). For a process with no characteristic timescale, a fluctuation $F \propto t^{\alpha}$ with $\alpha \neq 1/2$ is expected. More, the exponent α is related to the one (β) describing the power spectrum of the series through: $2\alpha = \beta + 1$. Self-similar fluctuations, described by an $1/f$ spectrum ($\beta = 1$), emerged from the analysis of some paleontological data (Solé et al., 1997). In contrast, from a preliminary analysis we get $\alpha \leq 1/2$.

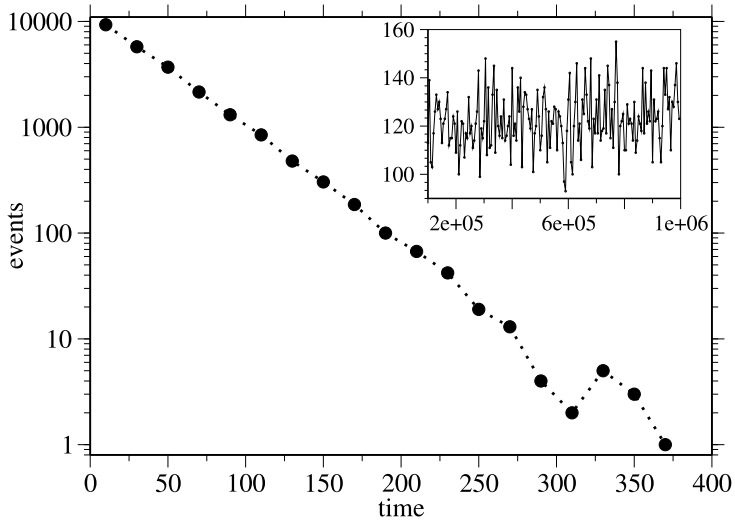


Figure 3.24: Exponential distribution of lengths of intervals without activity (periods of stasis). In the inset the temporal evolution of the number of extinction events as obtained collecting each value from a time interval of 5000 steps.

3.5.4 Conclusions

Our model, through a realistic microscopic dynamics driven by natural selection and mutation alone, presents promising results showing, in accordance with observations obtained from fossil records, power law behaviour for statistical distributions in time and size. Albeit these successful predictions, the last results show a difficulty in obtaining punctuated dynamics with mass extinctions, where long range correlations allow the clustering of the extinction events. These facts are somewhat intriguing and deserve further investigation. At the time, we can work out the following possible interpretations. Perhaps the recorded behaviour in our data is linked to the structure of our model, which is based on an autoregulating selection that does not allow big fluctuations in the population size.

Alternatively, we can argue to have found a hint against the possibility of a unified model that, from a microdynamics of speciation based only on natural selection force, can represent long-term statistics of evolution. It is necessary to decouple the dynamics, taking into account higher order interactions such as trophic relation at

several levels or an intrinsic dynamics added to the natural selection one(Gould and Eldredge, 1977). A possible shortcut could be the inclusion of qualitatively different processes, related just with mass extinction events, that can be taken into account by the introduction of some biotic or abiotic external stress (Newman, 1996). Anyway, would this last scenario be the right one, we could claim to have been able to describe, through the implementation of true speciation events, the statistical distributions related to the spontaneous rate of replacement of one species by another.

Chapter 4

Language competition

4.1 Introduction

The emergence of language capability in the human species allowed us to communicate easily without restricting ourselves to simple messages. The possibility to hand over an abundant amount of information to the following generations, or even to increase general knowledge by conserving new information in its written form, is one of the main ingredients of technological and cultural progress (although only a minority of the languages on Earth has a written counterpart). The language trait had a profound impact on the human phenotype, evolving from the capability of simple expressions up to its current complex structure.

The field of language evolution, or in other terms, evolutionary linguistics, bears a rather strong analogy to biological evolution (Darwin, 1859; Mesoudi et al., 2004). A short time after Darwin's publication of *The Origin of Species*, August Schleicher constructed the family tree of Indo-European languages by comparing their grammar, phonology, lexicon and history (Schleicher (1861) and Figure 4.1). He concluded that they descend all from a common ancestor language, similar to the existence of a common ancestor species for all biological species. Nevertheless, research on language evolution did not develop as fast as the one on biological evolution, and initiated again almost 100 years after Schleicher's publication with the work of Hockett (1960), who distinguished 13 design feature of languages not found in the communication systems of animals.

Computational approaches on language evolution began with the work of Hurford (1989). These approaches facilitate the description of the evolutionary behaviour of

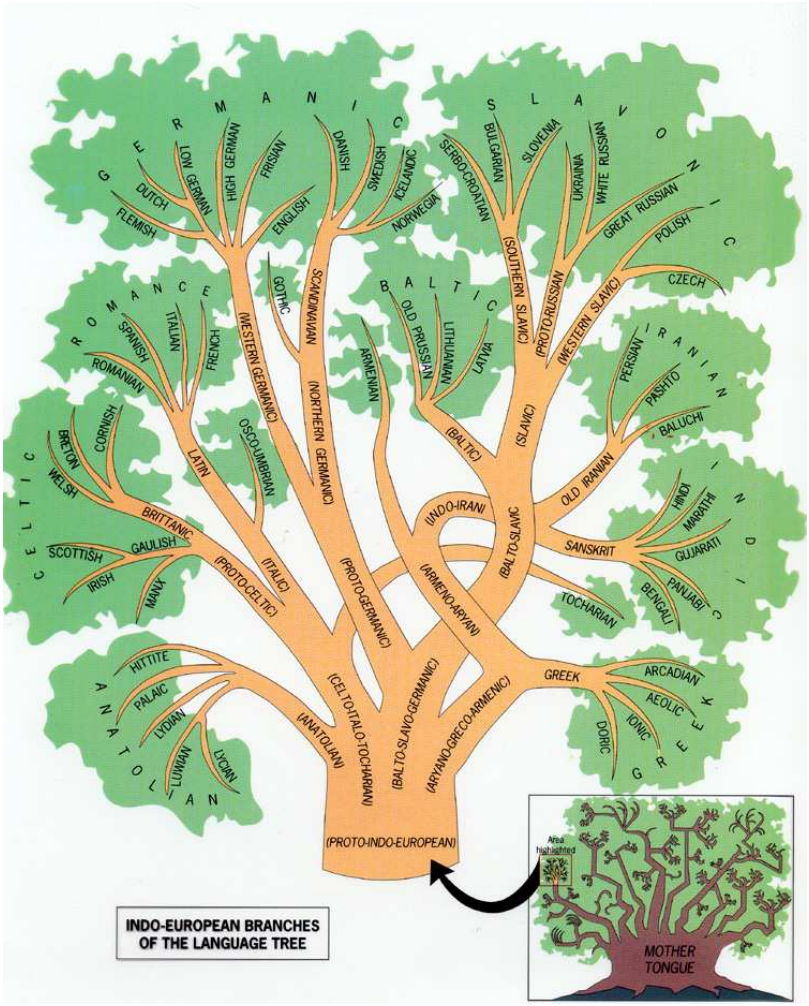


Figure 4.1: Language tree of the Indo-European family.

such systems. Computational as well as analytical research on evolutionary linguistics can be grouped into three main topics:

Invasion of language capability It is believed that modern language already existed about 50,000 years ago, and probably initiated the *cultural explosion* which happened at that time (Calvin, 2004). An indicator of this theory is the gene

FOXP2 in chromosome 7 of the human genome. This gene, mapped in the year 1998 (Fisher et al., 1998), is available only for the human species. It is known to have major impact on the formation of linguistic morpho-syntax (Hurst et al., 1990) and on comprehension (Vargha-Khadem et al., 1995). This so-called *grammar-gene* was estimated to have been fixed in the human species less than 120,000 years ago (Enard et al., 2002).

The emergence of different aspects of language has been focused in several computational models. Some of them are reviewed in Wang and Minett (2005) and Section 1.1.

Still there is no empirical evidence if language emerged simultaneously and independently at various locations, or if there exists an ancestral proto-language. In the scientific community prevails the theory of one proto-language (Bengtson and Ruhlen, 1994), and was found to be more likely as indicated in the computational model of Freedman and Wang (1996).

Invasion of linguistic innovations Languages change continuously in sound (phonetics) and appearance (lexicon). Studies on sound change and lexical diffusion have been carried out as well as simulations on the interaction between words. Most of these works are summarised in the nice review of Wang and Minett (2005).

Invasion, competition and extinction of languages The number of persons, who speak one particular language, changes with time and languages compete against each other if their speakers are in contact. A language becomes extinct as soon as no speaker of that language remains, and so most of its information gets lost. On the other hand a language can be the founder of new languages (as for instance Latin of Spanish, French, Rumanian and Italian). These processes of extinction and origination can be thought to be the analogies to extinction and origination processes of species in biological evolution. Similar to the biological case, the area of language competition is suitable for computational approaches in order to investigate in which way competition controls their extinction and origination. This chapter focuses on language competition and presents the scientific approaches for its understanding in the following.

About 6000 languages are now spoken in the world (Krauss, 1992) and their size distribution D is log-normal (Sutherland (2003), Figure 4.2):

$$D = e^{-const(logS)^2}, \tag{4.1}$$

where S is the number of persons which speak a language as mother tongue.

An estimate of over 50% languages will become extinct in the next century (Krauss, 1992), especially the ones which do not have many speakers (Sutherland, 2003).

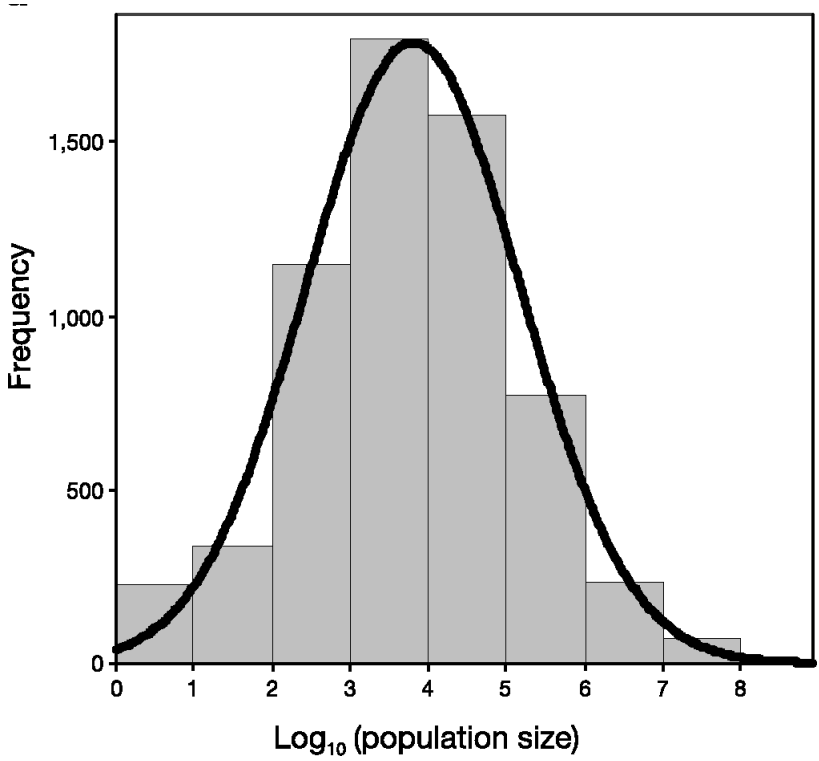


Figure 4.2: Size distribution (Frequency) of languages. The population size, S, is the number of people which speak that language as mother tongue (Figure from Sutherland (2003)). The rightest language is Chinese with more than a billion speakers. The distribution is very similar to a log-normal distribution with a maximum for languages spoken by about $10,000$ speakers.

Nowadays, socio-economic factors lead to the situation that an increasing number of people prefer to share a common first language in order to facilitate contact with a maximum number of persons. The future extinction of languages, and with it the loss of cultural diversity, has been announced several times in the present literature (see for instance Crystal (2000)).

Some research on grammar competition bases on the hypothesis of "Universal Grammar" (Chomsky, 1980), and can also be applied to account for language competition. There, language capability is connected directly to some innatist cognitive capabilities. The framework of Universal Grammar allows for a restricted set of grammars to be learnt by the human brain. Nowak and colleagues describe the competition of

these grammars by a system of coupled differential equations (Nowak et al., 2001; Mitchener and Nowak, 2004). Their results propose that the child's imperfect learning of its parents' grammar leads to a stable coexistence of many grammars, whereas for perfect learning one grammar dominates.

Theoretical research on current language competition began only recently with the model of Abrams and Strogatz (2003). In their simple analytical model, consisting of a non-linear differential equation, they find that one of two languages dominates the entire system after finite time. Their result of which of the languages wins, depends on their initial frequency and status. This model has been extended by the use of a one-dimensional reaction diffusion equation to account for local population densities (Patriarca and Leppänen, 2004). Within this model, languages can coexist by having speakers being located at different geographic locations. Another extension of the model considers bilinguals (Mira and Paredes, 2005).

Obviously, such theoretical approaches are rather restricted, because they neglect the effects of critical behaviour as well as the great complexity of language (as for example social structure, subsets of a language used in different social contexts, or learning capability with respect to the age of the individuals).

Computational agent-based models provide more possibilities to understand the complex aspects of language evolution. Simulations on language competition have been carried out first in 2005 by Schulze and Stauffer (2005c), Kosmidis et al. (2005) and Schwämmle (2005) (see also the following section of this chapter).

The field of language competition is surprisingly young compared to the well investigated corresponding fields in biological evolution, making this field suitable to strong progress in the next years. First approaches have been made in order to understand more of multilingual systems, i.e. of their emergence and their stability. The search for the fundamental mechanisms responsible for the macroscopic behaviour of such a systems is still in the beginning. The possibility to understand the dynamics of multilingual systems would enable us to predict, or even avoid, language extinctions.

The following section describes a work of the present author with respect to the stability of a system of two spoken languages on a square lattice (Schwämmle, 2005). The third Section analyses a system of many languages where age-restricted learning capability and error learning are taken into account (Schwämmle, 2006).

4.2 Simulation for competition of languages with an ageing sexual population

Recently, individual-based models originally used for biological purposes revealed interesting insights into processes of the competition of languages. Within this new field of population dynamics a model considering sexual populations with ageing is presented (Schwämmle, 2005). The agents are situated on a lattice and each one speaks one of two languages or both. The stability and quantitative structure of an interface between two regions, initially speaking different languages, is studied. We find that individuals speaking both languages do not prefer any of these regions and have a different age structure than individuals speaking only one language.

4.2.1 Introduction

Our model is based on the well understood Penna ageing model (Chapter 2, Penna (1995) and Moss de Oliveira et al. (1999b)) on a lattice (Makowiec, 2001; Sousa and Moss de Oliveira, 1999; Schwämmle et al., 2005b) which provides us a possibility to model a sexually reproducing stable population. We simplify the model by defining languages as an integer number, that is they are not composed of different words. Thus we are able to avoid changes in a same language. The parents pass their language entirely to their offspring. In order to stabilise their distribution, languages can be forgotten during lifetime.

The study of an interface between originally different regions under different parameter sets reveals certain characteristics of the geographical distribution of the languages on the lattice as well as of the age structure of the agents for different languages.

This section 4.2 is organised as follows: the next subsection explains the main features of the model and tries to justify the parameters we use. We present the results and the conclusions in the two following subsections.

4.2.2 The model

This subsection is separated into two subsections in order to provide the reader a small review of the Penna model on a lattice as well as to present our modifications adapting the model to a system where languages compete with each other, separately.

The sexual Penna model on a square lattice

The sexual version of the Penna model is explained in Section 2.3. The maximum number of deleterious mutations is here $T = 3$, the minimum reproduction age $R = 10$, the mutation rate $m = 1$ and the number of predefined dominant positions is 5. Unlimited population growth is avoided by the use of the additional death probability: $V = N(t)/N_{max}$. A female agent selects a male on the central site with a probability of 25%, and if it fails, it searches among its nearest neighbour sites, at each one with a probability of 25%. The offspring is placed on a nearest neighbour site of the mother even if the site is already occupied (which is different from the usual versions of the Penna model on a lattice). Every time step an agent moves to a randomly selected nearest neighbour site with probability p_m, if this site is less or equally populated. The bit-strings are initialised randomly with zeros and ones at the first time step.

Competition of language

For simplicity we define a language by an integer number and not by a bit-string which would describe, for instance, different words or an alphabet as in ref. (Schulze and Stauffer, 2005c; Kosmidis et al., 2005).

Every agent speaks a language l, an individual variable which can have three values: $l = 1$ and $l = 2$ mean that it speaks language 1 or 2, respectively. The third possibility, $l = 3$, describes the case where an agent speaks both languages. Our model contains two parameters dealing directly with these language values. At birth an offspring learns the language l of its parents if they speak the same one(s). In the case of parents with different values of l the offspring speaks both languages ($l = 3$) with probability p_b, otherwise it speaks only one language, each with the same probability. The other parameter is the probability p_f, with which an agent may forget an already learned language. Every time step an agent, which speaks *both* languages, counts the number of surrounding agents speaking language l in its neighbourhood. This neighbourhood is defined by the square of the distance of d sites from the central site, for instance the 8 nearest neighbours with $d = 1$ or 24 with $d = 2$. The central site is not counted. If and only if there is a majority of people in the neighbourhood speaking language 1 or 2 the agent forgets language 2 or 1 with probability p_f, respectively. Thus it speaks only the language which dominates in its surrounding. The lattice has free boundary conditions.

4.2.3 Results

In our simulations we restrict ourselves to the following initial conditions: half of the lattice of size L times L is filled up with agents speaking language $l = 1$ and the

other half with language $l = 2$. We study stability and shape of the interface between these two regions. Simulations with randomly distributed languages do not present a stable interface: The whole population speaks only one and the same language after a few time steps.

The initial population consists of $10,000$ males and $10,000$ females randomly distributed over the lattice with the values of l as described above. The carrying capacity is $N_{max} = 1,000,000$ on a 20 times 20 square lattice.

The simulations show that the interface is neither stable for p_b values smaller than one nor for low occupation (less than 100 agents per site), which is controlled by the carrying capacity P_{max} and the lattice size. After a short time the number of agents speaking l begins to fluctuate strongly and finally converges into the stable state where only one language is spoken. In general the stability of the interface depends crucially on the initial state as well as on the random seed. For instance, Figure 4.3 shows a simulation with $p_f = 0.1$, $p_b = 1$, $p_m = 0$ and $d = 1$ presenting such an instability.

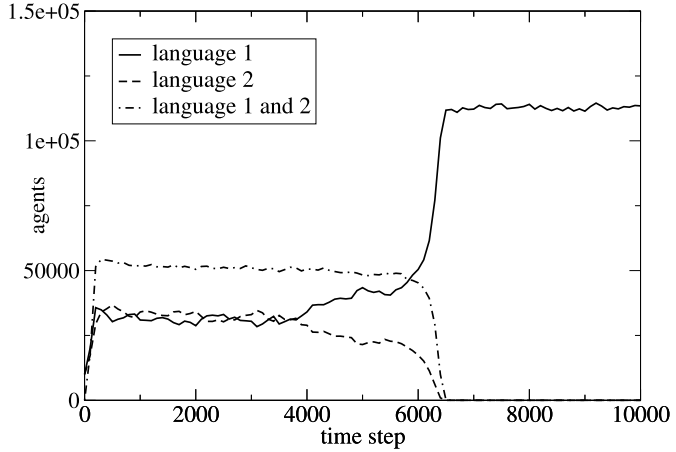


Figure 4.3: Unstable interface: Only agents with $l = 1$ remain.

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We concentrate now on the results of the simulations for different values of the parameter p_f, leading an agent to forget one of its languages. We fix the other parameters to $p_b = 1$, $d = 1$ and $p_m = 0$. As a function of age, Figure 4.4 shows the population of bilinguals (agents speaking both languages) divided by the monolinguals ($l = 1$ or 2)

for different values of p_f. The function decreases exponentially due to the effect that every time step a fraction of the bilinguals becomes monolingual. With increasing p_f a larger fraction of older bilingual agents forgets one of their two languages since in their environment they do not need both. The higher the probability to forget a language, the smaller is the number of older agents speaking both languages and thus less offspring with $l = 3$ are created. Figure 4.5 depicts the mean value of monolinguals and bilinguals during one simulation for different values of p_f. The number of bilinguals decreases with increasing p_f as expected. It seems that the fraction of bilinguals decays roughly as a power law with exponent -1 for higher values of p_f.

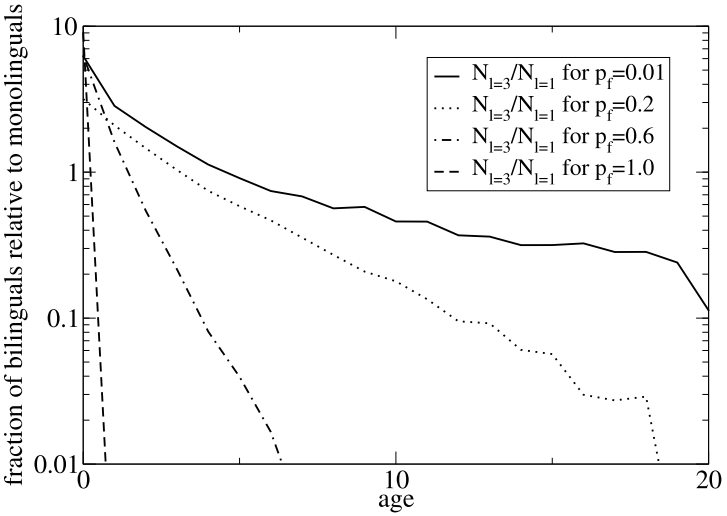

Figure 4.4: The number of agents speaking language $l = 3$ divided by the number of agents with $l = 1$, as a function of ages. The number of agents speaking both languages decreases drastically with age for large values of p_f.

Figure 4.6 shows the number of agents with a certain value of l versus their position in direction x perpendicular to the interface. The number is averaged over the direction parallel to the interface and is measured after $10,000$ time steps. We observe a quite stable interface between the two regions, each one with one of the two languages in majority. The number of agents speaking $l = 1$ and $l = 2$ decays exponentially at the interface as also reported in ref. (Schulze and Stauffer, 2005c). Interestingly, the number of bilinguals is constant over the whole lattice. The shape is not altered by

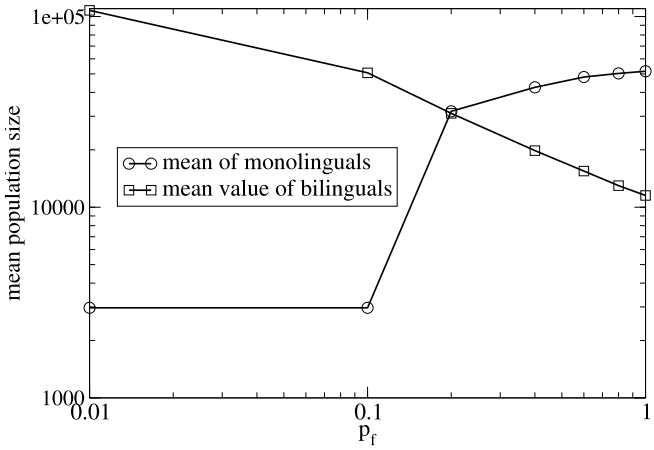

Figure 4.5: The mean value of the number of bilinguals seems to decay as a power law.

changing p_f.

We increased the number of agents by setting $N_{max} = 10,000,000$ and the initial populations to $100,000$ females and $100,000$ males for $p_f = 1$: Now the exponential decay at the interface is observed clearly, as seen in Figure 4.7.

In our simulations we have also changed the parameter d, defining the number of neighbours an agent with $l = 3$ examines, in order to know which language it can forget. The results for large d are the same as for $d = 1$.

The distribution of speakers on the lattice for different mobilities p_m is shown in Figure 4.8. We set $p_f = 0.2$ and $d = 1$. Higher mobilities lead to a smoother interface. Thus the exponential decay is weaker for large p_m. At very high mobilities the interface becomes unstable.

4.2.4 Conclusions

We present simulations where the population of speakers of two different languages are of similar size for at least $10,000$ time steps. This meta-stable state is obtained

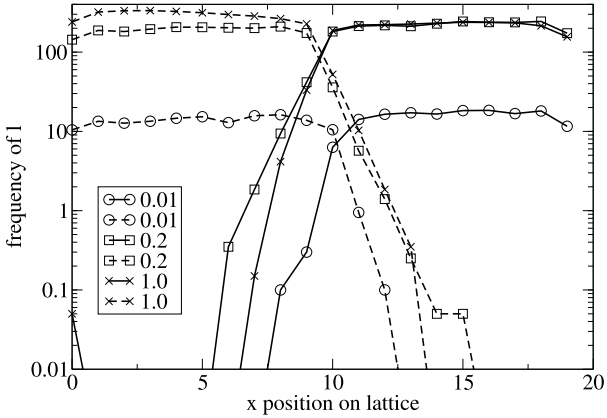

Figure 4.6: The frequency of monolinguals saturates for high p_f values. The dashed line corresponds to $l = 1$ and the solid line to $l = 2$. For all values of p_f we find an exponential behaviour at the interface.

only for a large number of agents per site and initialisation of the lattice by distributing the speakers of different languages on different halfs of the lattice. We can interpret that an interface of speakers in high populated areas, for instance at the Canal Street of New Orleans where on one side French and on the other side English is spoken, is more stable than in low populated areas. Different languages cannot survive for long times if their speakers are not geographically separated. Another result of the model is that the fraction of bilinguals relative to monolinguals decreases exponentially with age. Older people, living for long time at the same place, do not need a second language. The mean value of the number of bilinguals versus the parameter p_f to forget a second language shows a power law with exponent -1.

The results of ref. (Schulze and Stauffer, 2005c,a) are well reproduced in our model: At the interface the number of monolinguals decreases exponentially. Surprisingly, the number of bilinguals distributes rather homogeneously over the whole grid. The exponential decay of the number of monolinguals becomes steeper for smaller mobilities but is left unaltered by the lattice size. Higher mobilities lead to a more homogeneous distribution and can break the meta-stability of the interface. Nowadays, globalisation gives us the possibility to travel frequently over long distances and to stay larger periods at different places on Earth, one of the reasons why languages are becoming extinct.

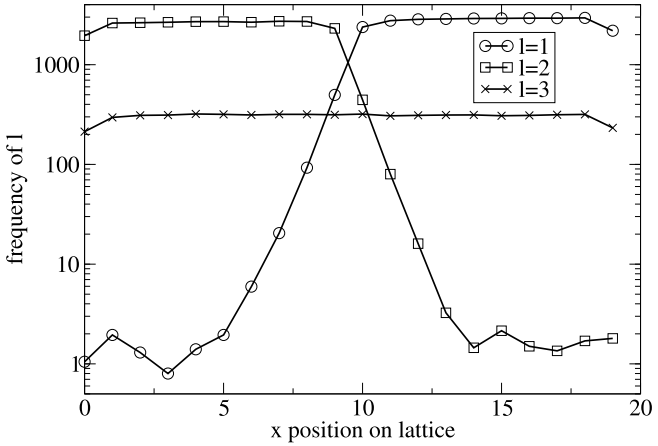

Figure 4.7: Frequency for all l on the interface. Bilinguals are homogeneously distributed. The number of monolinguals decays exponentially at the interface.

We presented here the first model for language competition including ageing and sexual reproduction and reproduced well the results of other models although they are quite different. Numerical agent-based models on the computer yield interesting results despite their simplicity, and we think that there will be much more to be done in future.

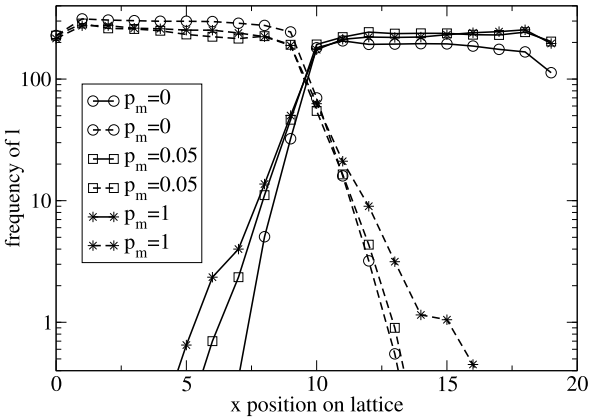

Figure 4.8: Frequency of monolinguals on the lattice for different mobilities p_m with $d = 1$ and $p_f = 0.2$. The larger p_m is, the smoother becomes the steepness of the exponential decay.

4.3 Phase transition in a model of learning foreign languages

The understanding of language competition helps us to predict extinction and survival of languages spoken by minorities. A simple agent–based model of a sexual population, based on the Penna model, is used in order to find out under which circumstances one language dominates other ones (Schwämmle, 2006). This model considers that only young people learn foreign languages. The simulations show a first order phase transition where the ratio between the number of speakers of different languages is the order parameter and the mutation rate is the control one.

4.3.1 Introduction

It is believed that the concept of an "Universal Grammar" is attached in some way to our genetic code, enabling humans to learn languages fast during childhood (Chomsky, 1980). Much attention has been paid on the evolution of this trait and the competition between different grammars (Nowak et al., 2001; Komarova, 2004). In these models the different grammars compete by attributing different fitness to agents carrying different traits. That is why it is possible to apply the models already successfully used in evolutionary biology to this field.

The competition between languages has been investigated in the last years by analysing the stability of systems consisting of two or more languages (see first section of this chapter) In our approach we use a computational model that does not attribute different fitness to agents with different language traits and that does not neglect bilinguals.

The knowledge of a new or different language does not imply a smaller death probability, which makes our approach different from those applied to biological systems. (We are aware that, unfortunately, there are many exceptions to this rule). In order to present an age–structured model we build our one based on the Penna model for biological ageing (Penna, 1995; Stauffer et al., 2006). The usage of an age–structured model enables us to merge an age–dependent learning procedure to language competition in order to get more insight into their correlation. We will focus on the question whether a phase transition exists, as already found in refs. (Stauffer and Schulze, 2005) and Komarova (2004), between a state where a single language dominates and another one where all languages are uniformly distributed (also called fragmentation).

This section 4.3 is organised as follows: the next subsection explains the model, the following one presents our simulations and in the last subsection we discuss the results and propose further extensions of the model.

4.3.2 The model

Our model combines population genetics with age–dependent language learning. For a more detailed description of the sexual Penna model and its applications we refer to Chapter 2, refs. Penna (1995) and Stauffer et al. (2006). Here, $T = 3$, $R = 10$, the chronological genome has five dominant positions and the mutation rate is one. The Verhulst factor $V = N/K$ avoids unlimited population growth.

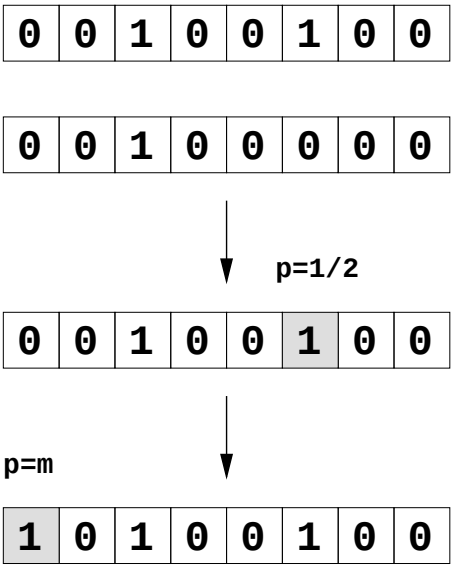

Figure 4.9: The language bit–string of the child is built from the two parent's language bit–strings. If both parents speak a given language, the child learns it as well. If only one of the two parents speaks it, the child learns that language with 50% probability. After constructing the child's bit–string, a mutation with probability m at a random position may generate a new language spoken by the child.

The language trait of an agent, representing its current ability to speak certain languages, is modelled by another single bit–string of L bits, which means that agents can speak at most L languages. In this way the whole structure a language could consist of is neglected, and each language is treated as a unit. So an agent who has, for instance, the third bit set in this non age-structured bit–string, speaks currently language $l = 3$ (eventually among others). An agent can learn or forget a language only in youth, during the first c iterations after birth. We call c the *maximum learning age*. Older agents are not able to change their knowledge on languages. The interac-

tion between a young agent and a randomly chosen teaching agent works as follows: At first, a random position on the language bit–strings is chosen, the same for both the young and the teacher. The young agent learns the language corresponding to that position if it is spoken by the teacher. If the agent already speaks more than one language, it forgets the language corresponding to that chosen position if the teacher does not know it, and forgets all its other languages as well. Let us illustrate the outcome of the interaction agent-teacher in computational terms: the language trait of an agent speaking zero or one language has an OR operation with the teacher's one at the chosen position; otherwise an AND operation is performed. The young agents's language traits are actualised in this way by f different teachers with whom it makes contact, at different positions per iteration. $f = 0.5$ means that the language traits are actualised once per iteration with a probability of 50%. At birth an offspring obtains its language trait as illustrated in Figure 4.9. If both parents speak the same language the offspring will speak it as well. In case only one of the parents speaks a language, the child learns it with a probability of 50%. Additionally, the child can learn a new language at birth with probability m (i.e. a randomly chosen bit is set to one). We call this probability m of erroneous learning the *mutation rate* due to its analogy to biological systems. The simulations start with all non age-structured bit–strings randomly filled with zeroes and ones.

4.3.3 Results

Two languages

This subsection will concentrate at first on the results of simulations considering traits of only two languages ($L = 2$). The simulations are carried out for at least $20,000$ iterations, depending on population sizes. All results correspond to a final stationary state of a simulation.

A phase transition can be observed in the behaviour of the ratio between the number of speakers of different languages (order parameter) as a function of the mutation rate m (control parameter). Each ratio is averaged over the last $1,000$ time steps. Figure 4.10 shows these ratios for different carrying capacities K. The simulations are carried out with a maximum learning age $c = 4$ and for $f = 0.5$. Each point represents the outcome of a separate simulation. The language ratio decreases fast at the critical point which is situated between $m = 0.2$ and $m = 0.3$. The transition separates the phase/state where the two languages have equal sizes, for small values of m, from the phase/state where one language dominates clearly the other one. We can also observe from Figure 4.10 that the population size does not alter the shape of the transition. Thus this transition can be found for arbitrary population sizes and therefore corresponds to a real physical phase transition.

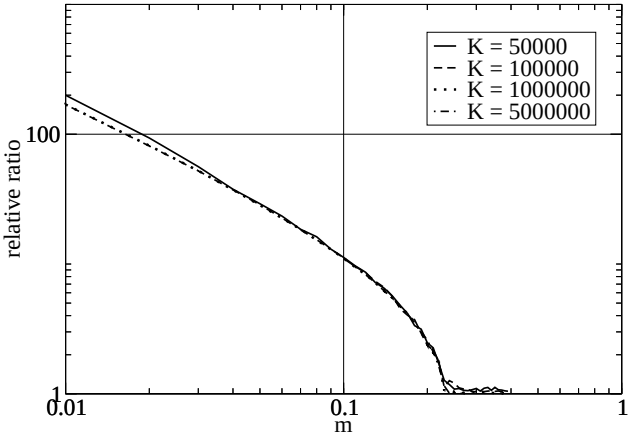

Figure 4.10: Ratio of the two language sizes versus mutation rate. The language size is the number of agents speaking that language. The figure shows a clear phase transition. Different population sizes alter neither the shape nor the position of the critical point.

The language ratio increases strongly for low mutation rates. A power law with an exponent of minus one is shown in Figure 4.11. The parameters in the simulations are $c = 4$, $f = 0.5$ and $K = 1,000,000$, i.e. the population consists of about $80,000$ agents. This same exponent has been obtained for different sets of parameters.

In Figure 4.12 the shape of the phase transition curve is compared for different values of c and f, the two parameters defining the amount of interactions between the agents. The carrying capacity $K = 100,000$ fixes the population size at around $8,000$ agents. The shape of the curves remains the same but the curve itself is shifted to higher values of the mutation rate for increasing c and f. The shift is linear with respect to each of both parameters. The shift decreases for values of $c > 10$, as expected, due to the small number of individuals surviving up to ages much older than the minimum reproduction age $R = 10$ (not shown). For very large values of f, the phase transition disappears and the only possible final state of the system is the one where one of the languages dominates.

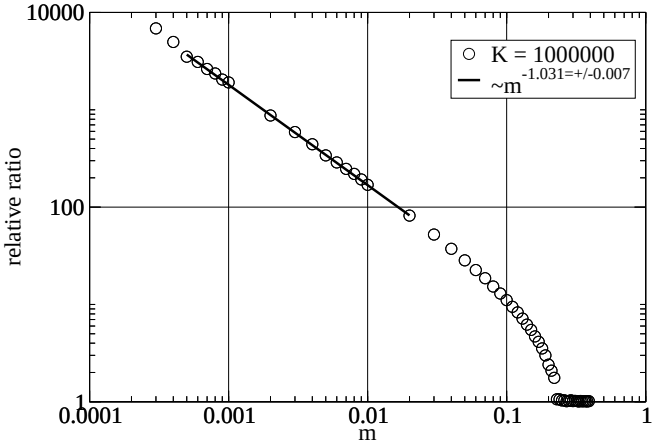

Figure 4.11: The language sizes ratio as a function of m shows a power law behaviour with an exponent of about minus one for small mutation rates.

4.3.4 Many languages

In what follows, the results of simulations considering more than two languages ($L > 2$) are compared to the previous ones. Now we measure the "language ratio", which accounts for a possible domination of one language over the others, in the following way: we store the number of agents speaking at least language l, that is, $N(l = 1)$, $N(l = 2)$, ..., $N(l = L)$. The language ratio is then defined as:

$$R = \frac{N(l_{max}) \cdot (L-1)}{\sum\limits_{l \neq l_{max}} N(l)}, \tag{4.2}$$

where l_{max} is *the most spoken language*, and L is the total number of languages. The simulations are carried out with $c = 5$ and $K = 100,000$. We have used $f = 0.5, 1, 2, 4$ for $L = 2, 4, 8, 16$, respectively, in order to keep the number of interactions per language constant. That is, if the number of languages is doubled, also the number of contacts f each young agent makes with teachers is doubled. Figure 4.13a compares the shape of the transition curve for different numbers of languages, starting with randomly distributed languages. The position of the critical point is shifted to smaller values of m for increasing number of languages, which means that the more languages compete against each other, the lower is the possibility to have a

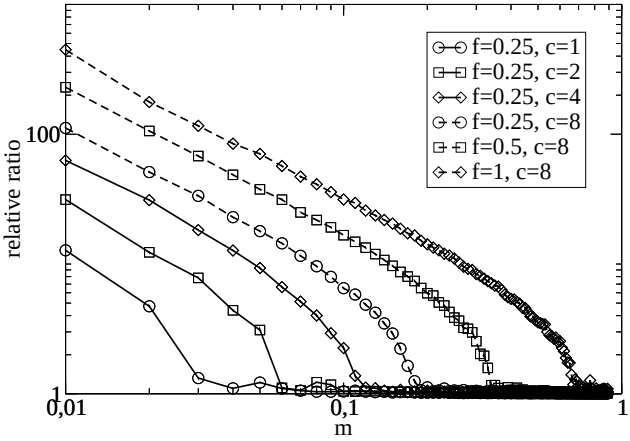

Figure 4.12: The phase transition curve is shifted to large mutation values for increasing learning time during childhood. The shift increases linearly with an increasing maximum learning age c as well as with an increasing amount f of interactions between agents per iteration.

scenario where one language dominates the others. Figure 4.13b presents the same comparison as in Figure 4.13a, but now starting with a configuration where all agents speak language $l = 1$. If we compare both figures, we see that the critical point is shifted to larger values of the mutation rate when a single language is initially spoken. This dependence of the final state on the initial one generally indicates that the phase transition is of first order. Here the point where there is a jump from the phase with domination to the fragmentation phase is located at the intersection of this transition curve with the transition curve for only two languages.

Figure 4.14 shows a histogram of the Hamming distances (Tessileanu and Meyer-Ortmanns, 2006) between all language traits for simulations with 16 languages, considering different values of the mutation rate. The parameters are the same as before. The Hamming distance is defined as the number of bits by which two bit–strings differ from each other. Or, in other words, the number of bits which need to be changed to turn the language trait of a given agent into that of another one. We can see that the peak of the histogram jumps from zero (dominance) to one (fragmentation) at the critical point (which here is located between $m = 0.006$ and $m = 0.007$).

Finally, two simplified models are compared to the previous one (Fig. 4.15). In both

we neglect ageing and thus at every iteration a new generation of agents is born and the old generation dies. The population size is kept constant. The number of interactions per iteration (generation), the mutation rate and the population size remain as parameters of both models. First we consider a sexual population where every female has two offspring, each one produced by a random choice of the mating partner. Then we consider an asexual population where the mother gives its language trait directly to its single offspring. The phase transition curve remains qualitatively the same for these simplified cases.

4.3.5 Discussion and conclusions

In our model of language competition a phase transition similar to the one obtained considering the competition of different grammars, ref. Komarova (2004), and the one obtained for the competition of languages, ref. Stauffer and Schulze (2005), is observed.

The phase where an uniform language distribution (fragmentation) appears suggests a scenario where these languages compete equally against each other, maintaining a stable state of coexistence. In the other phase one language clearly dominates the others. The dependence of the phase transition point on the amount of agent interactions as well as on the number of competing languages leads us to the following conclusions: There are no particular values for the maximum learning age of languages, c, and the number of teachers per iteration, f, where the transition changes is shape crucially. Increasing values of c simply shift the critical point towards larger values of the mutation rate m. Thus a larger maximum learning age prevents the coexistence of many languages because it increases the number of contacts among speakers of different languages making it easier for a single language to dominate. On the other side, the model also shows that a larger number of different languages decreases the possibility of dominance and increases the height of the transition jump. Hence, the extinction of a language in a multilingual system can drive the system to a state where one language begins to dominate all the other ones.

The main difference between our phase transition and the one obtained in Stauffer and Schulze (2005) is that in our case the transition is completely independent on the population size. Since we have obtained this same result using simplified models without ageing and sexual reproduction, we may say that such a result comes from the way our agents interact, which is different from that in Stauffer and Schulze (2005).

Our model has brought insight into the mechanisms of language competition but still much more has to be done. The fact that we neglect any kind of fitness, that is, different language traits do not give any advantage to the corresponding agents, and the fact that we consider sexual populations with random mating makes the simulations useful to characterise current language competition. The design of an analytical model,

for instance by diffusion equations similar to the ones used in ref. Patriarca and Leppänen (2004), but neglecting fitness would reveal if it is possible to obtain similar results with a mean field theory. It is also possible to extend the model by implementing it on a lattice in order to investigate how the transition behaves with respect to the geographic distribution of languages. Another possible extension would be the substitution of random mating by assortative mating (Kosmidis et al., 2005), a concept used frequently in the theory of biological speciation, in order to give different mating priorities to monolingual parents and bilingual ones. In order to understand language invasion, the model can be extended to include social structures.

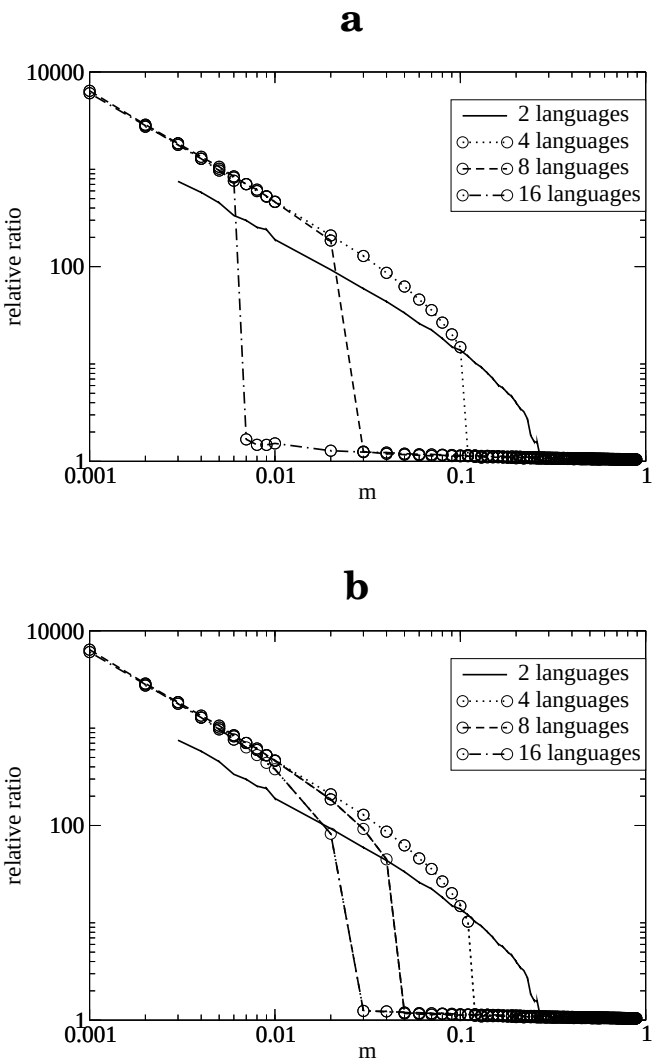

Figure 4.13: Ratio between languages versus mutation rate, for different numbers of languages, L. The critical point moves to smaller values of the mutation rate and the phase transition becomes more abrupt for increasing values of L. **a**: For $L > 2$ the curves collapse into a single one in the region where one language dominates the others. **b**: The initialisation with a single language leads to different results.

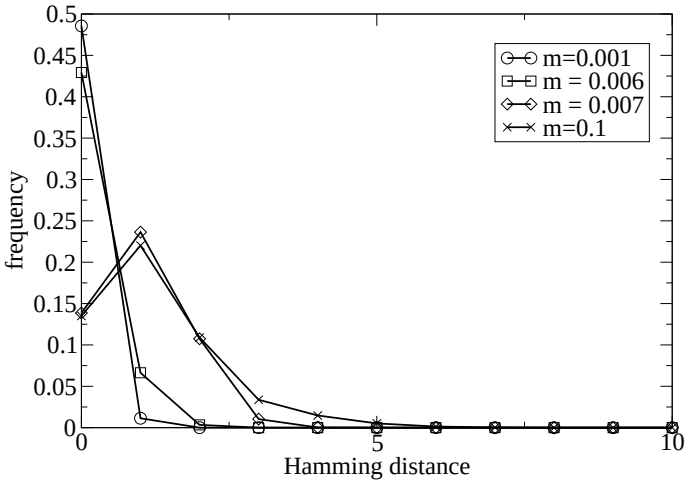

Figure 4.14: Histogram of the Hamming distances between the agents traits in a simulation of 16 languages, for different values of the mutation rate.

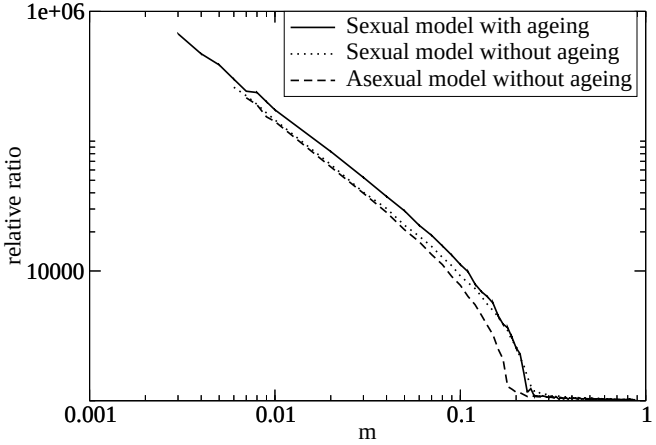

Figure 4.15: Comparison between the results obtained with the two simplified models and the former ageing model, for a system of two languages. Only small quantitative differences exist. The parameters of the model with ageing are $c = 4$, $f = 0.5$ and $K = 100000$. The sexual and asexual models without ageing have a constant population of 2000 agents and the number of agent interactions is one per generation.

Chapter 5

Conclusions

In this thesis different models were developed in order to find out more about the influence of the microscopic processes on the macroscopic behaviour of different systems. Criticality, found to be of crucial importance in evolutionary systems, and the impact of fluctuations on the various macroscopic behaviours were analysed. The results obtained with computational models on ageing, speciation and language competition will hopefully provide a step forward to understand these evolutionary phenomena and to test the underlying theories with experimental data. Resulting quantities like population densities, age structures, genetic configurations and language distributions can be measured in real systems and compared to the here presented results. Assuming that these comparisons will be successful, the results presented may help to develop a more general theory of evolutionary dynamics.

A consistent picture of ageing in humans and animals still does not exist. The Gompertz law of mortality, indicating the same exponential increase of mortality with age for humans and most animals, can be reproduced by using the Penna model. It is based on the theory of mutation accumulation and has been able to reproduce many evolutionary phenomena, among which the emergence of menopause and the catastrophic senescence of the Pacific Salmon are probably the most important ones. Using modified versions of the Penna model, simulations were carried out to understand more about the emergence of a plateau in the mortality curve for the oldest old. Such a mortality plateau was already obtained previously, also with a modified version of the Penna model for asexual populations (Coe et al., 2002). In the case presented here sexual populations were simulated and the usage of a smooth death probability function led to strong consequences on the population age-structure. The introduction of a Fermi-like death probability function, accounting for the genetic death of the individuals, described successfully the deceleration of mortality for very old individuals.

Interestingly, this approach of using the Fermi-like function forced the use of very high birth rates in order to obtain a visible oldest old effect. For this reason large population sizes were needed in order to avoid population meltdown due to large chaotic fluctuations. Using an extension of the model considering a constant population size, where the birth rate is adjusted automatically to exactly compensate the number of deaths, the effects of a smooth death probability could be illustrated more easily. Experimental data support qualitatively the results obtained: experiments on flies show that only very few reach an age where the effect of the oldest old can be observed. Additionally, the smoothness of the death probability led, in this case, to a change in the curvature of the population age distribution. This outcome can be tested on real populations.

The occurrence of sympatric speciation is still a controversial issue, but through recent experiments and theoretical models it became more and more accepted. Several models exist, mainly mean-field-like ones, which predict that sympatric speciation is possible for several scenarios. This thesis concentrates on the critical character of such bifurcations of one species into two, in order to find out which are the main mechanisms that drive a population to speciate. An individual-based model is presented, where the same phenotypic trait controls the mating choice and the fitness of the individuals. Depending on two control parameters (the amount of competition between individuals having extreme phenotypes with the ones having intermediate ones and the number of males from which the females select their mating partners), a clear out-of-equilibrium phase transition could be observed in the process of sympatric speciation. The transition is from a state of polymorphism within a single species to a state where the population splits into two new ones, reproductively isolated from each other. This phase transition presents strong similarities with those observed in physical systems. For instance, the time to reach the absorbent state of the system depends strongly on the values of the control parameters near the transition point. The results obtained for the macroscopic behaviour of the population are in agreement with those obtained with other models for sympatric speciation.

The collective behaviour of the population presents some particular features which were reproduced rather successfully with a mean-field-like model, based on the main ingredients of the individual-based model. The different phenotype densities display the same behaviour in both models, making the mean-field approach useful to investigate the results for arbitrary values of the parameters, and to compare them with those of real populations. In particular, the large fluctuations obtained in the phenotypic distribution of a species living near the transition point should be well observable in Nature.

Another model, performed to study parapatric speciation and also based on the Penna strategy for ageing, showed that fluctuations cannot be neglected. In fact, this type of speciation, which may occur in the absence of assortative mating, seems to be driven by stochastic fluctuations. The usage of age-structured populations made it possible

to check if hybrid individuals were surviving or not until they reach the minimum reproduction age, and in this way to establish if speciation had occurred or not in spite of the presence of a gene flux between extreme opposing phenotypes. Disruptive selection for different niches seems to be the crucial ingredient for parapatric speciation. This selection has to be strong enough to clearly divide the population into two subpopulations, each one adapting to a geographically different environment, but not too strong, in order to avoid that even small fluctuations drive all individuals to adapt to the same niche.

The main ingredients for a speciation process, natural selection and mutations, were implemented in a simple model which accounts for a large system of many species. Many features also found in fossil records were reproduced. The power laws obtained for the statistical distributions of the species, both in time and in size, could be compared successfully with experimental data. The simple approach allows for the origination of new species, instead of just refilling empty niches, and showed the difficulty to obtain punctuated equilibrium by only taking into account the interactions on the populational level. Mechanisms enabling the possibility of long–range correlations on the upper species level or interactions on the trophic level seem to be needed in order to obtain mass extinctions. Another interpretation would be that external events like meteor impacts are the real responsible for such extinctions. Anyway, the results of the model presented here are consistent with a background dynamics of a macro-evolutionary system of many species.

Language competition is a new field displaying evolutionary dynamics, which started in the last few years. Critical behaviour has been found, and the first steps to understand real behaviour have been realised. Our implementation of a model considering age-structured sexual populations made it possible to take into account new features, as for instance, age-dependent and sex-dependent learning. The stability of a system of two languages, where the agents can speak one or two languages simultaneously was tested. The results showed that in such a scenario the coexistence of two languages corresponds to a meta stable state. This state is more stable for large population densities, where the movement rate of the individuals does not alter the results very much. Monolingual agents populate the region they dominated at the beginning, whereas the bilinguals are of same frequency over the entire lattice.

Another model, which allows for more than two languages and age–dependent learning, suggests that the coexistence of languages is related to a certain amount of erroneous learning of other languages. Depending on this control parameter, there is a phase transition from a state of a single completely dominant language to a state where all languages have the same frequency. An optimal maximum learning age was not found. Models of language competition considering sexual populations are a promising tool to make a bridge between the field of language learning and language competition.

This thesis treats different problems, with the aim to understand better how microscopic features control macroscopic behaviour. In Statistical Physics, critical phenomena and phase transitions play a crucial role. Evolutionary systems like the ones studied in this thesis come from non-physical areas, but exhibit a general behaviour very similar to that found in Physics.

Outlook

The models described in this thesis can be extended to apply for more general cases, or to produce more exact results.

The Penna model and its extensions presented here can be merged with the implementations of other theories, as for instance the one including the effects of telomeres or the introduction of the effects of householder genes to explain ageing. This model, through its implementation of genes as the bits of a bit-string, is computationally fast, and thus can be applied to analyse the influences of almost arbitrary factors to the age structure of a population.

As done in the case of sympatric and parapatric speciation, the Penna model with its age structure provides a powerful tool to make simulations on population dynamics of ageing individuals. The models of sympatric speciation described in Sections 3.2 and 3.3 and the one of parapatric speciation (Section 3.4) provide results to be tested in real populations. For instance, the phenotypic distribution of a population near speciation is, according to these models, much broader than the one of a species far from speciation. Additionally, it would be interesting to obtain the critical exponents which determine the phase transition found with the model of sympatric speciation. Their values could determine the universality class of the transition. For this aim, extensive computer work needs to be done. In the case of the model for parapatric speciation, the statistical analysis of the number of speciation events for different values of the parameters will help to determine if there exist threshold values beyond which this type of speciation does not occur. Unfortunately, the simulation times are very long, and thus the model needs to be simplified.

The implementation of a model which allows many speciation events and a large amount of species (Section 3.5) leads to a vast number of results which correctly describe the long–term behaviour of a macro-evolutionary system. However, the comparison with data extracted from the fossil record leads to the conclusion that the dynamics of the system is still not completely reproduced. Additional features, as for example the introduction of food webs or some kind of external stress acting on the species, could help to find out, which details are still missing to obtain a general model of macro–evolution. Another approach to understand more about the principal

factors driving a system of many species would be an analytical model similar to the one of Fuentes et al. (2002), extended to take into account the effect of fluctuations.

Computer simulations on language competition already gave some insight into the rich behaviour of such systems. The main goal in the future will be the exact reproduction of the size distribution of languages on Earth, with models considering simple interactions, similar to the ones presented in this thesis. The astonishing similarity between the results of different computational agent–based models, that consider rather different interactions between the agents (compare the results of Schulze and Stauffer (2005c) and Stauffer and Schulze (2005) to the ones of the models presented here), suggests that there should exist some universal concept behind them. An approach would be to reduce the models to their very basic structures and to search for the mechanisms they have in common. An analytical approach, based on these main mechanisms, could give some more insight into the fundamental principles controlling such macroscopic phenomena, at least qualitatively.

The models described here have been built up on mean–field topologies (random interactions between the individuals) or on a square lattice. More realistic approaches take into account that some individuals interact much more than the others (Watts and Strogatz, 1998). In a possible extension of the here presented models the individuals sit on the nodes of a complex network of connections and interact according to such a topology.

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